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Spoken word processing and the effect of phonemic mismatch in aphasia

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Background: There is evidence that, unlike in typical populations, initial lexical activation upon hearing spoken words in aphasic patients is not a direct reflection of the goodness of fit between the presented stimulus and the intended target. Earlier studies have mainly used short monosyllabic target words. Short words are relatively difficult to recognise because they are not highly redundant: changing one phoneme will often result in a (similar-sounding) different word.

Aims: The present study aimed to investigate sensitivity of the lexical recognition system in aphasia. The focus was on longer words that contain more redundancy, to investigate whether aphasic adults might be impaired in deactivation of strongly activated lexical candidates. This was done by studying lexical activation upon presentation of spoken polysyllabic pseudowords (such as *procodile*) to see to what extent mismatching phonemic information leads to deactivation in the face of overwhelming support for one specific lexical candidate.

Methods & Procedures: Speeded auditory lexical decision was used to investigate response time and accuracy to pseudowords with a word-initial or word-final phonemic mismatch in 21 aphasic patients and in an age-matched control group.

Outcomes & Results: Results of an auditory lexical decision task showed that aphasic participants were less sensitive to phonemic mismatch if there was strong evidence for one particular lexical candidate, compared to the control group. Classifications of patients as Broca's vs Wernicke's or as fluent vs non-fluent did not reveal differences in sensitivity to mismatch between aphasia types. There was no reliable relationship between measures of auditory verbal short-term memory and lexical decision performance.

Conclusions: It is argued that the aphasic results can best be viewed as lexical "overactivation" and that a verbal short-term memory account is less appropriate.

Keywords: Aphasia; Lexical activation; Spoken word recognition; Speech processing; Phonemic mismatch.

The human system for recognition of spoken words is normally highly robust against distortions of external noise or accidental mispronunciations, in particular when words are long and thus contain more redundant information. Small mispronunciations can even remain undetected and may be immediately restored by the listener (Cole, 1973; Marslen-Wilson, 1985). Masked or missing speech sounds are not necessarily

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problematic for spoken word processing either, as long as the remaining speech signal is unambiguous with respect to lexical identity (Samuel, 1987; Warren, 1970). However, even though small mispronunciations in words can remain undetected in the context of a whole sentence, more and more evidence has accrued that (initial) lexical activation depends on the goodness of fit between the presented speech signal and the stored mental representation of the target word. This entails that deviations from the target are translated into reduced amounts of lexical activation of that same target. Strong evidence for gradedness in lexical activation comes from phoneme monitoring studies, such as that by Connine (1994), Connine, Titone, Deelman, and Blasko (1997), and Frauenfelder, Scholten, and Content (2001), in which the effect of phonemic mismatch on phoneme detection time is investigated. Other studies have shown that even alterations in fine phonetic detail influence the amount of activation of the intended target (Andruski, Blumstein, & Burton, 1994; Janse, Nootboom, & Quené, 2007; Mitterer & Ernestus, 2006; Salverda, Dahan, & McQueen, 2003; Shatzman & McQueen, 2006; Spinelli, McQueen & Cutler, 2003).

In language-impaired populations, however, this direct translation from bottom-up acoustic evidence to lexical activation may be disturbed, either because sound processing is hampered, due to the process of mapping speech to the lexicon being impaired, or because the lexical representations themselves are less clearly specified. Problems in spoken word processing in aphasia, an acquired language disorder often caused by stroke, have been described in terms of an overall decreased or increased level of lexical activation, depending on the aphasia syndrome (see Blumstein, 2007, for an overview). This description followed from the conclusion that aphasic patients' ability to perform sub-lexical speech tasks (phoneme identification, discrimination and rhyming tasks) double-dissociates from word comprehension ability. This means that patients who perform poorly on syllable discrimination and identification tasks may have good word-level auditory comprehension, and vice versa (Miceli, Gainotti, Caltagirone, & Masullo, 1980). Functional differences between aphasia syndromes in terms of patients' difficulty with language comprehension might be understood better if lexical activation upon auditory presentation of speech is investigated, rather than performance on ecologically less valid speech tasks such as discrimination—a task that is not involved in normal auditory sentence comprehension (see also Hickok & Poeppel, 2004). Lexical activation studies into aphasia (using on-line methods) started off with the mediated semantic priming study of Milberg, Blumstein, and Dworetzky (1988). Presentation of a non-word such as *gat* can activate the real word *cat*, which in turn facilitates recognition of the semantically related word *dog*. Thus, lexical activation of *cat* was investigated by studying how quickly listeners responded to the word *dog*, given that the word *dog* had been preceded by a prime item such as *cat*, *gat*, or *wat* (the latter two could be perceived as distorted versions of *cat*). Thus, the more quickly one recognises the word *dog*, relative to some kind of baseline condition in which the word is preceded by an unrelated prime, the more activation can be assumed for the word *cat*. Milberg et al. showed that activation of *dog* in the non-brain-damaged control group was proportional to the amount of bottom-up support for *cat*. However, in participants with fluent (Wernicke's) aphasia the amount of activation of *dog* was not as strongly dependent on the goodness of fit between the presented prime (*cat*, *gat*, or *wat*) and the “intended” word CAT, since these patients showed mediated semantic priming in all phonological distortion conditions (suggestive of increased lexical activation). The non-fluent group, on the other hand, showed

priming only in the undistorted related condition, which suggests decreased lexical activation.

Even though these mediated priming results involving rhyme primes (i.e., *gat* for *cat*) were difficult to replicate either in aphasic or in non-brain-damaged populations (Baum, 1997), similar “decreased lexical activation” results for non-fluent (Broca’s) aphasic patients were obtained by Utman, Blumstein, and Sullivan (2001). Whereas presentation of a distorted prime in which the initial consonant is manipulated in voice-onset time (*c*at*) normally results in a short-lived reduction in the amount of activation of the related word *dog*, relative to the presentation of undegraded *cat*, Broca’s aphasic patients turned out to be more vulnerable to such subphonetic variations. Subphonetic degradations resulted in an even greater reduction in lexical activation than found for control participants, but only in conditions of competition, i.e., when words such as *c*oat* were presented (having the competitor *goat*). A similar result was obtained in Misiurski, Blumstein, Rissman, and Berman (2005). Misiurski et al. showed mediated priming (VOT-manipulated *t*time* primes *penny* via *dime*) for non-brain-damaged participants. The Broca’s aphasic participants, despite showing effects of semantic priming from *dime* to *penny*, showed no mediated priming. Misiurski et al. (2005) suggest that bottom-up activation levels for acoustically manipulated prime items are not sufficient to overcome lexical competition in the Broca’s aphasic participants.

Thus, in both diagnostic aphasia types the amount of lexical activation is not a direct reflection of the goodness of fit between the presented stimulus and the target it is intended to activate. In Broca’s aphasic patients lexical activation seems to underrepresent the goodness of fit between the presented stimulus and the target (Misiurski et al., 2005; Utman et al., 2001). In Wernicke’s aphasic patients, on the other hand, mismatching information may not lead to proportional deactivation of once-appropriate candidates (Janse, 2006; Yee, Blumstein, & Sedivy, 2004). During the recognition of spoken words, lexical items compete for recognition. When enough sound information has been received to identify the “winning” lexical candidate, the co-activated competing candidates are consequently deactivated such that they are more difficult to recognise upon a next encounter, yielding inhibition when the overlap consists of at least two phonemes (Monsell & Hirsch, 1998; Slowiaczek & Hamburger, 1992). However, a study with form-overlapping primes (Dutch word pair *salaris* – *salami* “salary-salami”; Janse, 2006) showed an interaction between the effect of onset overlap and listener group—whereas an inhibitory effect of form overlap on recognition of the second item was found for a group of control listeners, a facilitatory effect of form overlap was found for Wernicke’s aphasic participants. Even when several items intervened between prime (*salaris*) and target (*salami*), co-activated word candidates still showed persisting activation, which suggests that Wernicke’s aphasic patients are impaired in suppression of once-activated word candidates. Or, put into terms of phonemic mismatch or bottom-up inhibition (cf. Frauenfelder et al., 2001), once mismatching information comes in, lexical activation of actually no-longer-appropriate items does not decrease proportionally.

In the studies mentioned above, competition between candidates played an important role. For the Broca’s aphasic patients, underactivation was found only in conditions of competition (Misiurski et al., 2005; Utman et al., 2001). For the Wernicke’s study, positive evidence for the winning candidate did not go hand in hand with deactivation of the (no-longer-appropriate) competitor candidate (Janse, 2006). Expanding on this previous study, the present study aims to investigate lexical

processing in aphasia in further detail by focusing on situations in which there is actually only one possible lexical candidate left but there is a segmental mismatch. If positive segmental evidence for a competing lexical candidate (*salaris* “salary”) does not deactivate the segmentally no-longer-appropriate candidate (*salami* “salami”) in Wernicke’s aphasic patients, one would certainly not expect a segmental mismatch to yield proportional deactivation if there is no competitor left (such as in a longer pseudoword, e.g., *candidape* for *candidate*). In other words, the present study investigates the sensitivity of the auditory lexical recognition system in aphasia: To what extent does segmental mismatching information translate into (proportionally) reduced amounts of lexical activation, in the face of overwhelming evidence for one particular lexical candidate? This also raises the question how “sensitive” the lexical recognition system of Broca’s aphasic patients is to segmental mismatches given overwhelming evidence for only one particular lexical candidate. Note that Milberg et al. (1988) found initial decreased lexical activation in non-fluent aphasic patients even in conditions without lexical competitors. Upon presentation of longer (polysyllabic) pseudowords with only one mismatching phoneme, as was done in the present study, a certain amount of activation of the target lexical candidate is expected for all aphasic participants, but activation may still under-represent the goodness of fit between the sound structure and the lexical target in Broca’s aphasic patients (cf. Misiurski et al., 2005; Utman et al., 2001).

Thus the hypothesis was tested that, even in the absence of appropriate competing candidates, Broca’s aphasic patients show underactivation upon presentation of word-like pseudowords, relative to non-brain-damaged control listeners. Secondly, “overactivation” was expected for Wernicke’s aphasic patients in the sense that upon presentation of a spoken pseudoword containing a phonemic mismatch (e.g., in a word-like non-word such as *procodile* or *candidape*) more lexical activation was expected for Wernicke’s aphasic patients than for a control group of age-matched listeners. Again, if positive evidence for a competing lexical candidate does not yield proportional deactivation of no-longer-appropriate candidates (Janse, 2006), deactivation due to mismatch is not expected to occur in the face of overwhelming evidence for one specific lexical candidate either.

Note that the role of auditory short-term memory problems in aphasia should also be taken into account. Blumstein et al. (2000) conducted a repetition priming study with aphasic participants, using word and non-word targets. This was done to investigate whether Broca’s and Wernicke’s aphasic participants were able to maintain a stable sound representation of words and non-words. Unlike the control participants, who showed repetition priming effects for words and non-words, *both* the Broca’s and Wernicke’s aphasic participants showed repetition priming for words only. No repetition priming for non-words was found, not even when no items intervened between prime and target. This suggests that all sound information that does not contact the lexicon fades relatively quickly. This is important if one considers what might happen upon presentation of a pseudoword with a mismatching phoneme in word-initial position (e.g., *pelephone* for *telephone*). Upon presentation of such a pseudoword, aphasic patients, like non-brain-damaged control participants, will gradually zoom in on the target word, despite the initial word fragment. Once past the theoretical uniqueness point (granted that aphasic participants may need more information than non-brain-damaged participants to recognise a word) the aphasic listeners will be faced with converging evidence for one particular lexical candidate. Yet any sound information that does not contact the

lexicon fades quickly, which makes it difficult to go back to this “echo” of the initial fragment. Thus, even though the initial phoneme did not provide bottom-up support for the real word target, this can be overruled by later arriving “convincing” evidence, *particularly* if there is no trace left of the actually presented fragment.

An auditory short-term memory approach would make the same predictions for the Wernicke’s aphasic participants as the “overactivation” approach mentioned above: both approaches predict that Wernicke’s aphasic participants will be less sensitive to mismatching information in pseudowords than their non-brain-damaged control participants. The hypothesis derived from this approach for the Broca’s aphasic participants, however, differs from the earlier “underactivation” prediction: since auditory short-term memory impairments are found across the aphasic spectrum, Broca’s aphasic patients, as all aphasic patients, are predicted to be *less* sensitive to mismatching information than non-brain-damaged control participants. The two views thus only make different predictions for the Broca’s aphasic patients. Furthermore, in this auditory short-term memory view, aphasic patients, including Broca’s aphasic patients, would necessarily be less sensitive to mismatching information in word-initial position than in word-final position. Any deficits in mapping sound to the lexicon may be reinforced by working memory demands (cf. Gathercole & Baddeley, 1990). The effect of a mismatching phoneme in word-initial position in longer pseudowords (such as *pelephone* for *telephone*) was therefore compared to that of a mismatching phoneme in word-final position (e.g., *cabinep* for *cabinet*).

Martin and Gupta (2004) hypothesised a relation between verbal short-term memory (the ability to maintain activation of representations in short-term memory) and severity of lexical processing impairment in aphasia. Therefore, apart from classifying patients into aphasia types or groups and comparing those data, the results of the present study will also be analysed in a more continuous way by correlating patients’ performance to a background measure of auditory verbal short-term memory (a standardised auditory discrimination task of the PALPA test battery (PALPA 1: discrimination of non-word pairs; Dutch version; Bastiaanse, Bosje, & Visch-Brink, 1995). The use of PALPA 1 has the advantage that it also involves non-words, as in the present study. In this way the relation between lexical processing and the ability to maintain activation in auditory short-term memory can be investigated.

The present study was set up to investigate sensitivity of the lexical processing system in aphasia. This is put to the test by presenting pseudowords that closely resemble one specific lexical item. The severity of the aphasic participants’ deficits in mapping sound to the lexicon was assessed by varying the salience of the mismatching information. This was done by manipulating the phonetic distance between target and mismatching phoneme (involving one or more phonetic features), and by having the mismatching phoneme occur either in a stressed or unstressed syllable. The obvious expectation is that the less salient the change (relative to the target word), the more likely it is that the mismatching information goes unnoticed.

The following hypotheses were tested:

1. Sensitivity to phonemic mismatch in lexical activation upon presentation of spoken polysyllabic pseudowords is lower in a population of Wernicke’s aphasic patients than in a control group of age-matched listeners.
2. For Broca’s aphasic patients, there are two conflicting hypotheses. In an “underactivation” view, sensitivity to mismatch would be higher than in a

control group. In a verbal short-term memory view, sensitivity would be lower than in a control group, particularly if the phonemic mismatch occurs in word-initial position.

METHOD

Experimental task

Speeded auditory lexical decision was chosen to test these hypotheses and to measure lexical activation, because of the simplicity of the task. Many studies on the effect of phonemic mismatch on lexical activation in typical populations have used (speeded) phoneme detection (Connine et al., 1997; Frauenfelder et al., 2001), which may be a difficult task for aphasic participants. Furthermore, when the number of participants is relatively low (as is often the case in aphasia studies), within-participant designs are desirable, which also ruled out tasks like (semantic) priming paradigms with blocked designs (to avoid repetition of the same target). In line with studies on the effects of lexical neighbourhood density on non-word processing, which also used auditory lexical decision (Luce & Pisoni, 1998; Vitevitch & Luce, 1999), it was assumed that lexical decision performance would reflect lexical activation levels upon auditory input and would thus be an appropriate measure of spoken word processing. The amount of lexical activation was assessed by studying accuracy and reaction time of a lexical decision response upon presentation of a pseudoword, compared to that upon presentation of a non-word without obvious lexical resemblance. In this way, accuracy and response time can be compared in two no-response conditions (“no, this is not a word in my language”). The larger the difference between the two conditions, the more lexical activation is assumed for the pseudowords. In other words, overactivation, or being relatively insensitive to phonemic mismatch, translates either in incorrect YES responses or in slow NO responses, relative to baseline performance on the non-words without lexical resemblance. If the control participants hardly make any errors, then the RT data may still show that some non-words are easier to reject than others, thus yielding (subtle) differences between conditions.

Similarly, a pattern of underactivation would be shown if the Broca’s aphasic participants show a relatively smaller difference (in terms of accuracy and RT) between the two non-word conditions than the non-brain-damaged control group: if presentation of pseudowords yields less lexical activation in Broca’s aphasia, these aphasic participants should be able to respond faster and more accurately than the control participants, at least relative to their baseline performance on the non-words without obvious lexical resemblance.

It should be noted, however, that lexical decision may involve decision processes that are normally not involved in spoken word processing. This issue will be taken up again in the Discussion.

Materials

The Dutch language material consisted of 80 mismatch pseudoword items (e.g., *figa’ret* for *cigaret* “cigarette”), 80 nonsense items (e.g., non-word *pego’leen*), and 140 real words (as fillers, e.g., *abrikoos* “apricot”). Each item had three or four syllables. All non-word items were phonotactically legal in Dutch. The pseudoword

items differed on three aspects: position of the phoneme mismatch, whether the mismatch was minimal or maximal, and whether or not the syllable containing the mismatching phoneme had main stress. It makes sense that the greater the acoustic/perceptual difference between the changed and canonical word form, the greater the possible effect on immediate lexical activation.

Of the 80 pseudowords, 40 had a phonemic mismatch in word-initial position (e.g., *fáprika* for *paprika* “paprika”) and 40 had a word-final mismatch (e.g., *krokodír* for *krokodil* “crocodile”). Because the pseudowords were relatively long, the mismatch occurred after the theoretical uniqueness point (or, for the initial mismatches, the remaining evidence also converged to one specific lexical item).¹ Within these position categories, half (20) of the items had altered phonemes constituting a minimal mismatch (e.g., *pelefoon* for *telefoon* “telephone”) in which only place of articulation, manner, or voice was altered,² and half (20) of the items had altered phonemes constituting a maximal mismatch (e.g., *sáraphon* for *marathon* “marathon”), in which all of them were altered. Note, however, that Dutch has final devoicing, so that only place and manner were modified in maximal mismatches in final position. Within these categories of 20 items, half of the items (10) had mismatching information in the syllable with main lexical stress (e.g., *kapitéif* for *kapitein* “captain”), and half (10) had the mismatching information in an unstressed syllable (e.g., *karáktef* for *karakter* “character”). This was another manipulation of mismatch salience: mismatching phonemes were expected to be more salient when they occurred in lexically stressed than unstressed syllables.

For each pseudoword, a control nonsense item was composed, which shared as many phonemes as possible as its pseudoword counterpart (e.g., *fákarip* from *fáprika* for *paprika* “paprika”). However, the phonemes were placed in a different order to avoid lexical resemblance. Because lexical activation upon presentation of pseudowords was compared to that upon presentation of matched nonsense items, lexical decision accuracy and response time (from word onset) were investigated. Table 1 provides an overview of the non-word conditions; a complete list of all non-word items is provided in Appendix A.

The 160 non-word stimuli, plus 140 real words as fillers and an additional 12 practice stimuli, were recorded by a female speaker of standard Dutch. Each item was stored as a separate sound file, and downsampled to 32 kHz. The pseudowords had a mean duration of 789 ms ($SD=94$). In order to match pseudowords and nonsense items as closely as possible (apart from lexical resemblance, obviously) the nonsense items were time-scaled by way of PSOLA time compression or expansion, to the exact same duration as their pseudoword counterparts.³ This did not yield any audible artefacts. Nevertheless, all real words and pseudowords were PSOLA resynthesised as well (without time-scaling), again without any audible artefacts.

¹ It was impossible to keep the position of the uniqueness point constant over items, or the number of competitors before the uniqueness point.

² Note that a change from /p/ to /f/ in *fáprika* (see Appendix A) is, strictly speaking, not only a change in manner, but in place of articulation as well, since /f/ is labiodental. These items (involving a /p/-to-/f/ change or a /p/-to-/v/ change) were however grouped with the minimal mismatches.

³ PSOLA (pitch-synchronous-overlap-add, as incorporated in most sound-editing programmes) is the most common technique to time-scale sound files: it yields high-quality output speech without affecting pitch height.

TABLE 1
An overview of the non-word conditions with stimuli examples

	Initial mismatch		Final mismatch	
	Pseudoword	Nonsense	Pseudoword	Nonsense
<i>Minimal mismatch</i>				
In stressed syllable	<u>d</u> róccoli (from <i>broccoli</i> “broccoli”) /drəkoli/	dróngila /drɔŋila/	krokod <u>ir</u> (from <i>krokodil</i> “crocodile”) /krokodir/	geufad <u>ir</u> /xɔfadir/
In unstressed syllable	f <u>i</u> garet (from <i>sigaret</i> “cigarette”) /fixaret/	firegát /firexat/	salá <u>ri</u> f (from <i>salaris</i> “salary”) /salaris/	losóerif /losurif/
<i>Maximal mismatch</i>				
In stressed syllable	<u>s</u> áration (from <i>marathon</i> “marathon”) /sarətɔn/	sátorran /satɔran/	kapité <u>if</u> (from <i>kapitein</i> “captain”) /kapiteif/	zielotéif /ziloteif/
In unstressed syllable	g <u>a</u> teriáal (from <i>materiaal</i> “material”) /xatərial/	gatielióom /xatəliom/	karák <u>t</u> ef (from <i>karakter</i> “character”) /karaktəf/	mikrógt <u>e</u> f /mikrɔxtəf/

Procedure

The experimental software programme TEMPO (Motta, Rizzo, Swinney, & Piñango, 2000) was used to present the participants with the stimuli in a random order. The material was presented in four blocks, preceded by a practice block of 12 stimuli (eliciting both YES and NO responses) to familiarise the participants with the task and the speech material. Participants were informed that they could pause in between the blocks. The materials were presented at a comfortable loudness level. Participants wore sealed headphones and were asked to respond as quickly as possible by pressing either of two response buttons (labelled YES and NO), without sacrificing accuracy. Following item onset there was a 3-second window in which the response could be given. After 4 seconds the next item was presented. Participants could choose whether they used one or two hands to press the YES and NO buttons.

Participants

A total of 21 aphasic patients volunteered to participate in the present study. All aphasic and control participants were native speakers of Dutch. Hearing acuity was not assessed in either the non-brain-damaged control group or the aphasic patient group. Participants reported having no hearing difficulties, except for one aphasic participant. This participant's hearing was consequently tested and he was indeed shown to have significant hearing loss; his results were therefore excluded. The aphasic participants were recruited via several rehabilitation centres and speech therapy practices in the Netherlands where they received speech/language therapy or occupational therapy. The design and procedure were approved by the Medical Ethics Committee of the University Medical Centre in Groningen. All patients gave their informed consent. The aphasic patients had been diagnosed on the basis of the Dutch version of the Aachen Aphasia Test (Graetz, de Bleser, & Willmes, 1992) as part of their speech and language therapy programme.

Of the 21 aphasic participants, 8 were classified as non-fluent: they were often apraxic, and were diagnosed as suffering either from Broca's aphasia or global aphasia. The other 13 participants could be classified as fluent: they were not apraxic, and were diagnosed as suffering from either Wernicke's or amnesic aphasia.

The results will be analysed for group differences between Broca's vs Wernicke's aphasic patients (Blumstein et al., 2000; Milberg et al., 1988) to investigate the underactivation/overactivation hypotheses for the two respective aphasia types. In line with some researchers who have grouped aphasic patients into fluent vs non-fluent aphasia type groups (e.g., Baum, 1997; Gordon & Baum, 1994), the results will also be analysed for such group differences to investigate whether the underactivation/overactivation differences might hold for this broader classification. The choice not to restrict the aphasic population to those with either a clear Broca's or Wernicke's aphasia diagnosis was not just a pragmatic one. More patients can be included if the selection criteria are less strict, but the choice to have a broader spectrum of aphasia severity also allows for the investigation of the link between verbal short-term memory deficits and lexical processing problems. Thus, apart from comparisons between aphasia types, the results will be analysed in relation to each individual patient's score on a verbal short-term memory measure.

A number of relevant patient characteristics are provided in Table 2. The 12 control non-brain-damaged participants (6 male, 6 female) had a mean age of 57;6

TABLE 2
Information about the aphasic patients

<i>Patient</i>	<i>AAT classification</i>	<i>Age</i>	<i>Sex</i>	<i>Aetiology</i>	<i>Months post-onset</i>
NF1	global	43	F	CVA-L, ACM ^a	13
NF2	global	53	F	CVA-L	4
NF3	Broca	53	M	CVA-L	4
NF4	Broca	58	M	CVA-L	12
NF5	Broca	63	F	Politrauma,tempo-parietal	13
NF6	Broca	54	M	CVA-L	30
NF7	Broca	53	M	CVA-L	120
NF8	global	43	F	CVA-L	14
F1	Wernicke	72	M	CVA-L, parietal	3
F2	Wernicke	59	F	CVA-L, temporal	7
F3	Wernicke	57	F	CVA-L, ACM	6
F4	Wernicke	65	M	CVA-L	7
F5	Wernicke	64	M	CVA-L, par-occip.	6
F6	amnesic	64	M	CVA-L	4
F7	amnesic	54	M	CVA-L	22
F8	amnesic	67	M	CVA-L, ACM	20
F9	Wernicke	50	F	CVA-L	19
F10	Wernicke	33	M	CVA-L, posterior	50
F11	amnesic	43	F	CVA-L, ACM	22
F12	mixed	67	F	CVA-L, par.-occip.	3
F13	Wernicke	48	F	CVA-L (SAB ACM)	6

^aCVA in the Arteria Cerebri Media.

years (range 36–68 years). This group was age-matched to the fluent aphasic patient group who were, on average, somewhat older (57;2 years; range 33–72 years) than the non-fluent aphasic patients (mean age 52;6 years; range 43–63 years). The fluent and non-fluent patient groups did not differ significantly in aphasia severity (as measured by Token Test performance): mean Token Test error score did not differ significantly between the non-fluent (mean TT error score=35.6; *SE*=4.4) and the fluent group (mean score 32.2, *SE*=2.3) *t*(19)<1, *ns*. Post-onset time in the non-fluent patient group (mean time=26.3 months, *SE*=13.7) did not differ significantly from that in the fluent group (mean time=13.5 months; *SE*=3.7) *t*(19)=1.0, *ns*.

RESULTS

Fluent/non-fluent aphasic patients and non-brain-damaged control participants

The results were first analysed in terms of a fluent/non-fluent classification of the aphasic participants. The hypothesis should then be rephrased: overactivation (lower sensitivity to mismatch) is expected for the fluent aphasic patients, relative to control participants, whereas underactivation (higher sensitivity to mismatch) is expected for the non-fluent aphasic patients. This was assessed by investigating the two dependent variables obtained in the auditory lexical decision experiment: accuracy rate and response times to the non-words.

Results were analysed by way of mixed five-way ANOVAs (the data were analysed by participants and by items). The only *between-participant* factor was

Participant Group (three levels: control adults vs fluent aphasic participants vs non-fluent aphasic participants). All word and non-word items were presented to each and every participant. The factor Non-word Type (two levels: pseudowords vs nonsense items) was varied within item (or actually within an item pair: remember that each pseudoword item had its matched nonsense counterpart). The following factors varied between pseudoword items: Position of the mismatch (two levels: mismatch in initial versus final position), Mismatch type (two levels: minimal versus maximal mismatch), and Stress (two levels: mismatch in a stressed versus unstressed syllable).

Accuracy to *nonsense items* would not necessarily differ between groups, such that differential sensitivity to mismatch should show up as interactions between Non-word type (pseudoword vs nonsense item) and Participant group. Differential sensitivity to mismatch could also show up specifically for some types of pseudowords, which would show up as interactions between Participant Group and, e.g., Mismatch Type (for the pseudoword subset of the non-words only). Thus, even though there are five factors in the design, the factors Mismatch Type, Mismatch Position, and Stress do not apply to the nonsense items. Hence not all interactions are meaningful, and pairwise comparisons are needed to address issues of sensitivity to specific types of mismatches. Nevertheless, the nonsense items provide baseline performance for each participant and item pair (accuracy and RT).

Overall lexical decision performance for the non-words is provided for the three participant groups in Figure 1. Lexical decision time was measured from word onset.

As background data, lexical decision accuracy for the real words was 98% for the controls (mean RT of 1044 ms; $SD=281$) and 94% for the aphasic participants (mean RT of 1329 ms; $SD=426$). The presence of so many word-like pseudowords may have made listeners somewhat cautious in responding YES to the real words.

Figure 1. Lexical decision performance (bars represent mean response time; lines represent accuracy rates) for the three participant groups. Error bars represent 1 *SE*.

Nevertheless, the proportion of false rejections (saying NO to a real word) was significantly lower than the proportion of false positives (saying YES to a pseudoword) in the aphasic patient population—paired *t*-test on accuracy rate for the pseudowords condition compared to that for the real words: $t(20)=4.22$, $p<.001$. Note that accuracy in the nonsense item condition was high for all participants.

Accuracy rates to the non-words were arcsine transformed because of ceiling effects in the nonsense conditions (and generally in all of the control participants' data). They were then entered into ANOVAs by participant and by item to test the effects of all factors. The results of the accuracy analyses are reported in Appendix B: the most important results (i.e., those involving participant group differences) are discussed here. There were significant main effects of Non-word Type, $F_1(1, 30)=88.64$, $p<.001$; $F_2(1, 72)=91.01$, $p<.001$, and of Participant Group, $F_1(2, 30)=9.57$, $p<.001$; $F_2(2, 71)=65.93$, $p<.001$. This indicates that accuracy was higher for the nonsense items than for the pseudowords, and that accuracy rates differed for the listener groups. There was also a significant Participant Group * Non-word Type interaction, $F_1(2, 30)=8.92$, $p=.001$; $F_2(2, 71)=67.31$, $p<.001$. Figure 1 clearly shows that the accuracy difference between non-word conditions is larger in the two aphasic populations than in the control group of non-brain-damaged listeners. Note, however, that even though the accuracy rates had been arcsine transformed, ceiling effects in the accuracy data (of the non-brain-damaged listeners) may have influenced the data.

More detail on accuracy rates in the different mismatch conditions is given in Figure 2. In this figure accuracy in the nonsense item conditions is left out because they are 99–100% correct for the aphasic patients as well.

The three-way interaction between Participant Group, Non-word Type, and Mismatch Type was significant by participants and items, $F_1(2, 30)=5.66$, $p=.008$; $F_2(2, 71)=4.20$, $p=.019$. We can zoom into this finding by carrying out pairwise comparisons (with Bonferroni adjustment) because the factor Mismatch Type in fact only applies to the pseudowords and not to the nonsense words. In the nonsense word conditions there were no accuracy differences between participant groups. In all pseudoword conditions accuracy rates differed between control participants and

Figure 2. Mean accuracy rate (%) in different pseudoword conditions (error bars represent 1 *SE*) for the three participant groups. The pseudoword contains a phonemic mismatch in either word-initial or word-final position, and the mismatch is a minimal or maximal deviation from the target phoneme.

aphasic participants (all p values $< .05$). Furthermore, running the ANOVA again on the pseudoword items only shows a significant interaction between Participant Group and Mismatch Type as well, $F_1(2, 30) = 5.57, p = .009$; $F_2(2, 71) = 3.36, p = .040$. This indicates that the difference between the minimal and maximal mismatch conditions was larger for the aphasic participants. This relates to the aphasic participants' sensitivity to phonemic mismatch in mapping sound to the lexicon. Aphasic participants were not insensitive to phonemic mismatch: Figure 2 shows that lexical resemblance mainly overruled the mismatching information in the aphasic participants if the phonemic mismatch was a minimal (non-salient) one. Note that there were no significant interactions between Participant Group and Mismatch Position: there was no evidence that the overall lower accuracy for mismatches in initial position was modified by Participant Group.

Importantly, differences were expected between the two aphasic populations: higher error rates to the pseudowords were expected for the fluent aphasic population, relative to the control participants, whereas the non-fluent aphasic patients were expected to be less sensitive to lexical similarity than the control participants. Post-hoc pairwise comparisons (with Bonferroni adjustment) showed that the significant main effect of Participant Group (mentioned above) was due to differences between both of the aphasic patient groups and the control group (both comparisons $p < .01$): there was no overall difference between fluent and non-fluent patients. The same applied to interactions between Participant Group and Non-word Type and that between Participant Group, Non-word Type, and Mismatch Type: post-hoc pairwise comparisons (with Bonferroni adjustment) showed that the two aphasic participant groups did not differ significantly in either of the (pseudoword) conditions (all p -values $> .1$). It follows that the effect of Mismatch Type on accuracy was therefore not larger for either aphasic participant group compared to the other.⁴ Thus, the interactions mentioned above were due merely to differential accuracy effects in the aphasic participants, relative to the control participants, rather than to differences between the two groups of fluent vs non-fluent aphasic participants.

Response times of correct NO decisions to the non-words were analysed as well to test the hypotheses of overactivation for the fluent aphasic participants and of underactivation for the non-fluent aphasic participants, relative to the control participants. Again, such differential activation patterns should show up as interactions between Participant Group and Non-word Type (possibly particularly for specific non-salient mismatches). Response times for correct NO decisions were transformed to $1/RT$ values to make the data distribution less skewed. These inverse response times were analysed by participants (nested under Participant Group) and items (nested under the item characteristics of whether the mismatch was minimal or maximal, in a stressed syllable or not, and whether it occurred word-initially or word-finally). Appendix C lists the ANOVA results of this RT analysis. There were main effects of Non-word Type—meaning faster NO responses to the nonsense items than to the pseudowords: $F_1(1, 30) = 169.81, p < .001$; $F_2(1, 72) = 171.88, p < .001$ —and of Participant Group, $F_1(2, 30) = 16.05, p < .001$; $F_2(2, 71) = 1571.32, p < .001$. The interaction between Participant Group and Non-word Type failed to reach significance, $F_1(2, 30) < 1, ns$; $F_2(2, 71) = 2.32, ns$. In other words, there were no

⁴This was verified with a separate ANOVA: if the (pseudoword) data are restricted to those of the aphasic participants, there is no significant Participant Group * Mismatch Type interaction.

differences between listener groups in terms of how quickly they could reject the pseudowords, in relation to how fast they were on the nonsense items.

Figure 3 displays mean response times in each listener group to the pseudowords (having either a word-initial or word-final mismatch), and response times in the two respective control conditions (remember that each pseudoword had its matched nonsense item). Figure 4 presents data of pseudoword conditions only (broken down by Mismatch Position and Mismatch Type), but note that there was no interaction between Participant Group and Mismatch *Type* in the RT data.

Figure 3 shows that the non-brain-damaged control participants responded relatively slowly to the final mismatch pseudowords. This makes sense since RT was measured from word onset: listeners can decide during presentation of the nonsense items and during the initial-mismatch pseudowords that these are not words of Dutch, but they have to wait till the end of the final-mismatch pseudowords before

Figure 3. Mean response times (error bars represent 1 *SE*) in different non-word conditions for the three participant groups. Non-word conditions are: pseudowords with an initial or final mismatch (MM) and the corresponding nonsense conditions.

Figure 4. Mean response time (error bars represent 1 *SE*) in different pseudoword conditions. The phonemic mismatch is either in word-initial or word-final position, and is a minimal or maximal deviation from the target phoneme.

they can make their decision. Figure 3 clearly shows that the RT difference between the initial-mismatch and final-mismatch pseudoword conditions is much smaller for the two aphasic participant groups. This was indicated by a significant interaction between Participant Group, Non-word Type, and Mismatch Position, $F_1(2, 30) = 6.36$; $p = .005$; $F_2(2, 71) = 15.66$, $p < .001$. None of the other interactions involving Participant Group reached significance.

Since the factor Mismatch Position only applies to the pseudowords and not to the nonsense words, this interaction between Position and Participant Group was also investigated by way of post-hoc tests (with Bonferroni adjustment) to specifically look at the pseudoword data. For the control participants, in the pseudoword conditions (and not in the nonsense conditions), pairwise comparison between the initial and final mismatch conditions showed a significant RT difference ($p < .001$). Even though Figure 3 suggests that this effect is reversed for the fluent aphasic participants, the post-hoc tests showed that the difference between the initial mismatch and final mismatch (pseudoword) conditions failed to reach significance ($p > .1$). The same held for the non-fluent aphasic participants: the RT difference between initial and final mismatch pseudoword conditions was not significant. This provides further support that the difference between the initial-mismatch and final-mismatch *pseudoword* conditions is smaller (in fact, absent) for the two aphasic participant groups than for the control participants.

As in the accuracy analyses, post-hoc testing was also required to investigate whether any of the interactions mentioned above could be attributed to differences between the two aphasic participant groups. Post-hoc testing (with Bonferroni adjustment) on the main effect of Participant Group showed that the effect of Participant Group was due only to a difference between control and aphasic participants (difference between fluent and non-fluent aphasic participants, by participants and items, $p > .1$). Post-hoc tests on the significant interaction between Participant Group, Non-word Type, and Mismatch Position showed no significant differences whatsoever between the two aphasic populations: the two aphasic participant groups did not differ in the nonsense conditions, or in the pseudoword conditions (not in the pseudowords with an initial mismatch, nor in those with a final mismatch; differences between the two aphasic populations $p > .1$). Thus, again, this interaction involving Participant Group was due to differences between the aphasic participant groups on the one hand and the control participants on the other hand.

Broca's/Wernicke's aphasic patients and non-brain-damaged control participants

Aphasic patients have been categorised as fluent or non-fluent up to now. Such a classification also includes patients diagnosed with an amnesic or global aphasia, whereas the overactivation/underactivation literature has actually shown a lexical activation difference between Broca's and Wernicke's aphasic patients. The same analyses as reported above (in the fluent vs non-fluent patients results section) were run again, except that the aphasic participants were restricted to patients who were either classified as Broca's ($N = 5$) or Wernicke's aphasic patients ($N = 8$; cf. Table 1) to investigate differences between patient groups.

Accuracy. The accuracy results for these restricted patient groups are shown in Figure 5 (again leaving out the nonsense item conditions).

Figure 5. Mean accuracy rates (%) in different mismatch conditions (error bars represent 1 SE).

In short, the statistical analyses showed the same results as found in the non-fluent/ fluent analyses: all interactions involving Participant Group were due to differences between control and aphasic participants, rather than to differences between the two aphasic participant groups.

Response times. Mean response times of correct NO responses to non-words (of the control participants and Broca's and Wernicke's aphasic participants) are shown in Figure 6.

RTs were analysed to test the hypotheses of overactivation for the Wernicke's aphasic participants and underactivation for the Broca's aphasic participants (testing the same main effects and interactions as noted in the results subsection above). Again, the statistical analyses showed the same results as found in the non-fluent/ fluent analyses. None of the post-hoc comparisons showed significant differences between Broca's and Wernicke's patients. This means that all interactions involving Participant Group were due to differences between control and aphasic participants,

Figure 6. Mean response times (error bars represent 1 SE) in different non-word conditions for the three participant groups. Non-word conditions are: pseudowords with an initial or final mismatch (MM) and their respective control conditions.

rather than to differences between the two aphasic participant groups. Figure 6 shows that the Broca's aphasic patients take roughly equally long to reject initial and final mismatches, relative to the respective nonsense-item conditions. However, the Wernicke's aphasic participants seem to take longer to reject the initial mismatch words. Post-hoc pairwise comparisons (Bonferroni adjusted) showed that, for both aphasic participant groups, RTs in the pseudoword conditions did not differ for the two mismatch positions.

Aphasic participants' results related to verbal short-term memory

As mentioned in the introduction, poor performance on sublexical tasks (e.g., discrimination) reflecting impaired auditory memory in Broca's and Wernicke's aphasic patients can be caused either by lesions involving posterior regions (such as the superior temporal gyrus), or by lesions in more frontal regions (such as Broca's area) involved in verbal short-term memory. Thus lesions in either location may still have the same result: sound information that does not contact (or mismatches!) the lexicon fades relatively quickly. The importance of verbal short-term memory was investigated by relating aphasic patients' lexical decision performance to their performance in tasks tapping verbal short-term memory. All aphasic patients (except fluent patient 11) also performed the standardised auditory discrimination task of the PALPA test battery (PALPA 1: discrimination of non-word pairs; Dutch version; Bastiaanse et al., 1995). This task taps the ability to keep auditory information, particularly pairs of non-words, in short-term memory. In Table 3 each aphasic participant's individual performance is given for lexical decision and non-word discrimination. As in other studies, discrimination performance (PALPA 1 performance) did not differ between the fluent and non-fluent aphasic participants ($t < 1$, *ns*), nor between the subgroups of Broca's and Wernicke's patients ($t < 1$, *ns*).

The correlation strength was computed (over all 21 aphasic patients) between patients' performance on PALPA 1 (auditory discrimination of non-word pairs) and accuracy rate collapsed over all mismatch pseudoword items. The relationship between performance on these two tasks was weak (Spearman's $\rho = 0.22$, *ns*): only 5% of the variance in mismatch accuracy could be explained by discrimination performance.

Even though this was not planned a priori, accuracy rate was also correlated with Token Test performance. Given that PALPA performance did not correlate significantly with performance in the present study, correlation strength between Token Test score and pseudoword accuracy rate was investigated as well, because the Token Test is more taxing in terms of auditory verbal short-term memory. This Token Test measure (also in Table 3) was available from each aphasic patient's language therapy record (as part of the Aachen Aphasia Test results). However, the correlation between the Token Test and sensitivity to mismatch was not significant either (Spearman's $\rho = -0.343$, $p > .1$). If we focus specifically on performance for the initial mismatch items, and relate that to Token Test error score, the correlation fails to reach significance when Bonferroni correction for multiple comparisons is applied (for two comparisons: $\alpha = 0.05/2 = 0.025$; Spearman's $\rho = -0.476$, $p = .029$).

It is important to note that these weak correlation coefficients fit in with the experimental results on mismatch position. Any account stressing the role of the speed with which sound information fades would predict that aphasic participants would be relatively more impaired on the initial mismatch than on the final mismatch pseudoword conditions, compared to the control participants. There was

TABLE 3
Individual performance

<i>Participant (aphasia syndrome in brackets)</i>	<i>Non-word discrimination accuracy (PALPA 1)</i>	<i>Token Test (no. of errors; max=50)</i>	<i>Lexical decision accuracy: Collapsed over all pseudowords</i>	<i>Lexical decision accuracy: Initial mismatch</i>	<i>Lexical decision accuracy: Final mismatch</i>
NF1 (global)	.97	45	.95	.90	1.00
NF2 (global)	.81	40	.91	.85	.98
NF3 (Broca)	.93	25	.96	.93	1.00
NF4 (Broca)	.92	23	.75	.73	.78
NF5 (Broca)	.89	17	.86	.88	.85
NF6 (Broca)	.75	48	.73	.63	.83
NF7 (Broca)	.85	37	.91	.83	1.00
NF8 (global)	.79	50	.54	.53	.55
F1 (Wernicke)	.86	37	.84	.80	.88
F2 (Wernicke)	.94	42	.71	.68	.75
F3 (Wernicke)	.96	22	.88	.88	.88
F4 (Wernicke)	.92	37	.86	.88	.85
F5 (Wernicke)	.75	37	.86	.80	.93
F6 (amnesic)	.89	26	.98	.95	1.00
F7 (amnesic)	.99	27	.71	.63	.80
F8 (amnesic)	.93	33	.88	.80	.95
F9 (Wernicke)	.82	36	.83	.78	.88
F10 (Wernicke)	.82	45	.69	.53	.85
F11 (amnesic)	NA	24	.95	.93	.98
F12 (mixed)	.90	17	.84	.83	.85
F13 (Wernicke)	.88	35	.85	.78	.93

Each aphasic participant's individual performance: PALPA 1 (discrimination of non-word pairs; proportion correct), Token Test (number of errors), and lexical decision accuracy for the pseudowords (proportions correct).

some evidence for this in the RT data: aphasic listeners were relatively slow to respond NO to the initial mismatch pseudowords. Whereas the control listeners were faster on the initial mismatch pseudowords than on the final mismatch words, this RT difference between conditions was smaller for the aphasic listeners. Yet there was no indication of a Participant Group (aphasic participants vs controls) by Mismatch Position interaction in the pseudoword accuracy data: all groups made more errors to initial than to final mismatch pseudowords. Thus verbal short-term memory plays a role in explaining the data, but it does not provide a complete account.

DISCUSSION

This study was set up to investigate the effect of phonemic mismatch on lexical activation in adult aphasic listeners. If the process of mapping incoming sound to the lexicon is (slightly) disturbed in aphasia, lexical activation might be affected less by phonemic mismatch, compared to unimpaired control listeners. The accuracy data clearly showed that the aphasic participants are less sensitive to phonemic mismatch in pseudoword conditions than control non-brain-damaged listeners. Thus, the process of mapping sound to the lexicon is disturbed, such that lexical activation upon presentation of pseudowords is higher than actually appropriate. This difference between aphasic and control participants was particularly exaggerated

for the minimal phonemic mismatches, which can be explained as a mismatch salience effect. In other words, in the presence of “overwhelming” evidence for one particular lexical item, mismatching information decreases the amount of lexical activation of the target word to a lesser extent in aphasic listeners than in control listeners, but this is mainly the case for the minimal mismatch pseudowords.

However, there was no evidence (neither in the accuracy data nor in the RTs) to support the more specific hypothesis concerning differences between aphasia types. Based on earlier results, the pseudowords were expected to yield “overactivation” for the Wernicke’s aphasic patients and “underactivation” for the Broca’s aphasic patients, relative to the control participants. However, no such differences were found, regardless of classification of the patients into fluent vs non-fluent groups or into Broca’s vs Wernicke type groups. Even though a number of lexical activation studies have been able to find (such) differences (Janse, 2006; Milberg et al., 1988; Yee et al., 2004), certainly not all studies found different patterns for the two aphasic syndromes (Baum, 1997; Janse, 2005). In an auditory lexical decision task with non-words differing in neighbourhood density (Janse, 2005), no differences between Broca’s and Wernicke’s patients were found either. This absence of differences between the aphasia syndromes also means that no link can be found between lexical activation and syndrome-specific comprehension problems.

It could be argued that there may have been an underlying lexical activation difference between Broca’s and Wernicke’s that did not show up due to limited statistical power. However, given that there was no trend towards different patterns either, this does not seem very likely. Furthermore, Broca’s and Wernicke’s aphasic patients’ equally poor ability to maintain sound representations in store cannot fully explain the present results. Despite different underlying causes and processes (Hickok & Poeppel, 2004), Broca’s and Wernicke’s aphasic patients are generally equally impaired on sublexical tasks involving the ability to maintain sound representations in store (Blumstein, Cooper, Zurif, & Caramazza, 1977; Miceli et al., 1980). The data did indeed show a relation between aphasic patients’ lexical decision accuracy and their Token Test performance, mainly for the pseudowords with a mismatching phoneme in word-initial position. If the initial fragment does not leave a stable sound trace to go back to, the fact that the initial phoneme did not provide bottom-up support for the real-word target can quite easily be overruled by later arriving “convincing” evidence for this one specific lexical item. However, the fact that the mismatch position effect was not stronger for the aphasic participant population, compared to their controls, shows that strong lexical resemblance led to “overactivation” in fluent and non-fluent aphasic patients, regardless of mismatch position. This result had been anticipated mainly for the Wernicke’s aphasic patients, given earlier evidence of their impaired inhibition/deactivation abilities of once-activated items (Janse, 2006; Wiener, Connor, & Obler, 2004).

The present results, suggesting overactivation rather than underactivation in Broca’s aphasic patients evidenced by their false acceptance of mispronounced words, yield an interesting discrepancy with earlier results. These earlier lexical activation studies have emphasised the vulnerability of Broca’s aphasic patients to subphonetic variation, yielding underactivation (Misiurski et al., 2005; Utman et al., 2001). Importantly, this was found only in conditions of competition (VOT-manipulated *c*oat* having the competitor *goat*). The present study specifically focused on situations in which there is no competition, because the use of longer polysyllabic words entailed that only one possible lexical candidate is left. It seems therefore that the present results, rather than

conflicting with these earlier ones, complement the earlier data. Whereas Broca's aphasic participants may initially face underactivation when mapping sound to the lexicon, this may change once there is converging evidence for one particular candidate. The Broca's results may be viewed in the light of insights from neuroimaging studies. Thompson-Schill (2005) hypothesised Broca's area to guide selection among competing sources of information. It is argued that selection of a representation may proceed entirely on the basis of bottom-up information, but in case of many weakly activated representations or one prepotent representation, top-down intervention is required to resolve the conflict among active representations in working memory. Thompson-Schill (2005) suggests that prefrontal cortex (or Broca's area) sends a modulatory signal to resolve selection in such situations. Thus, activity in Broca's area is increased by increasing demands to select one representation among competing representations (Fletcher, Shallice, & Dolan, 2000; Thompson-Schill et al., 2002). In the present study presentation of the pseudoword yields activation of the real word it mismatches. This real-word representation may be prepotent and therefore difficult to reject if prefrontal cortex is lesioned. Note that lesion data of the aphasic patients were not available, but the present account shows how "overactivation" can also result from more frontal lesions. Thus, whereas "overactivation" of the lexicon had been found to be characteristic of Wernicke's aphasia syndrome (Janse, 2006; Milberg et al., 1988), it may follow up on initial underactivation in Broca's aphasia syndrome in those conditions in which there is converging evidence for only one specific lexical item.

The present results therefore raise the question of how lexical activation builds up for these longer pseudowords in which the degradation is not subtle but even involves a phonemic "error". In other words, the time course of lexical activation upon presentation of mismatch non-words, such as *procodile* or *cabineq*, would be an interesting issue for investigation. A more direct and continuous measure of lexical activation, such as the eye-tracking paradigm (Tanenhaus, Magnuson, Dahan, & Chambers, 2000), might be a better choice than lexical decision and than, for example, a semantic priming paradigm with target presentation at various points from word onset. As noted in the method section, lexical decision also has its disadvantages because it may involve processes that are not necessarily involved in spoken word processing. Listeners became very much aware of possible phonemic changes and were thus extra cautious in their responses. An additional point to take into account in future research is to compare lexical activation elicited by the pseudoword *procodile* to that of the target word *crocodile* itself, rather than to nonsense items that resemble no lexical item in particular, as was done in the present study. Even though this had the obvious advantage that the comparison concerned non-words only, the nonsense items introduced their own peculiarities into the response time analyses.

In sum all groups, both aphasic and control participants, were clearly affected by the acoustic/perceptual difference between the changed and canonical word form: the less salient the mismatch to the target word, the more likely it is that the mismatching information goes unnoticed. The data have also shown that aphasic participants are less sensitive to phonemic mismatch than non-brain-damaged control participants: lexical activation upon presentation of speech material may over-represent the goodness of fit to a certain target representation. Because of impaired deactivation upon mismatch, longer pseudowords yield overactivation of the lexicon in the aphasia results. Note, however, that overactivation was mainly

found if the mismatch was minimal, such that the impairment in mapping sound to the lexicon seen in the aphasic participants was not so severe. Future research should preferably focus on the continuous build-up of lexical activation for both Broca's and Wernicke's aphasic patients.

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APPENDIX A

COMPLETE LIST OF NON-WORD ITEMS

<i>Initial mismatch</i>		<i>Final mismatch</i>	
<i>Pseudoword</i>	<i>Nonsense item</i>	<i>Pseudoword</i>	<i>Nonsense item</i>
<i>Minimal mismatches in unstressed syllable</i>			
1 figarét (sigaret)	firegát	41 notárig (notaris)	tamórig
2 banoráma (panorama)	bamalóna	42 pantóffen (pantoffel)	tunpéffen
3 symnastiek (gymnastiek)	simtiesnáak	43 salárif (salaris)	losóerif
4 sfekuláas (speculaas)	sfelakúus	44 karbonábe (karbonade)	durgokábe
5 pelefóon (telefoon)	pegoléen	45 mayonáite (mayonaise)	nomijáite
6 zitámíne (vitamine)	zinitáme	46 balustrábe (balustrade)	stubrílábe
7 tersonéel (personeel)	ternosáal	47 artiker (artikel)	kitróter
8 banuscript (manuscript)	baskritnúp	48 jerúzalep (Jeruzalem)	zujéurelep
9 rocomotief (locomotief)	romietoróf	49 horlóve (horloge)	larmóve
10 barallél (parallel)	barugél	50 theátel (theater)	jeekáutel
<i>Maximal mismatches in unstressed syllable</i>			
11 pournalist (journalist)	pourlisnáat	51 petróleus (petroleum)	tiprámajus
12 gaatschappij (maatschappij)	gaatvliemáap	52 kabóutem (kabouter)	godéitem
13 mokoláde (chocolade)	molakédo	53 compúter (computer)	moenkáatep
14 gateriáal (materiaal)	gatelióom	54 zigéunep (zigeuner)	hazáunep
15 nortemonnée (portemonnee)	norpiemanáu	55 karáktef (karakter)	mikrógtet
16 ribliothéek (bibliotheek)	ritobelóok	56 acquáriut (aquarium)	wakriajut
17 targaríne (margarine)	tarniráge	57 muséur (museum)	sanúujer
18 sonopólie (monopolie)	sotielóna	58 septémbes (september)	tuupóobes
19 pelicópter (helicopter)	petupéktem	59 obstákem (obstakel)	spatóokem
20 bymfonie (symfonie)	bitunafúu	60 bijzóndek (bijzonder)	vuubiendek
<i>Minimal mismatches in stressed syllable</i>			
21 lísico (risico)	líkosee	61 bioscóot (bioscoop)	bogikóot
22 gólibrie (kolibri)	góruubla	62 acrobáak (acrobaat)	torabáak
23 fáprika (paprika)	fákarip	63 appartemémp (appartement)	meteppomémp
24 finaasappel (sinaasappel)	fiepelanno	64 frikandén (frikandel)	krafidén
25 wárbecue (barbecue)	wárjoekep	65 bouleváal (boulevard)	luubeváal
26 skádion (stadion)	skánodi	66 neurolóof (neuroloog)	mazilóof
27 dróccoli (broccoli)	dróngila	67 krokodír (krokodil)	geufadír
28 nóminee (dominee)	nónamo	68 institúus (instituut)	sepmatúus
29 dédminton (badminton)	déttonuum	69 deodoránk (deodorant)	boosiedeeránk
30 fénior (senior)	féeroju	70 paradíjt (paradijs)	reupedíjt
<i>Maximal mismatches in stressed syllable</i>			
31 nángoeroe (kangoeroe)	nánnuigoo	71 microfóog (microfoon)	plomifóog
32 púcifer (lucifer)	púuselié	72 kapitéif (kapitein)	zielotéif
33 sáration (marathon)	sátorran	73 envelón (envelop)	vomuilon
34 júngalow (bungalow)	júnrowak	74 kabinéng (kabinet)	bogienéng
35 séduwe (weduwe)	séwaduú	75 kathedráap (kathedraal)	soekedráap
36 tórizon (horizon)	tóllozap	76 katholiém (katholiek)	tofelíem
37 déstival (festival)	déslatuuf	77 exempláak (exemplaar)	kesmiepláak
38 tándicap (handicap)	ténpakiep	78 kampióeg (kampioen)	poemiwóeg
39 hájesteit (majesteit)	háaweuskal	79 architésp (architect)	jierotésp
40 fúmmikub (rummikub)	fúmbukkan	80 republíer (republiek)	borreblier

Stressed syllable is indicated by a stress mark and each pseudoword has the word from which it is derived in brackets.

APPENDIX B

ANOVA ANALYSES ON LEXICAL DECISION ACCURACY TO NON-WORDS

	<i>Analysis by participant</i>	<i>Analysis by item</i>
Participant Group (control, non-fluent, fluent)	$F1(2, 30)=9.57, p=.001$	$F2(2, 71)=65.93, p<.001$
Non-word Type (pseudoword vs nonsense item)	$F1(1, 30)=88.64, p<.001$	$F2(1, 72)=91.01, p<.001$
Mismatch Position (initial vs final)	$F1(1, 30)=32.68, p<.001$	$F2(1, 72)=5.18, p=.026$
Mismatch Type (minimal vs maximal)	$F1(1, 30)=156.75, p<.001$	$F2(1, 72)=9.73, p=.003$
Stress (mm in stressed or unstressed syllable)	$F1(1, 30)=15.52, p<.001$	$F2(1, 72)=4.28, p=.042$
Participant Group * Non-word Type	$F1(2, 30)=8.92, p=.001$	$F2(2, 71)=67.31, p<.001$
Participant Group * Mismatch Position	$F1(2, 30)<1, ns$	$F2(2, 71)=3.15, p=.049$
Participant Group * Mismatch Type	$F1(2, 30)=5.13, p=.012$	$F2(2, 71)=2.54, p=.086$
Participant Group * Stress	$F1(2, 30)<1, ns$	$F2(2, 71)<1, ns$
Non-word Type * Mismatch Position	$F1(1, 30)=34.18, p<.001$	$F2(1, 72)=4.46, p=.038$
Non-word Type * Mismatch Type	$F1(1, 30)=152.41, p<.001$	$F2(1, 72)=12.32, p=.001$
Non-word Type * Stress	$F1(1, 30)=17.71, p<.001$	$F2(1, 72)=5.11, p=.027$
Mismatch Position * Mismatch Type	$F1(1, 30)=7.00, p<.001$	$F2(1, 72)=5.01, p=.028$
Mismatch Position * Stress	$F1(1, 30)=2.94, p=.097$	$F2(1, 72)<1, ns$
Mismatch Type * Stress	$F1(1, 30)<1, ns$	$F2(1, 72)<1, ns$
Participant Group * Non-word Type * Mismatch Position	$F1(2, 30)<1, ns$	$F2(2, 71)=3.20, p=.047$
Participant Group * Non-word Type * Mismatch Type	$F1(2, 30)=5.66, p=.008$	$F2(2, 71)=4.20, p=.019$
Participant Group * Non-word Type * Stress	$F1(2, 30)<1, ns$	$F2(2, 71)<1, ns$
Participant Group * Mismatch Position * Mismatch Type	$F1(2, 30)<1, ns$	$F2(2, 71)<1, ns$
Participant Group * Mismatch Position * Stress	$F1(2, 30)=1.40, ns$	$F2(2, 71)=1.63, ns$
Participant Group * Mismatch Type * Stress	$F1(2, 30)=2.25, p>.1$	$F2(2, 71)<1, ns$
Non-word Type * Mismatch Position * Mismatch Type	$F1(1, 30)=74.87, p<.001$	$F2(1, 72)=7.14, p=.009$
Non-word Type * Mismatch Position * Stress	$F1(1, 30)<1, ns$	$F2(1, 72)<1, ns$
Non-word Type * Mismatch Type * Stress	$F1(1, 30)<1, ns$	$F2(1, 72)<1, ns$
Mismatch Position * Mismatch Type * Stress	$F1(1, 30)=17.03, p<.001$	$F2(1, 72)=2.42, ns$
Participant Group * Non-word Type * Mismatch Position * Mismatch Type	$F1(2, 30)<1, ns$	$F2(2, 71)<1, ns$
Participant Group * Non-word Type * Mismatch Position * Stress	$F1(2, 30)<1, ns$	$F2(2, 71)=1.33, ns$
Participant Group * Non-word Type * Mismatch Type * Stress	$F1(2, 30)=2.72, p=.082$	$F2(2, 71)=1.98, ns$
Participant Group * Mismatch Position * Mismatch Type * Stress	$F1(2, 30)=1.31, ns$	$F2(2, 71)<1, ns$
Non-word Type * Mismatch Position * Mismatch Type * Stress	$F1(1, 30)=11.92, p=.002$	$F2(1, 72)=2.29, ns$
Participant Group * Non-word Type * Mismatch Position * Mismatch Type * Stress	$F1(2, 30)<1, ns$	$F2(2, 71)<1, ns$

Significant interactions involving Participant Group are highlighted.

APPENDIX C

ANOVA ANALYSES ON RESPONSE TIMES TO NON-WORDS

	<i>Analysis by participant</i>	<i>Analysis by item</i>
Participant Group (NBD control, non-fluent, fluent)	$F(1, 30)=16.05, p<.001$	$F(2, 71)=1571.32, p<.001$
Non-word Type (pseudoword vs nonsense item)	$F(1, 30)=169.81, p<.001$	$F(2, 72)=171.88, p<.001$
Mismatch position (initial vs final)	$F(1, 30)<1, ns$	$F(2, 72)=1.79, ns$
Mismatch Type (minimal vs maximal)	$F(1, 30)=31.85, p<.001$	$F(2, 72)=12.84, p=.001$
Stress (mismatch in stressed or unstressed syllable)	$F(1, 30)=8.46, p=.007$	$F(2, 72)=1.92, ns$
Participant Group * Non-word Type	$F(2, 30)<1, ns$	$F(2, 71)=2.32, ns$
Participant Group * Mismatch Position ⁵	$F(2, 30)=6.96, p=.003$	$F(2, 71)=22.74, p<.001$
Participant Group * Mismatch Type	$F(2, 30)=1.04, ns$	$F(2, 71)<1, ns$
Participant Group * Stress	$F(2, 30)=1.58, ns$	$F(2, 71)=2.08, ns$
Non-word Type * Mismatch Position	$F(1, 30)=3.26, p=.081$	$F(2, 72)=2.22, ns$
Non-word Type * Mismatch Type	$F(1, 30)=1.47, ns$	$F(2, 72)<1, ns$
Non-word Type * Stress	$F(1, 30)<1, ns$	$F(2, 72)<1, ns$
Mismatch Position * Mismatch Type	$F(1, 30)=4.11, p=.052$	$F(2, 72)=2.32, ns$
Mismatch Position * Stress	$F(1, 30)=1.38, ns$	$F(2, 72)<1, ns$
Mismatch Type * Stress	$F(1, 30)=1.80, ns$	$F(2, 72)<1, ns$
Participant Group * Non-word Type * Mismatch Position	$F(2, 30)=6.36, p=.005$	$F(2, 71)=15.66, p<.001$
Participant Group * Non-word Type * Mismatch Type	$F(2, 30)=1.18, ns$	$F(2, 71)=1.14, ns$
Participant Group * Non-word Type * Stress	$F(2, 30)<1, ns$	$F(2, 71)<1, ns$
Participant Group * Mismatch Position * Mismatch Type	$F(2, 30)<1, ns$	$F(2, 71)<1, ns$
Participant Group * Mismatch Position * Stress	$F(2, 30)=1.10, ns$	$F(2, 71)=1.13, ns$
Participant Group * Mismatch Type * Stress	$F(2, 30)<1, ns$	$F(2, 71)<1, ns$
Non-word Type * Mismatch Position * Mismatch Type	$F(1, 30)=1.09, ns$	$F(2, 71)<1, ns$
Non-word Type * Mismatch Position * Stress	$F(1, 30)=11.12, p=.002$	$F(2, 71)=1.61, ns$
Non-word Type * Mismatch Type * Stress	$F(1, 30)=7.90, p=.009$	$F(2, 72)=2.22, ns$
Mismatch Position * Mismatch Type * Stress	$F(1, 30)=18.95, p<.001$	$F(2, 72)=6.86, p=.011$
Participant Group * Non-word Type * Mismatch Position * Mismatch Type	$F(2, 30)<1, ns$	$F(2, 71)=1.43, ns$
Participant Group * Non-word Type * Mismatch Position * Stress	$F(2, 30)<1, ns$	$F(2, 71)<1, ns$
Participant Group * Non-word Type * Mismatch Type * Stress ⁶	$F(2, 30)=3.23, p=.054$	$F(2, 71)=3.80, p=.027$
Participant Group * Mismatch Position * Mismatch Type * Stress	$F(2, 30)<1, ns$	$F(2, 71)<1, ns$
Non-word Type * Mismatch Position * Mismatch Type * Stress	$F(1, 30)=9.47, p=.004$	$F(2, 72)=1.92, ns$
Participant Group * Non-word Type * Mismatch Position * Mismatch Type * Stress	$F(2, 30)<1, ns$	$F(2, 71)=1.09, ns$

Response times of non-brain-damaged controls, non-fluent aphasic and fluent aphasic patients. Significant interactions involving Participant Group are highlighted.

⁵ This interaction is not discussed in the paper because it is related to the three-way interaction of Participant Group $\times$ Non-word Type $\times$ Mismatch Position (which is discussed).

⁶ There was no a priori expectation about such an interaction, which turned out to be caused by specific differences between the two aphasic groups. These deviant response times in some specific cells of the design (for either the fluent or the non-fluent patients) could be related to two issues: either to the fact that the response time mean in the mismatch condition is not based on similar numbers of items across the listener groups (higher error rates in lexical decision), or because of the patients' relative slowness in responding to certain control items. No further account for this 4-way interaction is provided.