

THE ROLE OF THE OUTPUT PHONOLOGICAL BUFFER IN THE CONTROL OF SPEECH TIMING: A SINGLE CASE STUDY

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ABSTRACT

A patient with a selective impairment of speech output, associating phonemic paraphasias and pervasive preverbal pauses, is described. Experimental data are discussed in relation to current models of speech production. On clinical evaluation, the pattern of paraphasias was strongly suggestive of a functional lesion affecting the output phonological buffer. This hypothesis received substantial support from a study of word and non-word repetition, as well as from a study of picture naming latencies, with various imposed delays between stimulus presentation and vocal response. In order to determine the origin of the preverbal pauses, the patient was also submitted to other timed motor tasks implicitly probing higher levels of word production (gender decision and phonemic matching). We suggest that the pauses probably resulted from the conjunction of several mechanisms, including a general slowing apparent in all timed motor tasks, as well as a more specific time lag in speech initiation originating from the output buffer or from the articulator itself.

CASE REPORT

The fundamental manifestation of the language faculty is the ability to easily produce and understand continuous speech. The speech stream is normally constituted of an uninterrupted sequence of phonological units, with no acoustic discontinuity at word boundaries. Successive segments are produced at a fairly regular rate (Lehiste, 1977), which speakers are able to control and adjust depending on the immediate requirements of communication (Deese, 1984). However there is an apparent discrepancy between the ability to maintain a continuous, regular, and flexible speech output, and the complex psychological machinery that leads to overt speech production. Activation of successive words during sentence production does not occur serially but rather with some overlap between words. As demonstrated for instance by anticipatory speech errors, phonological encoding of a given word begins before processing of the foregoing words is over (e.g. Shattuck-Hufnagel, 1979). Moreover, each of the semantic, syntactic, lexical and phonological components which work together in language production is likely to obey specific timing constraints, which should not be expected to yield a steady and regular speech output. Finally, these various components are largely automatic and their operating speed is probably inaccessible to voluntary modulation (Fodor, 1983). How then should we account for the apparent contradiction between continuous, regular and controlled speech

output on the one hand, and discontinuous, independent and automatic speech production processes on the other hand?

Part of the answer is likely to come from a better understanding of the operation of the output phonological buffer. It is thought that just before being transmitted to the articulator for motor execution, phonological plan is stored temporarily in this buffering facility (Levelt, 1989). This allows the speaker to cope with asynchronies between formation of phonological plan and articulation rate. Additionally, phonological words stored in the buffer may be combined into a connected articulatory plan for entire phonological phrases, thus contributing to speech continuity (Levelt, 1989, p. 420; Bub, Black, Howell et al., 1987). Control over the output rate of the phonological buffer may also play a direct role in the regulation of speech rate, *inter alia* through the insertion of pauses.

A few patients with an impaired output phonological buffer have been reported previously. An overview of some of the most recent and carefully studied cases shows that all patients share a number of defining clinical features (Caramazza, Miceli and Villa, 1986; Caplan, Vanier and Baker, 1986; Bub et al., 1987; Belleville, Peretz and Arguin, 1992). Since words are assumed to be represented in the buffer as sequences of phonemes, an impairment on this level yields difficulties in correctly selecting and ordering the component phonemes. Therefore speech errors almost exclusively consist of phonemic paraphasias, phonologically close to the target phonemes (Caramazza et al., 1986). Similarly, patients with an impaired *graphemic* output buffer make errors in selecting and ordering the graphemes that compose the orthographic form of words (Caramazza et al., 1987). Errors affect all oral production tasks (naming, reading, repetition, etc.). Real words are better preserved than phonologically equivalent non-words, presumably resulting from the interaction between the lexicon and post-lexical phonological processing (Caramazza et al., 1986; Caplan et al., 1986; Bub et al., 1987). Such patients are generally classified as "reproduction" conduction aphasics, on the basis of the association of a preserved comprehension with phonological errors affecting repetition, among other speech production tasks (Shallice and Warrington, 1977). They usually display an associated reduction of their verbal span. It is plausible that in such cases span reduction results, at least partly, from the involvement of the output buffer in articulatory rehearsal during short term memory tasks (Baddeley, Thompson and Buchanan, 1975; Bub et al., 1987; Belleville et al., 1992). Finally, these patients are reported to have a reduced articulation rate, and to pause frequently between words in connected speech. This latter feature, which is generally mentioned as a marginal observation, will be discussed more specifically here.

In the present study, we report the case of a patient whose most salient anomalies in spontaneous speech consisted of phonemic paraphasias, associated with a general slowing resulting from frequent pauses she made between words. We will suggest that the patient's pattern of speech errors was suggestive of an impairment of phonological encoding, most probably affecting the output phonological buffer. The aim of the following experimental study will be twofold. First, the buffer hypothesis will be confronted with a series of repetition and timed naming experiments probing the time course of phonological encoding.

Second, we will discuss to what extent the general slowing of the patient's speech resulted from the impairment affecting her output phonological buffer, and eventually propose that other additional sources of slowing should also be considered in the present case.

CASE HISTORY

The patient was a 48-year-old right handed woman, who had had 15 years of schooling. She had been working as a translator from German and English into French, which was her mother tongue. She suffered a subarachnoid hemorrhage, revealing a large left parietal arteriovenous malformation. Following a subsequent attempt to artificially embolize a feeding artery, the patient suffered a recurrence of hemorrhage, accompanied by right sensory and motor deficit, and by almost complete muteness. The present study was carried out 2 years later. At that time, the patient still had severe right, mainly brachial, motor deficit. She could accurately imitate 5/6 simple bucco-facial gestures. Visual field was normal on confrontation, and language disorders had substantially receded.

LANGUAGE EXAMINATION

General Language Assessment

Spontaneous speech was rich and informative. The patient made use of complex and diversified syntactic structures. However, speech rate was reduced by frequent pauses occurring mainly between words. For instance, the patient took as long as 32 seconds to utter the sentence "J'ai repensé à la phrase que vous avez dite à propos de mon bras" (I have thought again about the words you said concerning my arm). The total duration could be broken down into 13 seconds of actual speech and 19 seconds of pauses between words. Additionally, the patient made phonemic paraphasias, some of which she attempted to correct.

The patient was submitted to a French version of the Boston Diagnostic Aphasia Examination (Goodglass and Kaplan, 1972; Mazaux and Orgogozo, 1982). The overall severity rating was 4. Verbal agility was reduced (score: 6/14). The patient scored at ceiling level in all subtests of auditory comprehension (word discrimination: 72/72, body-part identification: 20/20, commands: 15/15, complex ideational material: 12/12). Although she did not make a single error in the naming subtests, her scores were not quite perfect due to occasional delay before response onset (responsive naming: 24/30, confrontation naming: 98/105, naming of body-parts: 28/30). The patient produced 12 names of animals in the fluency in controlled association subtest. Automatic speech was correct (automatized sequences: 7/9, reciting: 1/2). Scores in repetition subtests were high despite rather frequent phonemic paraphasias and pauses during sentences (words: 9/10, high-probability sentences: 8/8, low-probability sentences: 8/8). This observation applies equally well to oral reading (words: 27/30, sentences: 7/10). The patient was perfect in all subtests of reading comprehension (symbol discrimination: 10/10, word recognition: 8/8, word-picture matching: 9/10, sentences and paragraphs: 10/10). Errors in comprehension of oral spelling (5/8) may be related to her severely reduced verbal memory span (digits: 3,

monosyllabic words: 4).¹ The patient could write quite effectively using her non-dominant left hand, and her scores in the writing subtests were very high (serial writing: 46/47, primer-level dictation: 14/15, spelling to dictation: 10/10, written confrontation naming: 10/10).

The patient scored 34/36 on the Token Test (De Renzi and Faglioni, 1978), confirming that speech comprehension was virtually normal.

The patient was presented with 80 line drawings printed on cardboard, selected from the pictures in Snodgrass and Vanderwart (1980). This set was made up of ten pictures belonging to each of 8 semantic categories (animals, tools, music instruments, body parts, vehicles, pieces of furniture, clothes, vegetables). The patient could flawlessly sort the pictures into the 8 categories, although she had not been informed of the number and identity of those categories.

The patient was presented with 115 simple line drawings for oral naming.² She made 14 errors (12%), of which 13 were phonemic paraphasias. The erroneous phoneme was generally close to the substituted phoneme (e.g. champignon [mushroom] → /sampignon/). As noted previously, naming onset was often preceded by a pause of abnormal length.

To summarize, the patient presented a selective impairment of verbal output, consisting of an association of phonemic paraphasias with abnormal pauses affecting connected speech as well as isolated words. Oral comprehension, reading and writing, were virtually normal.

Repetition of Words and Non-words

The above clinical assessment revealed that the patient met several diagnostic criteria for an impairment of the output phonological buffer. Similar patients generally display a strong advantage of words over non-words in a variety of verbal production tasks (Caramazza et al., 1986; Caplan et al., 1986; Bub et al., 1987). The aim of the following repetition test was to search for such a lexicality effect in the present case. Additionally, if the phonological buffer did not function properly, the patient's performance should deteriorate in proportion to the duration of the required storage. This prediction was tested by comparing immediate and delayed repetition.

Method

The experimental material consisted of 54 words and 54 non-words, matched with respect to syllabic length and initial phoneme. Stimuli were read aloud one by one by the experimenter to the patient and 5 control subjects matched for age and education. In the immediate condition, subjects were asked to repeat each item immediately. In the delayed condition, they were instructed to wait until the experimenter knocked on the table after a 4 second delay, and only then to repeat the stimulus. The block of 54 words as well as the block of

¹ During span assessment, elementary phonemic paraphasias leaving no doubt on the identity of the produced word were not scored as errors.

² These pictures were selected from among the 260 pictures in Snodgrass and Vanderwart (1980). The original set was presented to 15 normal subjects for naming. Those drawings for which there was perfect agreement among all subjects were digitized and used in the present study.

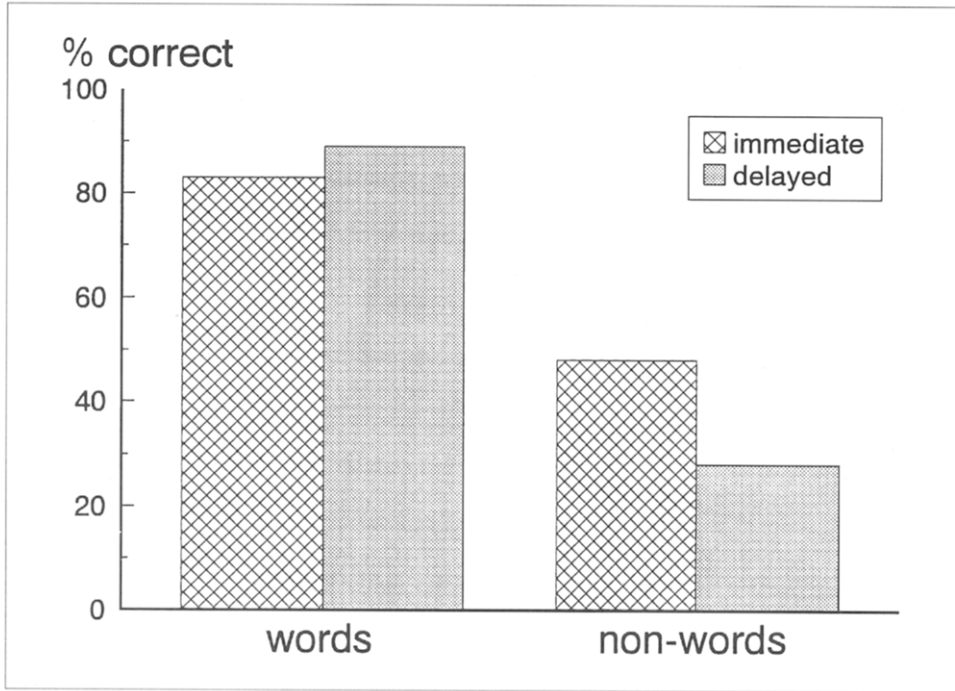


Fig. 1 – Percentage of correct responses in the repetition task. The patient produced significantly more errors with non-words than with real words. With non-words, performance level was significantly worse in the delayed than in the immediate condition.

54 non-words were successively presented in the immediate and in the delayed conditions.

Results

Controls made no errors over 540 trials with real words, and a total of 11/540 (2%) phonemic errors with non-words (2/270 [0.7%] in the immediate condition and 9/270 [3%] in the delayed condition). All conditions pooled, the patient made a total of 82 errors (40%). All errors were phonemic paraphasias similar to those produced in spontaneous speech and reading. The patient's performance showed two important features, apparent in Figure 1. First, non-words were significantly more error prone than words (67/108 [62%] and 15/108 [14%] errors, respectively; $\chi^2 = 53.15$; d.f. = 1; $p < 0.0001$). Only three out of 5 control subjects showed some tendency to make more errors with non-words than with words (difference in error rates: 1.9%, 2.7%, and 5.6%), albeit of an amplitude much smaller than the patient's 48% difference. Second, although the 4 second delay did not significantly modify the overall performance level ($\chi^2 = 0.96$; d.f. = 1), it affected differentially the repetition of words and non-words. With non-words, errors were significantly more frequent in the delayed than in the immediate condition (39/54 [72%] and 28/54 [52%] errors, respectively; $\chi^2 = 4.76$; d.f. = 1, $p = 0.029$), whereas these two conditions did not differ with real words (6/54 [11%] and 9/54 [17%] errors, respectively; $\chi^2 = 0.70$;

d.f. = 1). There was a small non-significant tendency for 3 out of 5 control subjects to show a similar effect of condition with non-words (difference in error rates: 3.7%, 3.7%, and 5.6%).

Discussion

The discrepancies observed here between words and non-words in terms of error rate, as well as with respect to the detrimental effect of delay, may seem at odds with the post-lexical character of the hypothesized buffer impairment. However, this observation is by no means an isolated finding, since comparable patients have consistently shown a similar advantage of words over non-words in repetition tasks (e.g. Caramazza et al., 1986; Caplan et al., 1986). During the repetition of non-words, the buffer presumably receives its input from a "surface" route directly translating input phonology into output phonology, with no intermediate lexical stage. When repeating real words, while this direct route may still be operative, it is supplemented by a lexical route allowing the buffer to receive its input from the phonological output lexicon. Thus the buffer stands at the confluence of the lexical and non-lexical routes, and it would seem natural that an impairment at this level should affect similarly the repetition of words and non-words. However, there are independent reasons to believe that the interactions of the lexicon and the buffer may be somewhat more complex. Spreading activation models of phonological encoding propose that any sequence of phonemes stored in the output buffer provides positive feedback to a number of neighbouring word nodes in the lexicon. These nodes reciprocally activate the corresponding phonemic sequences from top down (Levelt, 1989; Dell and O'Seaghdha, 1992). In particular, the phonemic sequence corresponding to a non-word activates a series of phonologically related words in the lexicon. These word nodes reciprocally tend to modify the sequence of phonemes in the buffer, yielding repetition errors through the activation of phonemes that are not present in the stimulus. In contrast, this phenomenon is of little consequence on the repetition of real words, due to the lateral inhibition of concurrent word nodes by the actual target word. This may explain why the repetition of real words should be easier than the repetition of non-words in normal subjects, and even more so in patients whose content of the output buffer is exceedingly fragile (see Caplan, 1992, p. 149).

More generally, and without commitment to any specific theory of phonological encoding, the advantage of words over non-words may simply result from the fact that non-words are only represented as transient phonological sequences, while real words also enjoy permanent lexical and semantic representations. Even if words and non-words are equally exposed to decay while stored in the phonological buffer, these stable higher level representations may act as a backup in the case of words, and help restore the adequate string of phonemes. In good agreement with our findings, this advantage of words over non-words may be expected to become larger with longer delays. Indeed long delays increase the probability of phonological deterioration, without substantially reducing the availability of these backup procedures only applying to real words.

Summary of Language Examination

It is now apparent that the main diagnostic criteria for an impairment of the output phonological buffer were met. First, all production tasks were affected irrespective of the input modality, indicating that the deficit was located in the verbal output system. Second, errors consisted of the replacement of target phonemes by phonologically close phonemes, suggesting that the impairment affected a phonological level of representation. Third, error rate could be increased by imposing a delay before overt production, confirming the lesion of a short term memory system. Fourth, real words were demonstrably more robust than non-words. Finally, our patient shared several related clinical characteristics with similar, previously reported, cases: A reduced verbal span, reduced articulation rate, and pervasive pauses before words. It must be stressed that these additional clinical features need not result from the buffer impairment itself. For instance, an additional impairment affecting an *input* phonological store may in principle contribute to the span reduction, and a slowed articulatory system may contribute to the speech flow abnormalities.

In the following section, we will further test the hypothesis of a buffer disfunction, using a series of timed speech production tasks.

EXPERIMENTAL STUDY

Before describing the experimental study, it may be useful to present a brief outline of the processes underlying word production, such as operating typically in picture naming. For instance, naming the picture of an apple may be analyzed into three successive stages (Levelt, 1989). First, the concept of an apple is activated, as a consequence of visual processing and identification of the picture. In a second stage, this conceptual representation triggers lexical access, making all information concerning the word "apple" available. Actually, there is evidence that different aspects of lexical information are not activated simultaneously but serially and in a fixed order (e.g. Schriefers, Meyer and Levelt, 1990). First come semantic features and syntactic information such as grammatical category and gender, constituting the lemma of the word. Second, the phonological characteristics of the word, or lexeme, are accessed (sequence of phonemes, syllabic structure, stress pattern, etc.). As mentioned before, the resulting phonological plan is stored in the output phonological buffer, to be eventually transferred to the articulator for motor execution, which constitutes the third and last stage of speech production. Although there is some disagreement as to the precise temporal course of these processes, there is reasonable consensus regarding their general architecture (Levelt, 1989; Dell and O'Seaghdha, 1992).

In the following experiments, we tried to tap selectively some of these processing levels. The patient's performance was systematically compared to that of the 5 matched controls mentioned above. The group of controls comprised 3 women and 2 men, all right handed, with an age ranging from 43 to 52 years, and an education level comparable to the patient's (15 to 18 years of education).

Experiment 1: Timed Picture Naming I

In this experiment, we studied the influence on naming latencies of imposing an obligatory delay between stimulus presentation and overt naming. It has been shown previously in normal subjects that picture naming latencies are significantly longer if subjects are requested to respond immediately than if they have to respond on a subsequent "go" signal (Klapp, Anderson and Berrian, 1973). It is thought that when producing their response in the delayed condition, subjects do not have to go through all the early stages of the naming task. They have ample time to formulate a phonological plan and store it in their output buffer. Upon seeing the "go" signal, they simply transfer the available phonological plan from the buffer to the articulator, and produce the corresponding sounds (Levelt, 1989).

Thus the beneficial effect of delay on naming times directly results from the operation of the output buffer. If indeed the patient's buffer did not function normally, an imposed delay preceding the vocal response may fail to yield a normal reduction of reaction times. The outcome of the repetition task reported before may help us specify further our prediction. After a 4 second delay, real words were repeated as accurately as when no delay was imposed, while the repetition of non-words deteriorated severely over this delay. We suggested that in the delayed condition, and with real words only, the patient could avoid using the deteriorated phonological plan stored in her buffer by engaging again in a complete phonological encoding on the basis of a higher level representation of the target word. If this hypothesis is correct, then the patient should behave identically whether a 4 second delay intervenes or not. In other terms, the patient should not benefit from the normal latency advantage that normal subjects are expected to show when naming with a 4 second delay, as compared to immediate naming.

Method

Thirty pictures were chosen from the preselected corpus of 115 unambiguous line drawings. Since it has been suggested that the number of syllables may affect differentially immediate and delayed naming latencies (Klapp et al., 1973), the material was controlled for length. The set of names corresponding to the selected pictures comprised 10 mono, 10 bi, and 10 trisyllabic words. The 3 classes of words were matched with respect to the phonological category of their initial phoneme, as well as to frequency. The block of 30 drawings was presented twice for delayed naming, and then twice for immediate naming. Presentation order was randomized within each block. Each target picture was displayed for 1500 msec. In the immediate condition, subjects were instructed to name the depicted item as fast as possible. In the delayed condition, they had to refrain from overt naming. After a subsequent 4000 msec blank screen, a black square appeared, indicating that subjects had to respond as fast as possible. In both conditions, response was followed by a 1500 msec blank screen, preceding the next trial. Pictures were presented in black on white, on a NEC Multisync 2A screen controlled by a Toshiba T-5200 computer. The microphone was connected to an OROS AU-22 digital board programmed to function as a voice key, which measured naming time to the nearest millisecond. All testing sessions were recorded for subsequent analysis of errors.

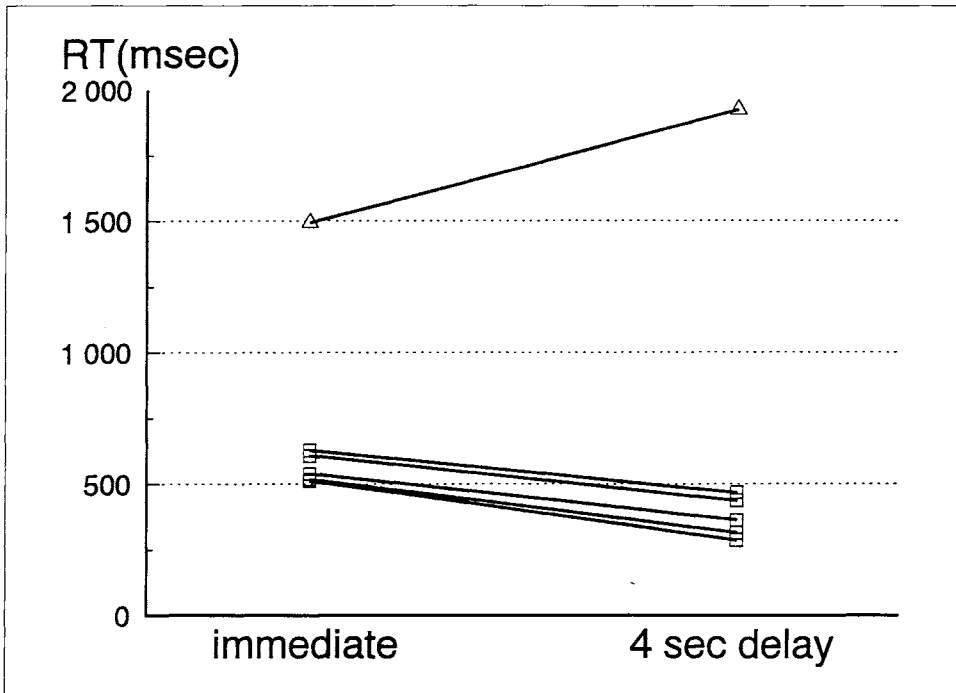


Fig. 2 – Mean picture naming latencies for the patient (triangles) and control subjects (squares) in Experiment 1. The patient was significantly slower than controls, and did not display the normal latency difference between immediate naming and 4 second delayed naming.

Results

Reaction times corresponding to the production of an erroneous word, to phonemic paraphasias affecting the initial phoneme or at least any two phonemes, to a stuttering answer, to a self correction, to a technical malfunction, or differing by more than 2 SD from the mean for the considered subject and experimental condition, were excluded from the analyses. This procedure led to the rejection of 28 values for the patient (23%) and 35 values for control subjects (6%). In the patient, an almost equal proportion of responses were rejected in the immediate and in the delayed condition (12/60 and 16/60, respectively, $\chi^2=0.42$; d.f.=1; $p=0.52$), preventing any problem of speed-accuracy trade-off in the interpretation of reaction times.

Mean latencies for the patient and control subjects are displayed in Figure 2. The controls' mean reaction time was 561 msec in the immediate condition (range: 511 msec to 629 msec), and 372 msec in the delayed condition (range: 286 msec to 465 msec). As could be expected, the patient was significantly slower than controls in both the immediate (mean = 1494 msec; $t=17.27$; d.f.=4, $p=0.0001$) and the delayed (mean = 1924 msec; $t=20.16$; d.f.=4; $p<0.0001$) conditions. In addition to this overall slowing, the patient's performance was also qualitatively abnormal. Control subjects were 189 msec faster in the delayed than in the immediate condition ($F=273.26$; d.f. = 1, 4; $p=0.0001$). The patient's

latencies did not obey this general tendency. Her results fell significantly off the distribution of difference scores of control subjects between immediate and delayed conditions ($t=24.26$; $d.f.=4$; $p<0.0001$). Actually, she even showed an opposite tendency, since she was 430 msec *slower* in the delayed condition ($t=1.63$; $d.f.=90$; $p=0.11$). Finally, it should be noted that no effect of syllabic length on mean reaction times was observed for the control subjects nor for the patient, in either immediate or delayed naming.

Discussion

In agreement with earlier studies, control subjects had shorter naming latencies in the delayed than in the immediate condition (Klapp et al., 1973). The fact that, as predicted, the patient did not show this pattern of performance is compatible with the idea of an impaired output buffer. If the phonological plan suffers a fast degradation while stored in the output buffer, it is natural that the longer the interval before articulation, the more severe the degradation. After a 4 second delay, the original phonological plan may be severely altered, inducing the patient to engage in a new naming procedure on the basis of some representation of the stimulus in memory. This procedure naturally produces reaction times, as well as error rates (just as in the repetition task), not different from immediate naming.

Experiment 2: Timed Picture Naming II

It may be objected that our interpretation of the patient's performance in Experiment 1 is essentially based on negative results, i.e. the absence of a normal latency difference between immediate and delayed naming. If, however, the account we have submitted is correct, this abnormal pattern prevailed only because the strip of phonological plan remained long enough in the buffer to be deteriorated to a degree justifying a complete new encoding from the lexicon. Therefore it should be possible to restore a normal beneficial effect of delay on latencies simply by reducing the 4 second delay to some shorter duration. If indeed the "go" signal is presented *before* the stored phonological plan becomes severely deteriorated, this plan should be used by the patient for overt utterance, just as it happens in normal subjects. In other words, resorting to a delay shorter than 4 seconds should restore a normal advantage of delayed over immediate naming in the patient. This prediction was tested in the present experiment by comparing immediate naming and naming with a short delay.

Method

Fifty line drawings were chosen from the corpus of 115 preselected pictures. The corresponding set of names comprised 20 monosyllabic, 20 bisyllabic and 10 trisyllabic words. All words had a consonant in initial position. The procedure was identical to Experiment 1, except that in the delayed condition, the "go" signal was preceded by a 2 second delay. The block of 50 drawings was first presented for delayed naming, and then for immediate naming.

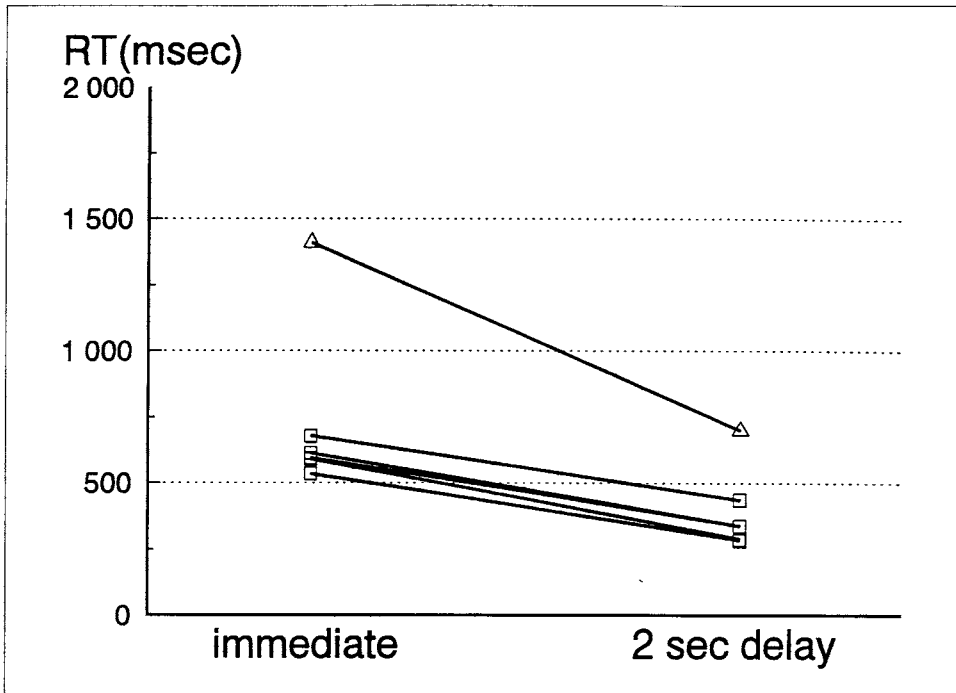


Fig. 3 – Mean picture naming latencies for the patient (triangles) and control subjects (squares) in Experiment 2. The patient was significantly slower than controls. However, she displayed the normal latency difference between immediate naming and 2 second delayed naming.

Results

Data were filtered according to the same principles as in Experiment 1, leading to the rejection of 36 values for the patient (36%) and 30 values for control subjects (6%). In the patient, an equal proportion of responses were rejected in the immediate and in the delayed condition (18/50 in both cases), preventing any problem of speed-accuracy trade-off in the interpretation of reaction times.

Mean latencies for the patient and control subjects are shown in Figure 3. The controls' mean reaction time was 601 msec in the immediate condition (range: 534 msec to 677 msec), and 337 msec in the delayed condition (range: 284 msec to 435 msec). The patient was significantly slower than controls in both immediate (mean = 1409 msec; $t = 15.76$; $d.f. = 4$; $p = 0.0001$) and delayed (mean = 699 msec; $t = 5.98$; $d.f. = 4$; $p < 0.005$) conditions. As in Experiment 1, controls were faster in the delayed than in the immediate condition ($F = 663.59$; $d.f. = 1, 4$; $p < 0.0001$). The patient, although slower than controls, nonetheless showed a significant advantage of delayed over immediate naming ($t = 7.715$; $d.f. = 62$; $p < 0.0001$). Comparing Experiments 1 and 2, it appeared that controls' mean latencies were not different between naming with a 2 second and with a 4 second delay ($F = 4.39$; $d.f. = 1, 4$; $p < 0.15$), whereas the patient was significantly slower in the latter condition ($t = 3.97$; $d.f. = 74$; $p = 0.0002$).

Discussion

In agreement with our prediction, the patient was significantly faster in 2 second delayed naming than in immediate naming, just as were normal subjects. It is important to stress that the apparently opposite influences of a 2 second delay and of a 4 second delay on latencies resulted in fact from a single impairment affecting the output buffer. After a 2 second delay, the phonological plan stored in the output buffer is still usable for articulation and the patient benefits from the same latency advantage as do normal subjects. However, after a 4 second delay, the phonological plan is too severely deteriorated, and naming latencies then return to the level of immediate naming. In sum, the pattern emerging from the comparison of immediate, 2 second delayed and 4 second delayed naming in Experiments 1 and 2 lends further support to the hypothesis of an impaired output phonological buffer.

However, this conclusion should not be taken to imply that *all* the abnormal time-dependent features of the patient's behavior, over and beyond the differential effect of delays on naming times, resulted from the buffer deficit. In particular, one should remember that the mean latency in immediate and in 4 second delayed picture naming was more than 1 second longer in the patient than in control subjects, and that a difference of about 400 msec was observed in the 2 second delayed condition. It is tempting to assume that this protracted silence preceding the onset of words is functionally equivalent to the pauses that pervaded the patient's connected speech. Four main possibilities should be considered in principle regarding the origin of this abnormal time lag. First, the delay may arise prior to the delivery of the phonological plan to the output phonological buffer. Second, it may result from an impairment affecting the buffer itself. Third, the starting of overt production may be abnormally delayed due to an impaired articulator. Finally, the hypothesis of a non-specific cognitive slowing affecting all timed motor tasks should be considered. In view of the deficit documented so far, the buffer hypothesis stands out as the most economical one. However this possibility should not be taken for granted so far. The following experiments aim at identifying other candidate sources of retardation upstream from the output buffer.

Experiment 3: Timed Gender Decision

The goal of this experiment was to evaluate the patient's speed of processing up to and including the first stage of lexical access in speech production, namely access to word lemmas. To this end, the patient was submitted to a timed gender decision task. In French, all nouns are either feminine or masculine. The grammatical gender of sexually neutral items is arbitrarily determined, and hence must be stored in the lexicon. Gender actually constitutes the only obvious grammatical feature that must be represented in the lemma of common nouns. We assumed that the only way to determine the gender of the name associated to a given picture is to go through all stages of the naming task, at least up to accessing the lemma. Gender decision may therefore be used as a probe to the grammatical stage of lexical access.

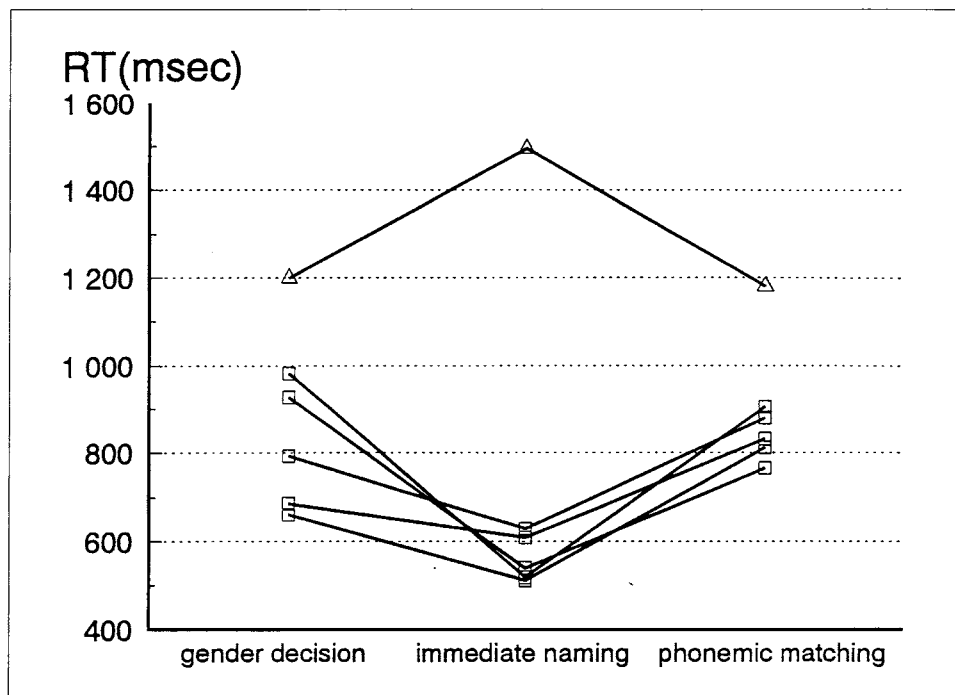


Fig. 4 – Mean latencies for the patient (triangles) and control subjects (squares) in Experiment 3 (left) and Experiment 4 (right). Immediate picture naming latencies from Experiment 1 are presented again for comparison (center). The patient was moderately slower than controls in grammatical decision and phonemic matching. Contrary to controls, she was faster in those tasks than in immediate naming.

Method

The patient was presented with a series of 30 line drawings chosen from the corpus of 115 preselected pictures. The corresponding names were masculine for half the pictures, and feminine for the other half. Names containing any morphological mark of gender were systematically avoided. Frequency and syllabic length were equated across the two gender categories.

Each picture was displayed on the computer screen for 3000 msec. Subjects were instructed to decide if the name of the depicted item was feminine or masculine, and to respond as fast as possible by tilting a joystick with their left hand to the right or to the left, respectively.

Results

Reaction times corresponding to an incorrect gender decision or differing from the subject's mean by more than 2 SD, which made up 10% of the data for the patient and 9% for control subjects, were excluded from the analyses. Results are presented in Figure 4.

The controls' mean reaction time was 810 msec (range: 660 msec to 982 msec). For the purpose of our demonstration, it is important to note that normal

subjects were significantly slower in this task than in immediate picture naming³ ($F = 11.11$; $d.f. = 1, 4$; $p = 0.029$). The patient's behavior was abnormal in two respects. First, she was marginally slower than controls (mean = 1199 msec; $t = 2.739$; $d.f. = 4$; $p = 0.052$). Second, she was significantly *faster* in the grammatical decision task than in immediate picture naming ($t = 2.52$; $d.f. = 73$; $p = 0.014$).

Discussion

In the present experiment, the patient was marginally slower than controls. This slowing may originate either from a processing stage upstream from access to gender information, or from some non-specific component of the classification task, such as decision making or motor response. At any rate, this non-significant slowing cannot account for the abnormal delay observed in immediate picture naming. First, the patient was more than 1000 msec slower than controls in picture naming, a difference obviously larger than the 400 msec difference obtained in the present experiment. Second, the fact that, in opposition to the controls, the patient was significantly slower in immediate naming than in grammatical decision directly points to some major slowing factor affecting the naming process downstream from lemma retrieval.

In short, although the patient was somewhat slower than controls in a task selectively tapping processing stages up to grammatical access, this difference cannot account for the preverbal time lag observed in immediate picture naming.

Experiment 4: Timed Phonemic Matching

Obedying the same logic as in Experiment 3, we tried to evaluate the patient's speed of processing up to and including the second stage of lexical access in speech production, namely retrieval of word lexeme. Lexemes are thought to specify the morphemic and syllabic structure of words, as well as their segmental composition, i.e. the sequence of constituent phonemes. We assumed that in order to determine the phonemic characteristics of the name associated to a given picture, one has to go through all stages of the normal naming task, at least up to accessing the lexeme. We therefore resorted to a timed phonemic matching task in order to probe the patient's access to the phonemic content of words, without eliciting overt speech production.

Method

The same 50 line drawings as in Experiment 2 were used. On each trial, the experimenter orally produced a syllable consisting of a consonant followed by a schwa, and subsequently triggered the display of a picture on the computer screen. On matching trials, the proposed consonant was the initial phoneme of the picture name. On non-matching trials, the proposed consonant was not correct, although it was always phonologically close to the actual initial phoneme. Subjects were asked to judge whether the proposed consonant and the picture

³ This comparison was made on the basis of immediate naming latencies obtained in Experiment 1. Data from Experiment 2 would yield exactly equivalent results.

were matching. They were instructed to respond as fast as possible upon seeing the picture, by tilting the joystick with their left hand to the right for matching trials and to the left for non-matching trials.

Results

Reaction times corresponding to an incorrect decision, or differing from the subject's mean by more than 2 SD, were excluded from the analyses, yielding the rejection of 11% of the data for the patient, and 9% for control subjects. Results are presented in Figure 4.

The controls' mean reaction time was 839 msec (range: 766 msec to 905 msec). They were significantly slower in this task than in immediate picture naming ($F = 83.22$; $d.f. = 1, 4$; $p = 0.0008$). Just as in Experiment 3, the patient's behavior was abnormal in two respects. First, she was slower than controls (mean = 1180 msec; $t = 6.19$; $d.f. = 4$; $p < 0.005$). Second, she was significantly faster in the phonemic matching task than in immediate picture naming ($t = 3.94$; $d.f. = 133$; $p = 0.0001$).

Discussion

The present pattern of results is in all relevant respects closely similar to that observed in Experiment 3. The patient was moderately but significantly slower than controls, but also faster than in immediate naming. We may therefore follow the same general argument as with grammatical decision, and safely conclude that at least most of the patient's abnormal time lag in immediate naming originated downstream from phonological access. However, it should be acknowledged that the phonemic matching task only allows for an incomplete evaluation of phonological access. It does not probe the retrieval of the complete phonological specifications of words, but only of their very first phoneme. It is in principle possible that an impaired access to further parts of the stored phonological records could contribute to the patient's slowing. Beside this possibility, the remaining candidates are logically the output buffer and the articulator.

Discussion of Experiments 1 to 4

The experiments we have reported aimed (1) at confronting the hypothesis of an impaired output phonological buffer with naming latency data, with a special emphasis on the influence of delay, and (2) at shedding some light on the origins of the general slowing of the patient's speech.

Since the first point was already discussed at length, it is sufficient to remember that the hypothesis of an impaired phonological buffer, which was first formulated following clinical language assessment and formal repetition tasks, received additional support from the influence of different delays on picture naming latencies (Experiments 1 and 2).

Regarding the second point, the origin of the patient's abnormal pauses preceding speech onset, apparent in spontaneous speech as well as in

Experiments 1 and 2, was explored using timed motor tasks implicitly probing higher levels of word production. Experiments 3 and 4 showed that the cascade of processing stages that lead from visual analysis up to and partly including phonological access to the output lexicon could not be taken as responsible for most of the pauses. However, the patient's mean latency exceeded the controls' by about 400 msec in both Experiments 3 and 4. Phonemic matching, although presumably tapping a later processing stage, did not reveal any additional slowing as compared to grammatical decision. It is tempting to suggest that the equivalent slowing affecting gender decision and phonemic matching resulted from a process common to both tasks, such as decision making or motor response. Some additional support to this hypothesis comes from Experiment 2. In the 2 second delayed naming task, the patient was about 400 msec slower than controls, just as in Experiments 3 and 4. To summarize, the patient's slowing may be considered as the conjunction of at least the two following factors: (1) a general 400 msec slowing affecting all manual or vocal timed motor tasks, a common observation with brain damaged patients (e.g. Benton, 1986), and (2) an additional slowing specifically affecting immediate naming (as well as the essentially equivalent 4 second delayed naming).

It is unfortunately difficult to elucidate precisely the mechanism that gave rise to the excessive time lag in immediate naming. We have suggested that an abnormally fast decay of the phonological plan in the buffer accounts for the fact that the patient was faster in 2 second delayed naming but not in 4 second delayed naming, as compared to immediate naming. However, this hypothesis does not explain why immediate naming should be slowed down in the first place. Actually, it is in immediate naming that the phonological plan is stored in the output buffer for the shorter duration, and that it should therefore suffer the least. Although our data do not provide an unambiguous answer to this question, we may formulate two plausible hypotheses. First, the output buffer may take an unduly long time to constitute a usable strip of phonological plan, that can be transferred to the articulator. This could result either from slowed feeding from the lexicon, or from some disfunction affecting the operation of the buffer itself. Second, there is evidence from normal subjects that before actual execution, motor programs must be retrieved and unpacked by the articulator. The retrieving and unpacking processes take measurable time, depending on the number and complexity of the required motor programs (Sternberg, Monsell, Knoll et al., 1978; Levelt, 1989; Wright, 1990). In brief, the patient's preverbal pauses in immediate naming may result from an abnormal operation of the buffer or of the articulator. Both hypotheses seem compatible with the results of Experiment 2, where it appeared that an additional 2 second delay allowed the patient to normalize her naming latencies, except for the general 400 msec slowing affecting all timed tasks.

CONCLUSION

We have reported the case of a patient presenting an isolated disorder of speech production consisting of phonemic paraphasias and pervasive preverbal

pauses. In the clinical section, we have shown that the pattern of errors was consistent with an impairment of the output phonological buffer. This hypothesis was further confirmed in the experimental section through the study of repetition and of picture naming latencies, with various imposed delays.

We have also tried to determine the functional origin of the patient's frequent preverbal pauses. These pauses probably resulted from the conjunction of several mechanisms that could only be partially disentangled. First, a slowing of about 400 ms was apparent in all timed motor tasks, and may have contributed to the overall delay in speech initiation. Second, a specific time lag prevailed in immediate naming. We suggested that its origin should be found in the operation of the output buffer or of the articulator itself. Third, and as a final point, we may speculate on a further source of slowing, namely the specific role of the phonological buffer in ensuring the continuity and fluency of connected speech. It has been suggested that, in normal speech, the tendency to produce phonological phrases of approximately equal duration may reflect constraints imposed by the size of the output buffer (Levelt, 1989, p. 386). A reduced or disfunctioning buffer would therefore naturally result in a severe reduction of the size of phonological phrases, down to the dimension of single words. In other terms, if the content of the buffer suffers an abnormally fast decay, it becomes impossible to prepare a ready-made phonological plan several words in advance. Each word has therefore to be produced from scratch, just at the moment when it must be uttered (Bub et al., 1987). This phenomenon may have contributed to the patient's preverbal pauses in continuous speech. However, our study was essentially limited to single word production tasks and such an hypothesis is still purely speculative. Further experimental work is needed in order to examine directly the timing of continuous speech in aphasic patients.

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