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Disorders of phonological encoding*

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Abstract

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Studies of phonological disturbances in aphasic speech are reviewed. It is argued that failure to test for error consistency in individual patients makes it generally improper to draw inferences about specific disorders of phonological encoding. A minimalist interpretation of available data on phonological errors is therefore proposed that involves variable loss of information in transmission between processing subsystems. Proposals for systematic loss or corruption of phonological information in lexical representations or in translation subsystems is shown to be inadequately grounded. The review concludes with some simple methodological prescriptions for future research.

Introduction

Normal speakers quite frequently make mistakes producing the intended sound of a word. They might, for example, say “corkical” instead of “cortical”, or “prostitute” instead of “Protestant”, or “Fats and Kodor” instead of “Katz and Fodor”, or “shrig souffle” instead of “shrimp and egg souffle” (Fromkin, 1973, Sections B, Q, C and V). Estimates of the incidence of such errors from recordings of normal conversation vary from 1.6 errors per 1000 words (Shallice & Butterworth, 1977) down to 62 (segment errors and haplogogies) in approximately 200 000 words of the London–Lund corpus (Garnham, Shillcock, Brown, Mill, & Cutler, 1981). In studies of aphasic speakers, these errors are called “literal” or “phonemic” paraphasias, and some, but not all, aphasic patients may make far more errors than normal speakers, although no comparable statistics for aphasic conversation are available. However, one patient described by Pate, Saffran, and Martin (1987) produced between 3% and 67% of phonemic paraphasias in

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spontaneous speech, with the variation depending on the length of the target (3% for one-syllable words, 67% for four-syllable words.) In picture-naming tests, which may be different in important ways from free speech, Howard, Patterson, Franklin, Morton, and Orchard-Lisle (1984) found that a sample of 12 patients produced between 0% and 14% of phonemic errors¹ in 1500 naming attempts per patient. Classically, frequent paraphasias in the patient's speech, combined with an impairment of repetition, has been the hallmark of a syndrome known as *conduction aphasia*. More recent research distinguishes repetition (short-term memory) capacity from the ability to reproduce accurately even single words (Shallice & Warrington, 1977).

It might be presumed, a priori, that sources of normal slips of the tongue are different from aphasic paraphasia; that aphasics, by hypothesis, suffer a deficit in one or more of the processes that lead from the thought to its expression in speech, whereas normals, by definition, do not suffer deficit. (See Caramazza, 1986, for the elevation of this definitional convenience into a metaphysical principle.)

To identify a *disorder* or deficit responsible for phonemic paraphasias, one needs to do two things. First, find a *consistent* pattern of deviation from the phonological norm in the responses of patients studied on a case-by-case basis: a small, but consistent deviation may be lost in group means, and even a large one may be cancelled out by an equally significant (interesting) deviation in the opposite direction. Second, show how these responses might result from a deficit to one or more subsystems of a proposed model of normal phonological encoding. Although we have models of the normal processes whose main features, if not well established, are at least widely held, the burden of this essay will be that the evidence currently available is inadequate for identifying specific disorders of phonological encoding, despite claims to the contrary, since the prerequisite of individual *consistent deviation* has rarely been satisfied in the existing studies.

By the term "phonological encoding" (henceforth PE), I shall mean those processes that intervene between ascertaining that there is a (single) word in the mental lexicon that can express the lexical intention or plan and the full phonetic description that realizes it. Of course, further processes will be required to turn this description into a motor plan for articulation, but these will not be dealt with here.

Intuitively, PE of a known word entails accessing a stored representation of the sound of that word, what I shall term the phonological lexical representation

¹Howard et al. (1984) were interested primarily in the variability of the ability to retrieve the target name, rather than in phonemic errors as such; they therefore treated single-segment deviations from the target as correct naming responses. Two deviations are required for the response to be counted as a phonemic paraphasia. Deviations that resulted in real words were treated as word substitution errors.

(PLR) ("lexeme" in Kempen and Huijbers' (1983) terminology). PLR has to contain sufficient information to specify, for the word in its intended speech context: (1) the syllabic structure of the word; (2) the stress pattern of the word; and (3) the segmental contents of the syllables. I leave aside entirely the question as to whether the PLR is an "underlying" or a "superficial" phonological representation (but see Caplan, 1987, for arguments in favour of an underlying representation). Following a number of authors, I will assume that these three types of information are represented separately (see Levelt, 1989, chapter 9, for a review). A set of processes must then *translate* the information stored in the PLR into the phonetic representation appropriate to the current speech context that is ultimately passed on to the articulators. Encoding subsystems "spell out", in Levelt's (1989) useful term, information in a PLR. One can think of a PLR as containing phonological information in a condensed or abbreviated form, which requires elaborating before it can be deployed by later processes. Elaboration may involve adding information on the basis of general rules of phonology, which the (normal) speaker may be assumed to know. For example, it may involve generating allophonic variants appropriate to the current syllabic context, like lengthening a vowel before a voiced obstruent. Unfortunately, we have insufficient evidence to be precise about how this spelling out might work. Allophonic variation may already be explicitly encoded in the PLR. PLRs may also be underspecified in a more technical sense; that is, some phonetic features will systematically not be represented in the PLR so that, for example, the consonant following /s/ in *start* will be marked for place but voicing is left unspecified, and is represented instead by an archiphoneme /T/ that could be realized as either /t/ or /d/. Other types of underspecification in underlying lexical representations have been suggested in phonological theory (e.g., Archangeli, 1985; Kiparsky, 1982), and some of the ideas have been recruited to explain normal speech errors (Stemberger & Treiman, 1986) and aphasic paraphasias (Beland, 1990).

I shall follow several authors (Levelt, 1989, chapters 8 and 9; Shattuck-Hufnagel, 1987) in assuming that translation processes have the form of a slot-and-filler device. The slots are defined jointly by spelling out the syllabic structure (how many syllables, and their form) and spelling out the prosodic structure – the stress (and pitch, where relevant) of each syllable. Information about the segmental content is spelled out and inserted into the appropriate slots. (This account is a considerable simplification, as well as a slight modification, of Levelt's in several ways, and the reader is urged to consult his book for a fuller description of the processes that might be at issue.)

The reader may also note similarities between the slot-and-filler model, and an earlier model proposed by Shaffer (1976). In this model, a "structural representation", derived from lexical representations, contains coordinated information

concerning syllabic position and segments, where segments are designated by abstract symbols, called the "name" of the segment. A translation device converts names to featural descriptions of each segment on a level-by-level basis; that is, each name at the syllable-initial level is translated, then each name at the nucleus level, then each name at the coda level. The results of the translation process are called the "command representation". The level constraint ensures that error interactions between syllables are confined to homologous syllable positions (which is the normal pattern of interaction errors in spontaneous speech). The priority (or other temporal differences) of initial position translation may be related to the predominance of syllable (and word)-initial interactions (Shattuck-Hufnagel, 1987, this issue). In effect, this model, like the slot-and-filler model, takes two sources of information – syllable structure and segment identity – and combines them into a structured, featural description of the segments in their syllable positions as commands to the motor system. (See Butterworth & Whitaker, 1980, for a critical discussion of Shaffer's model.)

I have assumed, so far, that PE begins with the PLR; however, there is evidence to suggest that an earlier stage in word retrieval makes use of phonological information. A number of authors (e.g., Butterworth, 1980, 1989; Fromkin, 1971; Garrett, 1984; Kempen & Huijbers, 1983; Levelt, 1989) have argued that word retrieval takes place in two separate stages: the retrieval of an abstract representation, called a "lemma" by Kempen and Huijbers (1983) and Levelt (1989), from a separate lexicon of such representations, called a "semantic lexicon" by Butterworth (1980, 1989) since it is claimed that these items are organized semantically so that each semantic input specification is paired with a "phonological address". According to Butterworth (1980, 1981, 1989) these representations provide a phonological address (Garrett's, 1984, "linking address") for locating PLRs in the "phonological lexicon" where items are organized according to their phonological properties. According to Butterworth (1981) and Garrett (1984) information about the form of the intended word can be recovered from the addresses themselves since the addresses are systematically linked to phonological characteristics, like number of syllables, initial segment, etc. The address locates a PLR in a multidimensional phonological space in which similar-sounding words will be at neighbouring addresses. Information from the address can be used when a lexical search fails to locate a PLR in the phonological space, as for example in the tip-of-the-tongue state. Here the word itself is inaccessible but the speaker is nevertheless able to report at least some phonological features correctly, especially first segment and number of syllables (Brown & McNeill, 1966). These states are explained in the following way: the lemma has been successfully retrieved, along with the phonological address. When the PLR proves inaccessible, the speaker can recover information from the address which can then be reported to the experimenter, or used to generate candidates by

guessing the missing information.² We will return to specific deficits of addressing in section 3 below.

I differ from Levelt (1989) in not considering here the morphological structure of the planned word. I shall assume without argument that word forms for known words are not derived on-line from morphemic components. This is not to say that morphology is unrelated to the phonological form of a word (cf. Kiparsky, 1982), nor that the rules for derivation and inflexion are unknown to the speaker, nor even that a PLR contains no morphological information, but only that information about morphology and lexical rules are deployed just when word search fails to retrieve a PLR meeting the retrieval specification – the phonological address.³

This outline account, presented diagrammatically in Figure 1, leaves open many details, some of which will be discussed below.

Control processes

An important point to note is that each subsystem in translation is served by a control process which, *inter alia*, retrieves information from an earlier subsystem as indicated by a directional arrow, and that the transmission of information from one subsystem to another is subject to transmission loss. A control process checks the output of the subsystem it serves. I assume that it does this by running through the process twice and comparing the results. (See Butterworth, 1981, for further explication.)

Back-up

I also assume that control processes provide access to back-up devices that can be invoked when things go wrong. One way this may happen in PE is for default values of phonological parameters to be generated when information cannot be

²Later processes, which I shall not discuss here, deal with other aspects of the precise phonetic realization in context of the filled slots: for example, overall properties of the output due to register, like rate of speech, volume, across-the-board phonetic features, like the palatalization of solidarity in Basque (Corum, 1975, cited by Gazdar, 1980) or dismissive or sarcastic nasalization in some English and American dialects, or sentence-final lengthening. Accounts of resyllabification processes – like ‘John is’ to ‘John’s’ for informal talk or the dropping of word-final post-vocalic /r/ in British English and some American dialects – are arguably a systematic part of the translation from PLR, though some forms, like epenthetic vowels and liaison in French, seem to be later processes, in part at least, pragmatically conditioned like the former examples.

³A defence of this position can be found in Butterworth (1983). The use of morphological rules by an aphasic patient to construct new forms when search fails is described in Semenza, Butterworth, Panzeri, and Ferreri (1990).

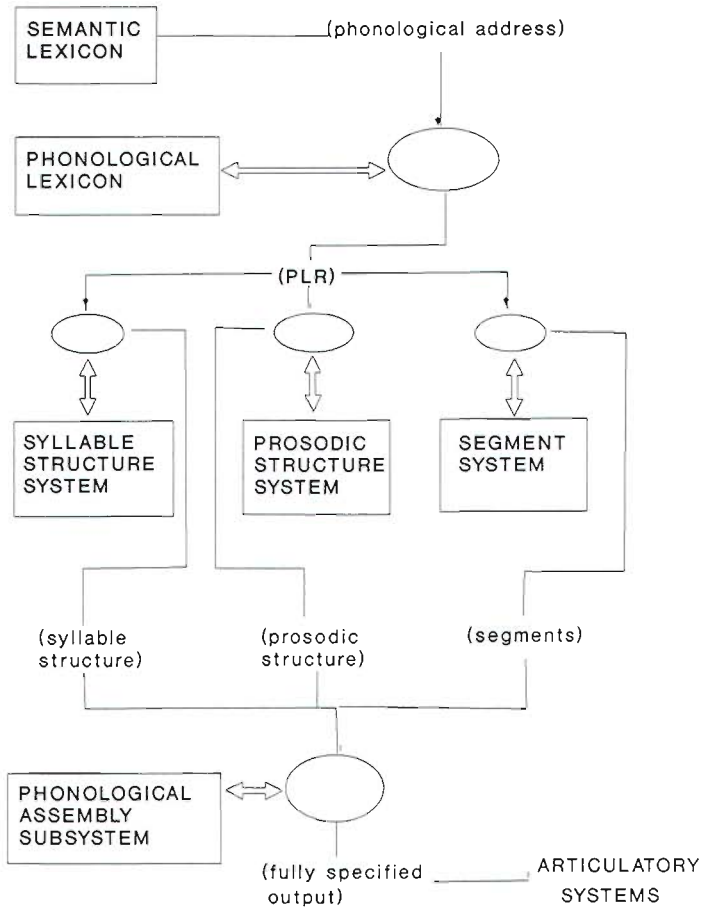


Figure 1. Outline model of phonological encoding processes. The operation of this model can be best illustrated by stepping through the encoding of the word *tenant*, and the paraphasia /'semənt/. (See section 1.1 for further details.)

1. A phonological address is retrieved from the SEMANTIC LEXICON and can be thought of as an n-tuple defining a location in the PHONOLOGICAL LEXICON; for example, <2, 3, 6 . . .>. It will have as neighbour, say, tennis at <2, 3, 7 . . .>. The generation of both target and paraphasia are held to start with the retrieval of the correct address.

2. The PHONOLOGICAL LEXICON associates the address with a PLR (phonological lexical representation) that contains information as to how *tenant* should be pronounced – the segments it contains, and its syllable and prosodic structures. For reasons that are explained in section 1.1, the PLR for *tenant* has not been corrupted in store.

3. This information is spelled out by dedicated, independent systems for SYLLABLE STRUCTURE, PROSODIC STRUCTURE and SEGMENTS. For the correct output, *tenant*, all the information in the PLR is correctly spelled out. For /'semənt/, syllable and stress information may be fully available, though defaults could yield similar outcomes, but it is assumed that some or all of the information about the first consonant has been wholly or partly lost in transmission; the segment system generates a default segment, /s/, either from scratch, or from residual information about place of articulation, with manner information lost.

retrieved from the PLR. Thus, if a syllable specification is unavailable, then a default pattern is generated – for example, CVC; if a stress pattern is unavailable, then perhaps the (English) default strong–weak for a two-syllable word will be generated; or if a segment value is unavailable, a default segment is generated, though it is unclear on what basis: perhaps, only unmarked segments are generated, or in the case where some but not all features of the segment are available, then an unmarked version consistent with the available features is constructed. We will consider these possibilities below.

A further point is that the phonological assembly subsystem (PASS) can contain the phonological description of more than one word; this appears necessary to explain errors with source in another word, like segment and feature movements (“heft lemisphere”, “capsy tag”; Fromkin, 1973), word blends (“shromkin” – “she” + “Fromkin”) and environmental contaminants (“ungutted frish” from planned “fish” and heard “fresh”; Harley, 1990).

This model has many features in common with the interactive activation model proposed by Dell (1989). Words are linked to phonemes for output via direct links and via a wordshape network specifying the syllabic structure. This mirrors the slot-filling idea. One could develop the analogy between links and addresses, and explore the possibility that explicit checking can be modelled as feedback activation. In any event, little of what is said here will discriminate between these modelling frameworks. (See Butterworth, 1989, and Levelt, 1989, chapter 9, for further discussion of these issues.)

One omission from our model, and from others discussed, is how non-words are intentionally generated. Non-words are important methodologically, since they permit us to assess whether brain damage has disturbed the translation processes themselves since PLRs are not implicated. Typical tests deploy the reading or repetition of non-word stimuli. I shall assume that the processes of reading and repetition generate phonological information in the same format as PLRs – a list of segments, a syllable structure outline and a stress pattern – which can be spelled out by the relevant systems and assembled by PASS. This has some plausibility given that a novel stimulus may become a genuine lexical entry; however, a definitive account of non-word generation awaits further investigation.

4. *These are then assembled by the PHONOLOGICAL ASSEMBLY SUBSYSTEM, which fits the segments into slots in a prosodically specified syllable structure. Thus the initial /t/, or the default /s/, is fitted into the onset position of the strong first syllable. The fully specified output needs to include all relevant information for the ARTICULATORY SYSTEM.*

The control processes, indicated by ovals, enable the generation of default information from the associated systems, or elsewhere, in the event that relevant information is missing from the PLR. For further explanation, see text.

1. Deficits of phonological lexical representations versus deficits of translation

The most basic and apparently the most straightforward issue is whether a phonological error arises as the result of a corrupted stored PLR or as a malfunction in the translation processes, in particular whether the malfunction is due to some impairment in one of the translation processes.

I shall begin by looking at five kinds of study that try to address this issue. The outcome will be, I am afraid, rather dull from a theoretical perspective. To the extent that a deficit can be identified, it lies not in the systematic, or even unsystematic, corruption of information in the PLRs; it is simply one of *noise in transmission* from PLRs to the translation, so that translation processes have to spell out specifications with variable, perhaps random, holes in them. Picturesquely, we could contrast seeing a hill through swirling mist – the hill is still there but the features are only intermittently visible (transmission loss) – with a word written in washable ink that has had water splashed on it (loss or corruption in storage).

The methodological moral I shall draw is equally trite, and moreover, rather old. In 1926, the British neurologist, Henry Head wrote:

It is not a sufficient test to hold up some object and ask the patient to name it; at one time he may be able to do so, at another he fails completely. No conclusion can be drawn from one or two questions put in this way; his power of responding must be tested by a series of observations in which the same task occurs on two or more occasions. (1926, Vol. 1, p. 145)

To determine the source of an error or phonological encoding, it is necessary to observe in each patient several attempts to say the same word, for example by testing the patient several times with the same naming stimuli; in addition, it is necessary to see whether comparable errors are made on words alike and unlike in the theoretically relevant respects. To take an (imaginary) example: does patient X always say “chee” instead of “tea”, and “sip” instead of “ship”, but is correct on “toast” and “sap”? Or does he say “fis” instead of “fish” *and*, in general, always says /s/ instead of /ʃ/, in the manner of the child Amahl, whose developing phonology is described by Smith (1973)? That is to say, are there *item-specific* errors which show up on just some words with a particular feature, or are there *feature-specific* errors that apply to all words (and non-words) with a particular feature? In the case of Amahl, the contrast, in production, between /s/ and /ʃ/ had not yet developed.

1.1. An example of the difficulties of identifying the locus of an error

Because phonological representation and processes are implicated in, and link, lexical representations to phonetic plans for output, the presence of a speech error may be difficult to interpret with respect to candidate processes and representa-

tions. For example, a jargon aphasic patient, DJ, produced ['semənt] instead of the target, *tenant* (Butterworth, 1985). Single phoneme substitution paraphasias are not uncommon in aphasic speech (e.g., Fry, 1959), nor indeed in normal speech (e.g., Shattuck-Hufnagel & Klatt, 1979), yet a definitive locus in the production system is uncertain. DJ may have a corrupted lexical representation for *tenant* in which the initial /t/ has been replaced by an initial /s/; or the /t/ may have lost elements of featural specification, in the PLR, so that only something like [-sonorant, +coronal] remains to specify the initial segment and additional features will need to be generated, in this case incorrectly; or the initial phoneme /t/ has just been lost in its entirety, yet syllabic structure clearly indicates that an initial consonant is required; or, like normal errors, where loss of information at the level of PLR is not usually an option, something has gone wrong translating the PLR /t/ into the phonetic plan; or some arthric or praxic difficulty prevents /t/'s, or stops more generally, being properly articulated.

To eliminate some of the candidate interpretations, one needs to see what other types of error the patient makes; in particular, one needs to see whether the patient makes the same error every time he or she tries to produce the target.

In the case of DJ and *tenant* we do have some relevant data. DJ was a publican, working as the tenant of a brewery, a fact he was at pains to convey, since a tenant, who holds a lease on the pub, has a different, and, in DJ's view, a higher, status than a mere manager who simply operates the pub for a salary. Five examples, in two sessions, of his attempts to produce *tenant* in spontaneous conversation were recorded, and are reproduced in example (1):

(1) ['emnənt . . . 'semənt . . . 'tenənt . . . 'tenəmən . . . 'tɛneɪt]

Each attempt came from a separate sentence, so the sequence cannot be regarded as *conduite d'approche* (see section 1.2). Taking the incorrect attempts as a whole, one can see that each segment, /t, e, n, ə, n, t/, is produced in its correct word position at least once, and no error is produced more than once; and one attempt was fully correct. It is reasonable to suppose, therefore, that the PLR of the word has not suffered permanent corruption, neither through the replacement of a target segment by an intrusion, nor by the loss of a phonetic feature on one or more of the segments. Leaving aside arthric or praxic problems, from which DJ did not suffer, some problem in translating an intact PLR into a phonetic plan seems the most likely explanation, though the variability of error forms precludes proposing a specific locus in the translation process. The most likely account is that on each attempt to say the word some of the information about the segments was lost in transmission, but in a rather unsystematic, perhaps random, way. (This is not, of course, to say that DJ had a fully preserved vocabulary that sometimes got scrambled in his attempts to talk, but only that for this target the translation explanation appears the most consistent with the evidence so far presented.)

A similar analysis can be adduced for the syllabic structure of the PLR: in three examples it corresponds to the target – [‘semənt, ‘tenənt, ‘təneɪt] – while the other two show distinct error types. In [‘emnənt] the structure of the syllables CVC\$CVCC rather than CVC\$VCC, with stress remaining on the first syllable, and in [‘tenəmən], we find an additional epenthetic weak syllable, and a final syllable reduced from CVCC to CVC, though the first syllable is like the target. It is plausible to interpret this pattern also as due to variable loss in transmission from an intact PLR.

Without an examination of other attempts to say the same word the translation account of the original error, [‘semənt], for this patient would not have been adequately grounded. In the case of normal speech errors, we are entitled to assume that the target PLR is intact, unless there are good grounds for thinking otherwise, as there are for Sheridan’s character, Mrs Malaprop.

Of course, we were fortunate in finding several examples of the same identifiable target in free speech. This permitted the inference of loss of information from one intact PLR to the translation processes. However, if only one PLR resulted in this pattern of errors, then a reconsideration of this inference would be needed. To see whether other words suffered the fate of “tenant”, we tested DJ on a picture-naming task in two separate sessions one month apart. Overall, he named three pictures in Session 1 that he was unable to name in Session 2, and five pictures in Session 2 that he could not name in Session 1. The paraphasic errors indeed showed a similar pattern to the *tenant* example:

(2) Target: eskimo

Session 1: [‘esiməu]

Session 2: [‘æstiməu]

Target: hedgehog

Session 1: [‘dɪdʒɒg]

Session 2: [ɪg, ɒs, ‘hɪdʒɒg, ‘egɒg], H-E-

Target: jacket

Session 1: [‘dʒækə, dʒæk]

Session 2: [‘dʒækə], zipper, [zɪpəweɪz]

Although we found evidence that he knew all the segments of *hedgehog*, the other two examples show different errors on each occasion, as would be expected if there was variable loss in transmission, but the errors do not contain in sum the whole segmental specification of the target. Perhaps with further trials the remaining segments would have emerged (/k/ in *eskimo*, /t/ in *jacket*). With only two trials it is hard to say. However, in most studies of phonological encoding, there is only one trial for each word presented as evidence, which means that, at best, the pattern across different errors remains the only evidence to identify the locus of the deficit.

1.2. Evidence from successive attempts

One interesting exception comes from a study by Joannette, Keller, and Lecours (1980) of a familiar aphasic symptom, *conduite d'approche*, in which the speaker makes successive attempts to say an intended word, in contrast to our study of DJ, which analysed separate attempts at the same word. (See also Kohn, 1984, for a similar analysis of a single case.) Joannette et al. noted that for French-speaking Broca, Wernicke and conduction aphasics, successive approximation tended to approach the target pronunciation. The only example they offer is:

- (3) target: /krejõ/ (crayon)
 approximations: /kreb . . . krevõ . . . krejõ/
 (Transcriptions as in the original.)

They argue that because approximations tend toward the target, this indicates a degree of control by the patient over what we have called the translation process. The control consists of an awareness of error (in comparison with the target) and the availability of a "monitor" to modify the output. It is perhaps significant that the strongest trend towards accuracy was found in the conduction aphasics, where comprehension is typically more intact than in the other two groups, suggesting the involvement of comprehension processes in monitoring, along the lines indicated by Levelt (1989). However, for this to be established one would need correlations on a patient-by-patient basis between the degree of comprehension ability (especially for single words) and the incidence of errors corrected, as well as with the trend of the approximations. A further interesting finding is that the trend disappeared in conduction aphasics for a non-word repetition task and was very weak for a real-word repetition task (data were not available for the other groups). These patients are partly defined by poor repetition ability, and at least some will have especial difficulty in maintaining a phonemic trace of the input. This suggests that the approximations are based on a comparison between the attempts and the representation of an internally generated lexical target.

Since conduction aphasics appear to end up on or very close to the target, it is reasonable to suppose that the target PLR is more or less intact, so that the comparison term which the monitor is using to assess attempts will be correct, but that some aspect of the translation process is malfunctioning, at least on occasion. For the other groups, this interpretation is less clear, and it may be that more severely impaired patients do indeed suffer corruption or loss of stored PLRs, though whether for all words, or some category of words (e.g., low-frequency words) cannot be inferred from the data Joannette et al. present. One useful analysis that they might have done would have compared, say, the word frequency of those targets where the approximations show a trend toward the target with the frequency of those which do not show this trend.

In any event, these data also suggest that one aspect of the encoding cycle – the PLR – can be intact while the translation processes sometimes fail to spell out the information therein contained. One problem here is that although translation occasionally fails, overwhelmingly it seems to work satisfactorily, producing the intended output. Again, occasional loss of PLR information in transmission to the translation processes is indicated.

1.3. Variability and consistency in naming errors

To separate storage deficits of PLRs from translation impairments, a minimal requirement is to know whether particular words usually lead to phonemic errors in a given patient and whether there are some aphasics who characteristically produce phonemic errors. This requires testing and retesting a group of patients on the same words on several occasions. A study by Howard et al. (1984) goes some way to answering these questions. Twelve aphasic patients were given a set of 300 pictures to name on three separate occasions, plus another set of 300 different pictures with the same names (plus a test of reading the names). Although subjects were highly consistent in the proportion of names correctly produced in the various tests (correlations by subject, $r > .97$), they were much less consistent on an item-by-item analysis. The contingencies of correct/incorrect on test x and correct/incorrect on test y ranged from .282 to .536. Thus one patient named correctly only 73% of his successes from a previous test, and 38% of his failures. Two pieces of evidence suggest that naming performance depended on an ability to retrieve PLRs of the target: first, for 9 of the 12 patients, accuracy correlated with word frequency, though the correlations were low (from .081 to .340); second, for six patients, there was no correlation between accuracy and word length, and for the others correlations were very low. If the naming deficit were confined to the translation processes, one would expect high correlations with length (since there would then be more opportunities to make translation errors) and purely lexical factors should not be important since the trouble would take place downstream of the successful retrieval of the PLR. Length effects were largely confined to the non-fluent patients, who often suffer arthric and praxic problems in addition to specifically psycholinguistic difficulties.

Howard et al. (1984) also analysed the consistency of the *types* of error made. Four patients made no phonological errors (See footnote 1), while nearly 14% of errors were phonological in two patients (one fluent, one non-fluent), and in general patients were highly consistent in the proportion of phonemic errors from one test to the next (correlation of .933). Thus some patients characteristically make phonemic errors, others do not. Moreover, where a patient makes a phonological error on test 1, he or she will, on average, produce the *identical* error

15% of the time,⁴ and another phonological error 10.5% of the time. The remainder were non-responses (44%), correct responses (26%) and semantic paraphasias (4%). None of the responses were unrelated real words (including "malapropisms", see section 2) or neologisms. What is not clear is whether the average conceals some patients in the group who reliably produce identical phonological error responses.

If a patient suffered corruption of stored PLRs, then we would expect identical error responses each time this item was retrieved, *ceteris paribus*. We might also expect that lexical factors would characterize the corrupted items. For example, it may be that infrequent words will be more prone to corruption, as well as to loss or to retrieval delays (Newcombe, Oldfield, & Wingfield, 1965). From Howard et al.'s report, we cannot tell whether infrequent words are more liable than frequent words to phonemic distortion, in particular, to consistent phonemic distortion. Until we know this, we cannot tell, for any patient in the study, whether the phonological errors are due to corrupted PLRs or translation problems or both.

1.4. Phonemic errors in reading

A detailed study of one conduction aphasic patient (NU) by Pate et al. (1987) is the most thorough attempt known to the author to assess item-specific consistency of errors. The primary data come from tests of reading, and reading may involve the generation of phonological information by a non-lexical procedure, by mapping graphemes onto phonemes, and this may play a role in the generation, and prevention, of phonemic errors. Nevertheless, Pate et al. claim that NU's reading errors were qualitatively similar to errors found in spontaneous speech. The same words occurred in five different reading contexts, from single words, word blocks and phrases to whole sentences, and responses to some of the words were further presented auditorily in a repetition task. Generally, words that prompted a paraphasia in one test prompted paraphasias in other tests, and words correctly produced in one test were generally correctly produced in others. The number of identical error responses in two test conditions was quite low (11 examples) and

⁴It would be interesting to know the probability of producing an identical error when, say, one segment substitution is selected at random from the usual frequency distribution of English segments in the substitution context. For this study, at least two deviations were required for a form to count as a phonemic error, so one would further need to calculate the probability of two segment substitutions resulting in an identical response. There is a further complication: some syllable positions, and some segments, appear more likely to be involved in error than others. (See, for example, Pate et al., 1987, described in section 1.4). It is therefore uncertain whether the identical error responses of Howard et al.'s patients should be treated as candidate corruptions of PLRs or not.

only one word prompted exactly the same error in all five test conditions (diseases /dɪzɪzə z/ → /dɪsɪzə z/). The main factor in errors was the length of the target (as it was with Caplan's, 1987, patient RL), and this does not appear an artefact of the greater number of opportunities for error in longer words (again like Caplan's RL).

Like DJ, the various attempts at a target can show different errors, yet jointly realize all the phonological information in the target:

- (4) Target: product /pradəkt/
 Session 1 /pradək/, Session 2 /pradək/, Session 3 /pradənt, prabən/, Session 4 (omission), Session 5 /pradək/
 (Pate et al., 1987, Table 7.)

Notice also that the syllable structure is slightly different in Session 3, though unlike the other attempts is closest to CVC\$CVCC structure of the target. (It is difficult to resist the following speculation: suppose NU had spelled out the syllable structure CVC\$VC_iC and had spelled out /t/ as the final consonant, yet had no segmental information for the content of C_i, a likely default filler generated by a back-up device would be something with alveolar feature, for example /n/ or /s/, rather than the /k/ of the target.)

Pate et al. conclude that the striking effects of length on paraphasic errors, combined with the lack of cross-word interaction errors, "reflects a constraint on the amount of phonological information which can be programmed within a unit". That is to say, there is a kind of channel capacity limit to spelling out apparently intact information in PLRs, which is perhaps combined with loss of information in transmission, especially for unstressed material (cf. Caplan's (1987) account of similar length effects in a similar patient).

1.5. The generation of phonemic paraphasias and neologisms

Many fluent aphasics produce neologisms. There is no agreed definition of neologism: a single phoneme substitution can lead to the creation of a form not in the dictionary, yet is nevertheless an identifiable distortion of an identifiable target – what is usually termed a phonemic paraphasia – while others bear no apparent relation to the target. The critical point is not how these forms are called, but what account is offered of them. From the framework offered here, the question is, "Can neologisms be explained in terms of a disorder of PE, either as corruptions of stored PLRs or as a systematic malfunctioning of the translation processes?" Unfortunately, most neologisms are nonce occurrences. (Less than 4% of Howard et al.'s (1984) neologistic naming responses were the same on two test sessions). Where they are not, they are stereotypes produced in a wide range of settings for presumably a wide range of targets. These facts reduce the value of

repeated testing of the same stimuli. However, something can be learned from the pattern of neologistic production in spontaneous speech.

For those not familiar with neologistic speech, I will offer a brief characterization of the kinds of patient where it most commonly occurs. Neologisms are found in greatest abundance in the speech of "jargon aphasics". This is a term which describes a rather heterogeneous group of speech types and patients (see Butterworth, 1985, for a brief review, and Brown, 1981, for a more extended account). Most neologistic speakers are fluent, with poor comprehension, though the degree of impairment varies widely and is unrelated to the incidence of neologisms (Butterworth & Howard, 1987, p. 27). Usually, perhaps always, they suffer posterior damage and may be considered a subtype of Wernicke's aphasia (Buckingham & Kertesz, 1976). A reasonably accurate extended transcript of a classic case can be found in Butterworth (1979).

According to Butterworth (1979) and Panzeri, Semenza, and Butterworth (1987), non-words have three distinct sources (data from patient KC):

- (i) They may be phonemic distortions of the target; e.g. [tʃæk] for chair, [dɒkjumən] for doctor.
- (ii) They may result from the intrusion of phonemic material from a prior or following word; e.g. "she has to do things [wʌmən] a woman who helps".
- (iii) They may be generated by a back-up "device" and bear no relation to any target. Phonemic variants of a device neologism may be used five or six times in different sentential contexts; e.g. [bæklænd . . . bændɪks . . . ændɪks . . . zændɪks . . . lændɒks . . . zæpriks].

It is argued by Butterworth (1979) that these forms are deployed when there has been a partial or complete failure to retrieve a PLR. For type (i), only part of the target has been retrieved, and a device has to fill in the missing information about syllable structure and segments. For type (ii), the target PLR has not been retrieved, but another word in the plan for the current utterance is active, or available, which in this case has lost some information and the back-up device again fills in the missing information. (Sometimes KC appears to produce a contextual word correctly, but in the wrong place, rather like word-movement errors in normals.) For type (iii), no information is available from the target PLR, or from any other word, and a filler has to be constructed from scratch. If this happens more than once in quick succession, information from the previously constructed neologism(s) can be exploited, hence the sequence of similar-sounding neologisms. A key piece of evidence that Butterworth (1979) used to make this argument is that the pauses before type (iii) were longer than before types (i) and (ii), which in turn were longer than pauses before real words, whether correct or verbal paraphasias, suggestive of search times that depended on the amount of information retrieved. These data are again consistent with the idea that PLRs are

uncorrupted in store, but information is lost in transmission to the translation processes.

It is worth noting that the neologisms use the full repertory of phonemes (and no non-English phonemes) in phonotactically legal ways, suggesting that there are no specific deficits in translation. However, given the absence of repeated attempts at the same words, it is also possible that PLRs are corrupted. Certainly, patients like KC suffer word-finding difficulties, and it is likely, but not demonstrated, that some PLRs may be lost, rather than simply being inaccessible. Clearly, further studies of these patients need to be carried out. DJ (Butterworth, 1985; Butterworth & Howard, 1987), who was similar in many respects, showed evidence of variability of accessibility to a target from session to session, but nevertheless he too may have lost vocabulary items. What is critical here is that, on occasion, KC needed to institute translation when no or insufficient phonemic information about the target was available, resulting in forms that were well formed and sometimes quite unrelated to the target. It is implausible to suggest that the PLRs for matches, matchbox, telephone and dial, which were the targets of the neologisms in (iii), were all corrupted so as to yield similar-sounding items.

The claim that neologisms strategically substitute for search failures has been substantiated in a longitudinal study of an Italian jargon aphasic, PZ. Over the course of seven months, the incidence of type (iii) neologisms was drastically reduced, apparently as the result of a developing strategy to avoid contexts in which word search was likely to fail completely. It was argued that stereotyped utterances were increasingly used to avoid search failure. The other types of neologism remained more or less constant.

Another source for neologisms has been suggested by Pick (1931) and Howard et al. (1985). They claim that many are phonemic distortions of a verbal paraphasia (wrong word). Howard et al. report the following examples from their study:

- (5) Utterance: /spaidɪd/ from Target: Web via Spider
 Utterance: /ætə/ from Target: Globe via Atlas

Although these examples are well attested and plausibly explained, this account seems unlikely for all the type (iii) neologisms of KC. For our current purposes, these non-word forms do not allow us to distinguish between storage deficits and translational deficits, though they are consistent with the idea that there has simply been loss in transmission of phonemic information from the PLR, in this case the wrong PLR.

2. Is there good evidence for specific deficits in translation processes?

In the above brief and selective review, it was argued that there was little direct evidence for corruption of stored PLRs, and that the data presented were

compatible with a rather minimalist account of what was going wrong in translation, namely that information from PLRs was “lost in transmission”. However, it has often been claimed that aphasic patients suffer more specific disorders of PE. In the next selective and brief review some of these claims are examined. Bear in mind that none of the studies satisfy the basic methodological desideratum of repeated observations of attempts at the same word. Blumstein (1973) has argued that this essentially does not matter:

Variability in performance is not directly at issue in considering the phonological patterns underlying aphasic performance; i.e. although the quantity of errors may in fact vary from day to day, the direction and types of error should remain qualitatively similar. (1973, p. 22n)

Now it may not matter from the point of view of phonological theory, but it seems to matter when attributing a pattern of errors to a specific processing locus.

2.1. Deficits in spelling out segmental structure

In our outline model two subsystems involve segmental information: the subsystem that spells out segmental information in PLR, and the PASS, where segments are inserted into their syllabic slots. We look first at possible evidence for a disorder of reading segmental information from PLR. What might this look like? One possibility is that the subsystem fails to encode certain phonemic distinctions; for example, the voicing feature may be consistently lost, so that all voiced segments come out as unvoiced. More generally, there may be a tendency for marked segments to be produced as unmarked. Of course, without repeated testing of each target, we cannot be confident that the cause of error patterns such as these, should we find them, is to be located in segmental translation or in systematically corrupted PLRs.

In a pioneering study, Fry (1959) analysed the phonemic substitutions in the tape-recorded oral reading of CVC words by one patient. The patient is described as having hesitant speech, with no verbal paraphasias or neologisms, and good comprehension and reading. Nineteen per cent of the responses were phoneme substitution errors (21% of consonant targets and 16% of vowel targets). Errors of place, manner and voicing were found for consonant targets, but with no obvious pattern, except that there appeared to be an unusually high proportion of voicing errors. Some consonants (/ʃ/ and /tʃ/) produced no errors, while /d/ and /v/ produced 43% and 35% respectively. The order of difficulty of consonants was unrelated to the order of difficulty found either in children’s errors (Morley, cited by Fry) or in normal adult segment errors (Shattuck-Hufnagel & Klatt, 1979). Although the patient apparently suffered articulatory problems, as evidenced by hesitation and severe difficulties with consonant clusters (which therefore were not tested), this does not seem to account for the pattern of errors. Fry writes:

The most interesting feature of this type of [voicing] error was that the phonemic substitution was complete and hence involved a re-arrangement of the time scheme of a whole word, particularly when a consonant followed a vowel. The patient said /m^k/ for *mug*, for example, with appropriate vowel length for its voiceless consonant . . . Much more is involved here than the failure to make the larynx work when required; the whole organisation of the syllable has to be changed . . . It was not that at the level of articulation he merely made a poor attempt at the correct phoneme. (1959, p. 57)

In a study of 17 aphasic patients, Blumstein (1973) found that overall patients were able to use the full inventory of American English phonemes. In contrast to Fry, the patients' overall probability of an error on a phoneme was inversely related to the phoneme's frequency of occurrence. It may be that some patients were unsystematic in this respect, like Fry's patient; unfortunately individual patient data are not presented. Different types of aphasia – Broca, Wernicke and conduction – produced similar types and distributions of error types. Although there was a slight tendency for unmarked segments to substitute for marked segments, this did not apply to the conduction aphasics as a group, and may not hold for every patient in the other two groups. It would be interesting to know whether there were some patients for whom this tendency was highly marked, and others who did not show it all. Unlike Fry, Blumstein did not analyse the effects of substitutions on the overall organization of the syllable in which it occurred.

Nespoulous, Joannette, Béland, Caplan, and Lecours (1984) have examined the phonological output of 4 Broca's and 4 conduction aphasics for tendencies to produce unmarked in preference to marked forms. They seem to believe that Blumstein (1973) found no differences between these two groups, and seek to establish more firmly that Broca's do, but conduction aphasics do not, have a tendency toward unmarked forms in their errors. They certainly find that Broca's as a group show clearer preferences for certain phonemes as substitutes, with more unmarked responses. However, it is also clear that patients move as readily from the unvoiced (unmarked) to the voiced form as vice versa. At the same time, Broca's are more likely to reduce clusters to single segments (marked to unmarked) than are conduction aphasics, who are as likely to create new clusters in error as to reduce target clusters.

2.2. Deficits in spelling out syllabic and prosodic structure

There is very little evidence available here. Pate et al. (1987) found that their patient, NU, was much more likely than chance to omit unstressed syllables than syllables with primary or secondary stress. Moreover, more phonemes were omitted from unstressed syllables than stressed, when word position is controlled. Kean (1977), in a discussion of agrammatic patients, suggested that phonological elements not taking lexical stress were the most prone to omission.

2.3. Deficits in assembling segments and syllable structures

It is known from the studies of normal speech errors that phonological distortions are not confined to intrusive substitutions of one or more phonemes; some involve the interaction between two words in the current speech plan – as in segment anticipations, perseverations and metatheses. Are aphasic speakers more prone to these errors? Do they produce different ordering errors? Garrett (1984, p. 189) suggests that an impairment to PASS in conduction aphasics would result in several of the commonly occurring symptoms, including sound-exchange errors.

In Pate et al.'s (1987) study of the reading errors of patient NU, overwhelmingly phonemic movement errors were confined to within-multiword interactions, despite a tested ability to construct phonological phrases appropriately. This effect was checked out in a separate experiment.

In an interesting recent study, Kohn and Smith (1990) analysed the sentence repetition performance of a conduction aphasic, CM. This patient made many segment anticipations and perseverations, but no exchanges. Most of these errors copied a segment so that it replaced a similarly positioned segment, usually the nucleus and/or coda, in the interacting word:

- (5) Jane road → Joan
 Nurses tend patients → /Nei, Nei/
 Matthew broke his ankle → /Maeku, Maek/
 (Transcriptions as in the original.)

Now although errors of these types occur in normals, a variety of other types have also been observed in normals, notably exchanges.

Kohn and Smith (1990) offer an explanation of these phenomena which involves a malfunction in a device that “clears” a planning buffer in what we called PASS, so that when the segments from the next PLR come to be inserted into the syllabic slots, some of those slots will still be occupied by segments from the previous word. It is not clear whether the clearing mechanism is more than a terminological variant of Shattuck-Hufnagel's (1987) mechanism that deletes a segment once it has been used: the notional locus is different, to be sure, in the assembled slots rather than in the list of segments to be inserted, but the only data that favours Kohn and Smith's account are the absence of addition errors – that is to say, attempts to insert the correct segment alongside an incorrect segment, to produce a cluster or a new diphthong do not succeed. All interaction errors are simple replacements.

To account for anticipatory errors, it has to be assumed that words are not assembled in strict order of output, and if the predominance of anticipatory errors is evidence, then *typically* words are assembled out of their final utterance order. This may strike the reader as implausible, and certainly independent motivation for this apparently strange way of doing things is needed. For example, one might

run an argument to the effect that some particular category of words has priority in PASS, for example, that heads are assembled before modifiers, or that words receiving sentence accent are assembled before others. I know of no good reason to propose this, nor do Kohn and Smith offer an analysis of the data which would allow the reader to investigate such a possibility. Another problem concerns the pattern of phonemic paraphasias that cannot be attributed to movement errors. This is not described, but from the number of sentence stimuli repeated correctly, there appear to be a lot of them. It is possible, at least, that apparent pattern of between-word errors is an artefact of a range of phonemic processes implicated in errors, including exchanges. Within-word exchanges are excluded from the analysis, for example. Further analyses are needed before an interpretation of the locus of these errors can be confidently proposed.

The bottom line, if I understand Kohn and Smith's presentation correctly, is this: (1) between-word interactions are never exchanges, though within-word interactions may be, whereas normal interaction errors produce between-word exchanges routinely (perhaps, predominantly; see Shattuck-Hufnagel, 1987); (2) interactions typically involve the rhyme portion of syllables, whereas normal interactions typically involve onsets (Shattuck-Hufnagel, 1987). In tongue-twisters, the latter pattern is also observed. Butterworth and Whittaker (1980, Experiment 1) asked normal subjects, but including lawyers, to repeat two-syllable items ("mat rat", "pap pack") as quickly as possible. For all types of interaction – anticipations, perseverations and exchanges – rhymes were more often implicated than onsets (though note there are two rhyme positions but only one onset), and perseverations and anticipations were far more common than exchanges. Shattuck-Hufnagel (1982, reported in Shattuck-Hufnagel, 1987) found that the repetition of four-word lists ("leap not nap lute") tended to produce more rhyme interactions than onset interactions, while the same words in a phrasal context ("from the leap of the note to the nap of the lute") showed the opposite pattern of errors, which she attributes to words being "protected" by the phrasal organization provided by the phrasal condition.⁵ It is possible that CM has a tendency, for some reason, to treat his output in a more list-like, and less structured manner.

⁵Shattuck-Hufnagel's (1982, cited 1987) experiment does not appear to have used the appropriate controls – (a) differs from (b) in more than just the presence of phrasal organization:

- (a) leap note nap lute
- (b) from the leap of the note to the nap of the lute

Example (b) contains an additional eight unstressed syllables which may act as a buffer between the critical items. The apparent likelihood of interactions between words are conditioned by proximity, and it is possible that the type of interaction is also conditioned by it. Perhaps Shattuck-Hufnagel should have tried strings like (c):

- (c) the from leap the of note the to nap the of lute.

3. Disorders of phonological addressing

Garrett (1984) suggests that phonological addresses may be impaired, or lost, in anomic conditions, but not in conduction aphasia, and cites as evidence an observation by Goodglass, Kaplan, Weintraub, and Ackerman (1976) that anomics can only produce information about the initial letter of a word they cannot retrieve in 5% of instances, while conduction aphasics can do this on 34% of instances. If, as I suggested above, information from the phonological address can be recovered when search is incomplete, then failure to recover it may indicate loss of the address in the anomic cases. For the conduction aphasics, by contrast, their difficulty seems to occur later, in loss of PLRs or the translation of PLRs into fully assembled output.

Our conception of the speech production system contains a semantic lexicon which associates semantic specifications with phonological addresses for locations of PLRs in the phonological lexicon. This raises a further possibility, namely that the addresses may be corrupted. In our model, a corrupt address would have the following consequences: either the corrupt address locates a blank space in the phonological lexicon, in which case no PLR will be retrieved, or it will locate a neighbouring PLR, which will be similar-sounding, but most likely semantically unrelated.

A relevant case was recently reported by Blanken (1990). The patient, RB, was a fluent anomic patient who managed relatively few content words, and sometimes made phonemic paraphasias and neologisms. He had poor auditory comprehension of single words. On the whole his reading and writing seemed less impaired than the speech modality. The phenomenon that characterized his speech, and has never previously been reported, is a large number of malapropisms, or what Blanken calls "formal paraphasias". These are real words that sound similar to the target but are usually unrelated in meaning. (Some are similar in both sound and meaning.) Examples are:

- (6) a. Schrank (cupboard) → Strand (beach)
 b. Kreide (chalk) → Kreise (circles)
 c. Kerze (candle) → Berge (mountains)
 d. Kasper (Punch) → Kassen (tills)

It is hard to explain these errors in terms of single-segment errors that by chance sound like a real word. If that were the case, one would expect there to be a large, perhaps larger, number of non-word single-segment errors than formal paraphasias. There turn out to be only a few examples of these, and Blanken has calculated that chance errors would not explain his results.

Lack of data on item specificity prevents us ruling out with complete confidence an alternative explanation for the errors in (6). Perhaps RB had obtained the correct address for, say, *Kreide*, but the target PLR /kraidə/ was lost (or seriously

damaged); in these circumstances, RB may simply have retrieved its nearest neighbour in the phonological lexicon, *Kreise*. However, although Blanken does not report different attempts at this target, he does offer some relevant indirect evidence. In a repeated naming task, RB was cued with the first phoneme of the target. This manipulation not only improved overall performance (in terms of number of items correct) but also reduced the proportion of formal paraphasic errors (Blanken, 1990, Table 5), suggesting that targets that otherwise led to formal paraphasias were still in store, but not retrieved (or perhaps retrievable) without a cue. The value of cues in aiding retrieval of hard-to-find words has been extensively demonstrated in the aphasic literature (e.g., Howard & Orchard-Lisle, 1984; Howard et al., 1985); and even where explicit cueing does not help, semantic priming may have a sizeable effect on retrieval (Chertkow & Bub, 1990). Thus cueing and priming are useful techniques for establishing loss versus inaccessibility of lexical information. Exactly how the additional phonemic information provided by cueing works is not well understood, but it seems to have both a different time course and other differential effects from priming, where presumably the target PLR receives activation from the prime. It is at least conceivable that cues are incorporated into the phonological address, which would be consistent with our account of the formal errors found in RB.

Examples (6b)–(6d) are interesting in another way: target and response do not show number agreement (singulars become plurals), but like 92% of the examples they do agree in number of syllables, and of these (presumably, Blanken is not clear on this point) 98% agree in stress pattern (the exception involves loan word targets). If, as I suggested above, the phonological lexicon contains full forms rather than roots (or stems), then these outcomes are unproblematic: a near neighbour is selected purely on the basis of the phonological properties of the full form (e.g., *Berge*). On the other hand, if the phonological lexicon contains roots and affixes separately – in the extreme case where grammatical affixes are part of a separate system in a separate location (e.g. Garrett, 1980) – then it will be hard to explain why, in the examples above, the address does not locate a two-syllable root rather than a one-syllable root, which then gets inflected. Unfortunately, Blanken does not report the likelihood of failure of grammatical agreement between target and response; nor does he report whether agreement failure depends on the structure of the target. So there are some unresolved issues here.

4. Conclusion: specific deficits or just more normal errors?

The errors observed in our review of the patient data have been, as far as one can tell, of the same types as those found in normal speech deficits. There is some evidence that certain patients have a predilection for certain types of error; for

example, for unmarked over marked segments. However, there is absolutely no evidence as to whether individual normal speakers also have these predilections. Where comparisons between normal and patient errors have been explicitly drawn, the normal data come from the collectivity of errors made by unspecified numbers of different people. Of course, we may find someone not clinically referred, who, like Kohn's patient CM, made anticipatory and perseveratory segment movements, but not segment exchanges. Or, we might find in our control population a speaker whose errors were confined to real words, rather like Blanken's RB. What would we say about such cases? Caramazza (1986) would doubtless have to say that such cases cannot exist (by definition), or that perhaps they are (by definition) neurological cases wandering the streets that ought to have been referred for neurological examination.

The point here is that it is hard to draw inferences about specific deficits if the only data consist of idiosyncratic preferences, often very slight, for one class of normal errors. Taking the studies reviewed above as a whole, the only thing that can be said with any confidence is that in some patients there seems to be loss of information in transmission between one subsystem and another; in which case, *idiosyncratic preferences may reflect the functioning of the back-up systems rather than the normal systems themselves*. There is little evidence that the storage of phonological information in the lexicon, or in the subsystems that spell out lexical entries, is disturbed in any of the patients here discussed; nor is there evidence that the operation of these subsystems has suffered a long-term malfunction. Of course, such things may happen. We just do not have the data to tell, yet.

The data we need should be collected in accordance with the following minimal list of methodological desiderata.

Item specificity and deficits of storage

1. Several observations of the production of each target are required.
2. Such tests should be carried out on a patient-by-patient basis.
3. The probability of error for each target should be assessed in relation to known lexical factors, word frequency being the most obvious.

Feature specificity and deficits of translation

4. The probability of error should be assessed in relation to the phonological features (characteristics) of the targets. Length, structure and the presence of certain segments are obvious candidates.
5. Non-word repetition (and reading) tests can be used to eliminate lexical, and hence potential storage, deficits. Bear in mind that aphasics, especially conduction

aphasics, may suffer problems at the input end of such tests which will affect repetition of such stimuli.

6. Non-word tests can be designed to assess the involvement of specific phonological features of the targets.

Articulation versus internal generation of phonology

7. Tests of phonological judgment – rhyme or homophony, number of syllables or segments, etc. – can be deployed, where the intactness of later, articulatory, processes is in doubt.⁶

References

- Archangeli, D.B. (1985). Yokuts harmony: Evidence for coplanar representation in nonlinear phonology. *Linguistic Inquiry*, 16, 335–372.
- Beland, R. (1990). Vowel epenthesis in aphasia. In Nespoulous, J.-L., & Villiard, P. (Eds.), *Morphology, phonology and aphasia* (pp. 235–252). New York: Springer-Verlag.
- Blanken, G. (1990). Formal paraphasias: A single case study. *Brain and Language*, 38, 534–554.
- Blumstein, S. (1973). *A phonological investigation of aphasic speech*. The Hague, Mouton.
- Brown, J.W. (1981). *Jargonaphasia*. New York: Academic Press.
- Brown, R., & McNeill, D. (1966). The “tip of the tongue” phenomenon. *Journal of Verbal Learning and Verbal Behavior*, 5, 325–337.
- Buckingham, H., & Kertesz, A. (1976). *Neologistic jargon aphasia*. Amsterdam: Swets and Zeitlinger.
- Butterworth, B. (1979). Hesitation and the production of verbal paraphasias and neologisms in jargon aphasia. *Brain and Language*, 8, 133–161.
- Butterworth, B. (1980). Some constraints on models of language production. In Butterworth, B. (Ed.), *Language production Vol. 1: Speech and talk* (pp. 423–459). London: Academic Press.
- Butterworth, B. (1981). Speech errors: Old data in search of new theories. *Linguistics*, 19, 627–662.
- Butterworth, B. (1983). Lexical representation. In Butterworth, B. (Ed.), *Language production Vol. 2: Development, writing and other language processes* (pp. 257–294). London: Academic Press.
- Butterworth, B. (1985). Jargon aphasia: Processes and strategies. In Newman, S., & Epstein, R. (Eds.), *Current perspectives in dysphasia* (pp. 61–96). Edinburgh: Churchill Livingstone.
- Butterworth, B. (1989). Lexical access in speech production. In Marslen-Wilson, W. (Ed.), *Lexical representation and process* (pp. 108–135). Cambridge, MA: MIT Press.
- Butterworth, B., & Howard, D. (1987). Paragrammatisms. *Cognition*, 26, 1–37.
- Butterworth, B., & Whittaker, S. (1980). Peggy Babcock’s relatives. In Stelmach, G., & Requin, J. (Eds.), *Tutorials in motor behavior* (pp. 647–656). New York: Plenum Press.
- Caplan, D. (1987). Phonological representations in word production. In Keller, E., & Gopnik, M. (Eds.), *Motor and sensory processes of language*. Hillsdale, NJ: Erlbaum.
- Caramazza, A. (1986). On drawing inferences about the structure of normal cognitive systems from the analysis of impaired performance: The case for single-patient studies. *Brain and Cognition*, 5, 41–66.
- Chertkow, H., & Bub, D. (1990). Semantic memory loss in dementia of Alzheimer’s type. *Brain*, 113, 397–417.
- Dell, G. (1989). The retrieval of phonological forms in production: Tests of predictions from a

⁶Feinberg, Rothi, and Heilman (1986) report three patients able to perform similar tasks, though quite unable to vocalize the words involved.

- connectionist model. In Marslen-Wilson, W. (Ed.), *Lexical representation and process* (pp. 136–165). Cambridge, MA: MIT Press.
- Feinberg, T., Rothi, L., & Heilman, K. (1986). Inner speech in conduction aphasia. *Archives of Neurology*, 43, 591–593.
- Fromkin, V. (1971). The non-anomalous nature of anomalous utterances. *Language*, 47, 27–52.
- Fromkin, V. (1973). Appendix. In Fromkin, V. (Ed.), *Speech errors as linguistic evidence*. The Hague: Mouton.
- Fry, D. (1959). Phonemic substitutions in an aphasic patient. *Language and Speech*, 2, 52–61.
- Garnham, A., Shillcock, R., Brown, G.D.A., Mill, A.I.D., & Cutler, A. (1981). Slips of the tongue in the London–Lund corpus of spontaneous conversation. *Linguistics*, 19, 805–817.
- Garrett, M. (1980). Levels of processing in sentence production. In Butterworth, B. (Ed.), *Language production Vol. 1: Speech and talk* (pp. 177–210). London: Academic Press.
- Garrett, M. (1984). The organisation of processing structure for language production: Applications to aphasic speech. In Caplan, D., Lecours, A.R., & Smith, A. (Eds.), *Biological perspectives on language*. Cambridge, MA: MIT Press.
- Gazdar, G. (1980). Pragmatic constraints on linguistic production. In Butterworth, B. (Ed.), *Language production Vol. 1: Speech and talk* (pp. 49–68). London: Academic Press.
- Goodglass, H., Kaplan, E., Weintraub, S., & Ackerman, N. (1976). The “tip of the tongue” phenomenon in aphasia. *Cortex*, 12, 145–153.
- Harley, T. (1990). Environmental contamination of normal speech. *Applied Psycholinguistics*, 11, 45–72.
- Head, H. (1926). *Aphasia and kindred disorders of speech*. Cambridge, UK: Cambridge University Press.
- Howard, D., & Orchard-Lisle, V. (1984). On the origin of semantic errors in naming: Evidence from the case of a global aphasic. *Cognitive Neuropsychology*, 1, 163–190.
- Howard, D., Patterson, K., Franklin, S., Morton, J., & Orchard-Lisle, V. (1984). Variability and consistency in picture naming by aphasic patients. In Rose, F.C. (Ed.), *Advances in neurology Vol. 42: Progress in aphasiology* (pp. 263–276). New York: Raven Press.
- Howard, D., Patterson, K., Franklin, S., Orchard-Lisle, V., & Morton, J. (1985). The facilitation of picture naming in aphasia. *Cognitive Neuropsychology*, 2, 49–80.
- Joanette, Y., Keller, E., & Lecours, A.R. (1980). Sequences of phonemic approximations in aphasia. *Brain and Language*, 11, 30–44.
- Kean, M.-L. (1977). The linguistic interpretation of aphasic syndromes. *Cognition*, 5, 9–46.
- Kempen, G., & Huijbers, P. (1983). The lexicalization process in sentence formulation. *Cognition*, 14, 201–258.
- Kiparsky, P. (1982). From cyclic phonology to lexical phonology. In van der Hulst, H., & Smith, N. (Eds.), *The structure of phonological representations: Part 1* (pp. 131–176). Dordrecht: Foris.
- Kohn, S. (1984). The nature of the phonological disorder in conduction aphasia. *Brain and Language*, 23, 97–115.
- Kohn, S., & Smith, K.L. (1990). Between-word speech errors in conduction aphasia. *Cognitive Neuropsychology*, 7, 133–156.
- Levelt, W.J.M. (1989). *Speaking: From intention to articulation*. Cambridge, MA: MIT Press.
- Nespoulous, J.-L., Joanette, Y., Béland, R., Caplan, D., & Lecours, A.R. (1984). In Rose, F.C. (Ed.), *Advances in neurology Vol. 42: Progress in aphasiology* (pp. 203–214). New York: Raven Press.
- Newcombe, F., Oldfield, R., & Wingfield, A. (1965). Object naming by dysphasic patients. *Nature*, 207, 1217–1218.
- Panzeri, M., Semenza, C., & Butterworth, B. (1977). Compensatory processes in the evolution of severe jargon aphasia. *Neuropsychologia*, 25, 919–933.
- Pate, D.S., Saffran, E., & Martin, N. (1987). Specifying the nature of the production impairment in a conduction aphasic: A case study. *Language and Cognitive Processes*, 2, 43–84.
- Pick, A. (1931). *Aphasia*. Springfield, IL: Thomas (Translated by J.W. Brown, 1973).
- Semenza, C., Butterworth, B., Panzeri, M., & Ferreri, T. (1990). Word-formation: New evidence from aphasia. *Neuropsychologia*, 28, 499–502.
- Shaffer, L.H. (1976). Intention and performance. *Psychological Review*, 83, 375–393.

- Shallice, T., & Butterworth, B. (1977). Short-term memory impairment and spontaneous speech. *Neuropsychologia*, 15, 729-735.
- Shallice, T., & Warrington, E.K. (1977). Auditory-verbal short-term memory impairment and conduction aphasia. *Brain and Language*, 4, 479-491.
- Shattuck-Hufnagel, S. (1987). The role of word-onset consonants in speech production planning: New evidence from speech error patterns. In Keller, E., & Gopnik, M. (Eds.), *Motor and sensory processes of language* (pp. 17-51). Hillsdale, NJ: Erlbaum.
- Shattuck-Hufnagel, S., & Klatt, D. (1979). Minimal use of features and markedness in speech production. *Journal of Verbal Learning and Verbal Behavior*, 18, 41-55.
- Smith, N.V. (1973). *The acquisition of phonology*. Cambridge, UK: Cambridge University Press.
- Stemberger, J.P., & Treiman, R. (1986). The internal structure of word-initial consonant clusters. *Journal of Memory and Language*, 25, 163-180.