Phonological Factors in Aphasia*

Harold Goodglass
Boston VA Hospital and Boston University School of Medicine

I approach the problem of phonological factors in aphasia with misgivings, first, because articulatory disorders have not been my special interest and I have never done any research on this topic; second, perhaps because of this lack of specialization, I consider the problem very complex and have my own reservations about the simple subdivisions which I have drawn. Almost every time that I listen to a speech sample with someone whom I am trying to indoctrinate into my way of listening, I hear instances which break the rules that I have formulated.

The rules are very simple and are based on linguistic levels, because of the dogma that I follow that aphasia is a linguistic disorder. The rules of phonology are, just like those of semantics and syntax, in the domain of linguistics, so I regard any organic breakdown of articulation as aphasic, provided that it varies with the communicative intent of the speaker.

Within the scope of the term 'phonological disorder' there are two levels, the phonetic and the phonemic, and it is in the distinction between these two that I feel the major effort of the clinician should be directed, as well as the principal thrust of phonological research in aphasia. Phonetics refers to the science that relates the precise configuration of the vocal tract to a corresponding sound pattern and deals with systems for most usefully classifying human speech sounds. Articulatory disorders at the 'phonetic' level are those which are manifested by a deficiency in the voluntary control of articulatory gestures, such that the output no longer corresponds precisely to the sounds of the speaker's language. Performance may range from complete inability to produce any speech sounds (except perhaps simple recurrent utterances) all the way to comprehensible speech with many portions that are phonetically incorrect in that they are labored, prolonged, over-aspirated, explosive, and have their consonant blends simplified. In other words, the general context is of attempts at English phonemes which, even if identifiable, are phonetically off target.

In contrast, what I refer to as the phonemic level of disturbance is one which occurs in a context of runs of fluently articulated syllables. The phonological errors are typically at the level of substitution, anticipation, or transpositions of phonemes or syllables. However, the important thing to note is that the phonemes themselves are well formed English sounds,

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produced at a normal rate. The subjective impression which is produced on the listener is that the articulatory mechanisms are intact, but that the patient is unable to program the selection and sequencing of his intended phonemes. Now, when unintended English segments are produced in an effort to say something we term the output 'paraphasia'. The phonological errors of the patient with a breakdown at the phoneme level are called 'literal paraphasias' or phonemic paraphasias—terms which I presume are familiar to you. In contrast, going back to the patient with a phonetic level breakdown, the off-target approximations or gross distortions which we hear are articulatory defects rather than 'paraphasias'.

To bring this rather abstract formulation down to earth let me read you a transcript from a patient with a phonological disorder at the 'phonemic' level.

Sample of conversation with a conduction aphasic:

(What is your address?) "Seventy Humboldt Way, Ray...(for Humboldt Way) Marmal...Marble...hay...hay (for Marblehead Massachusetts)."

(How did you get into the hospital?) "Well my doctor called them up and I had to have sexes...that's sess and I had plenty of them." (Plenty of what?) Sex. That's sess. Y-E-S-T-S: -sesx. Plenty of sest...tex (Tests?) Thats right."

(Were you sick, or what?) Well the first time I know, I had a piece of pa...paker like that and I rock...I locked the paper on the floor.

(You're retired?) But I do a fishing lies...ries...(he ties flies) and also make rods (Flies?) No...thats right. Rie...rlies...flials. E-L-Y-S. (Flies?) Flies, that right.

It is easy to transcribe this defective speech—every sound corresponds to an English phoneme—it is simply that the patient gets the wrong sound in the wrong place, with rather funny results. In contrast let me play for you the painfully labored wpeech of a patient who has a disorder at the phonetic level. (Two samples of conversation with Broca's aphasics were presented to illustrate labored and distorted articulation.)

These individuals differ in severity, in the consistency of the effortful quality, and very notably in the fact that sentence structure is intact in the first and telegraphic in the second patient. What is true for both is that the gross and subtle phonetic distortions, related to effortful and awkward quality, cannot be represented by standard phonetic symbols. I included a sample of repetition with each of these, to show that articulation tends to improve under repetition—a feature which usually characterizes the patient with lesions in the anterior speech zone, whereas repetition merely aggravated the problems of the conduction aphasic.

Why do I lay so much emphasis on the distinction between
phonetic distortions and phonemic paraphasia? Indeed, as we will see, there is a very fuzzy distinction between literal paraphasia and articulatory distortion. Yet, in spite of the overlap, it is a fundamental diagnostic distinction, because it identifies two forms of aphasia which have different cerebral foci and many other different linguistic features. I intend to spend the middle part of this presentation in listening with you to instances which are boundary cases and which make us sharpen up our criteria for distinguishing between phonemic and phonetic defects and which also make us humble about whether any scoring system can pretend to offer absolute distinctions.

In fact it is not only in the phonological aspect of aphasia, but in every modality that we are confronted with the dilemma of choosing between so-called 'objectivity' in scoring, on one hand, and drawing inferences about the mechanism of a particular wrong performance by examining the context in which it occurs. For instance, suppose our objective is to examine auditory comprehension of words, using the medium of pointing to a multiple choice array. One patient may respond by wrinkling his forehead and saying 'I don't get you--say it again'. You say it many times for him and finally a light may dawn, he may repeat it perfectly and point to the object--or this may never happen. We have here the typical performance of the patient with 'pure word deafness'. In contrast, in the case of Wernicke's aphasia a patient may uncomprehendingly repeat the stimulus after you and then select another stimulus from the same class as the target. If we are hypnotized by the precision with which we can say 'right' or 'wrong' these really crucial qualitative distinctions go by the board, because the number of 'correct' items may add up the same in both instances.

I could give you illustrations in the area of naming, oral reading, and purposeful movement--but let us return to articulation. I am arguing that there are two primary mechanisms which may both end up causing the patient to make the same or approximately the same misarticulation; that the evaluation of the particular error depends on the context in which it appears.

When I say two primary mechanisms, I am of course still referring to the phonetic versus the phonemic levels, but I wish to formulate them in slightly different terms: namely, whether the speaker's level of articulatory activity is organized around well formed English phonemes and phoneme sequences which may be mistargeted as whole units; or, on the other hand, whether he is primarily trying to get together the motor components to make the individual phoneme come out right. Either of these causes can result, for example in the substitution of a 't' for a 'd'. In order to evaluate the type of problem the patient is having we must listen to the overall pattern of this speech output, not merely make sound-by-sound judgments.

In order to proceed further with the problem of phonology in
aphasia we should backtrack again and try to redefine what makes a disorder one of language rather than merely of a defect in one of the instrumentalities—that of articulatory control, which by itself would lead to dysarthria. As I indicated earlier, it does not work to draw the lower limit of 'language' at word finding and grammatical formulation, and to exclude from aphasia disorders which are chiefly in phonology. The essential element is that the use of the instrumentalities of speech vary as a function of the communicative intent of the speaker. For example, a recent article by von Stockert in Brain and Language was entitled 'Aphasia sine aphasia.' Von Stockert claimed that one of his patients who named perfectly well to confrontation and repeated complete sentences with intact grammar did not fulfill the criteria for 'aphasia' because his disorder was not 'linguistic'. The point that von Stockert missed is that his patient's language performance varied grossly depending on whether it were self-initiated, automatized, or given in repetition. The unavailability of a speech act for purposeful, self-initiated, communication, when it may be available under other circumstances is an important criterion for aphasia. It applies equally to the phonological level. When a patient cannot correctly articulate the sounds of a word in conversation or in naming, but does so correctly in repetition or as part of a memorized sequence we have the requisites for considering the defect aphasic, quite apart from the status of other components of language. Or conversely, if the patient's phonological output in free conversation approaches normal and deteriorates when he is asked to name or even to repeat from a model we again are dealing with an aphasic phenomenon.

This is precisely why it is so difficult to come up with a satisfactory test of articulation, by approaching it the way one approaches ordinary articulation problems; that is requiring the repetition of words with target sounds in initial, medial, or final position. Often, the patient with free and facile articulation in conversation produces remote paraphasic responses, while the patient with labored and awkward speech output repeats fairly well at the one-word level.

Picking up the earlier thread of this discussion, how successful can we be in imposing linguistic categories on articulatory disorders? I must admit that, while I feel comfortable with the phonemic level, I have grave misgivings about how far linguistic analysis will help us understand the more severe levels of phonetic disintegration. As we listen to and watch the slow, clumsy movements, the difficulties in initiation, the changes in voice quality, it seems obvious that something is wrong at the level of motor organization and that there are no linguistic units small enough to categorize what the patient is doing. Where there are parallels with the speech of the young child--e.g., the simplification of consonant blends, the usual earlier recovery of front than back consonants, these seem to me secondary to the order of
differentiation of the sensorimotor control of the articulators and to the intrinsic physical difficulty of certain maneuvers. That is, we can describe phonemic errors in terms of recognizable speech segments—i.e., 'phonemes'—which are misused. We can only to a limited extent, describe phonetic failures in terms of subphonemic components—i.e., distinctive features—which are dissociated from each other. Rather we must move to the language of motor innervation, where we are in the dark. We know only that this level of dysfunction is also a part of the specifically language directed system of the brain, because these movements may be impaired when all other non-speech activity is undisturbed.

The relation between apraxia for non-speech, oral-facial activity (what we refer to as bucco-facial apraxia) and speech movements is also far from clear. While De Renzi, et al. (1966) have reported that bucco-facial apraxia most frequently occurs in conjunction with motor-articulatory aphasic defects it would be very risky to conclude that a common apraxic disturbance underlies both speech and non-speech disorders. I say this because of the clear instances in which severe oral apraxias occur in a context of fluent articulation and vice versa. In his discussion of this issue Critchley (1970) points out that severe global incoordination of the oral apparatus may not properly come under the heading of apraxia at all.

Earlier in this presentation I played some tape extracts of patients with a breakdown of articulation which is predominantly at the phonetic level. I want to remind you of the fact that their repetition to command showed, if anything, a little better articulation than that in their spontaneous speech. From time to time—quite frequently, in fact, there are clearcut phonemic substitutions in their speech, but the general background of motoric clumsiness leads us to question whether these errors are not caused by disorders of motor control. Now I would like to play some tape recorded extracts of the other type of phonological disorder—that in which scrambled phoneme selection occurs in a context of completely facile articulation. In each case I have included some examples of repetition to command, because I wish to emphasize that to evaluate the aphasic nature of the articulatory disorder, it is important to compare performances under different conditions. There is an important group of patients for whom articulation completely disintegrates under repetition conditions. These are called 'conduction aphasics' and they invariably have post-rolandic lesions. The first tape illustrates the classic form of this disorder.

(Patient J.R. - extract of conversation)

Here we hear unambiguously a background of fluent, normally articulated speech, periodically disrupted, chiefly at points of high information, lexical items by a very distinctive type of phonological difficulty, in which the patient makes repeated stabs at the word, producing a variety of wrong syllables. A very
important feature is that repetition does not help this patient. If anything, it heightens his difficulties.

Not all cases of this diagnostic class are so classical in the contrast between a background of facile, grammatically fluent production, and a phonological tangle on encountering substantive terms. The next extract shows a case in which the selection and ordering of phonemes also goes into total collapse both during repetition and at key lexical items during free conversation. However, you will quickly hear that the general background level of phonological production is slow and motorically awkward.

(Patient A.W. - extract of conversation)

The question occurred to us some time back whether a careful linguistic analysis of phonological errors would confirm the clinical impression that different mechanisms underlay the sound substitutions of patients who had primarily phonetic distortions and those who had primarily phonemic substitutions. The study was carried out by my colleague Dr. Blumstein as her doctoral thesis. While these data have been published, they are so relevant to the subject matter of this talk that I would like to review the highlights and integrate them with the clinical observations we have been making.

Dr. Blumstein collected free speech samples from 18 patients—six each in the category of Brocas aphasia (i.e., phonetic distortion), conduction aphasia (i.e., primarily phonemic substitution errors in a phonetically well preserved context) and Wernicke aphasia (patients who, like conduction aphasics, are phonetically well preserved, but make a variety of both literal and verbal paraphasic errors). Each patient was interviewed until 2000 words, excluding small grammatical words, had been recorded. After phonetic transcription all transcribable errors with known targets were classified for further analysis. It is important to emphasize that a good many types of production errors were excluded from the analysis. Exclusions consisted of misarticulations whose target could not be inferred; grossly untranscribable phonetic distortions or neologisms, and verbal paraphasias. In other words, we are looking only at misarticulations which can be compared to their intended realizations. Moreover, only consonants were studied.

Errors were placed in four categories, as follows: a) Phoneme substitution (e.g., 'reems' for 'teams'); b) Simplification: omissions of phonemes (usually one of a consonant cluster) or omissions of syllables; c) Additions of phonemes or syllables and d) Environmentally determined errors. These refer to errors which can be related to adjacent phonological elements—such as forward or backward assimilation and inversion of phoneme order.

Before examining the qualitative features of the errors, a few of the overall quantitative questions should be examined. First, how many errors are produced by each type of patient? Here the results are as expected: Brocas aphasics made a total
of 1,993 classifiable errors, over three times as many as the next category, Conduction aphasics, who made 590. They in turn, made almost three times as many as the Wernickes, with a total of 219. These differences are not surprising since they correspond to the original basis for classifying the subjects.

As we examine the breakdown of errors we begin to see the trend which runs all the way through the rest of the data, namely the surprising parallels among all three groups in what determines their errors. Look first at the distribution of overall error types, expressed in percentages. Figure 1 shows that for all three diagnostic classes phoneme substitutions (represented by the white bar) are the most frequent, followed in turn by simplifications, environmentally determined errors, and additions. The one difference which appears, is that Wernickes aphasics do not show the heavy predominance of simple phonological substitution, but have a relatively large proportion of errors in the other three categories, particularly "additions". However, the parallels between the first two groups are quite striking.

The emphasis in the error analysis was placed on the direction of phoneme substitution errors, because it is with respect to these that the most trenchant linguistic issues could be posed. However, before examining these errors, it was necessary to determine whether the frequency distribution of aphasic's consonants is similar to that of normals. If there were marked deviations in frequency one might infer that the aphasics had an incomplete phonological inventory. Figure 2 compares the percent frequency of normals consonants to those of aphasics. The aphasic data is based on 1,000 consecutive phonemes as actually transcribed from each of six subjects--two per diagnostic group. The parallel is dramatic and a rank order correlation is .95.

Next, we may ask how error frequency relates to phoneme frequency. (Figure 3) Here we see a striking inverse relationship between the frequency of phonemes and the percent of errors contributed. There are two possible conclusions--first that there is a "law of least effort" in the development of the phonological system of any language, such that the phonemes which are motorically simplest find the most frequent use. Alternatively, we might conclude that the aphasic makes the most errors on the sounds which are the least practised by virtue of their lower frequency in daily use. Somehow it is easier for me to believe that it is not frequency so much as the intrinsic motor difficulty of the sound which determines both the error rate, and its distribution in the language.

Now proceeding to the analysis of phoneme substitutions, these were examined with respect to two closely related linguistic dimensions. The first basis for data analysis was the phonological distance between phonemes, as measured in distinctive features; the second was the direction of substitution from marked to unmarked versus the reverse direction.
To review these concepts briefly, you are probably familiar with the notion introduced by Roman Jakobson that each phoneme in a language is uniquely defined by the presence or absence of each of a small group of phonetic elements called 'distinctive features'. This leads to the notion that a phoneme is a bundle of distinctive features, and further, to the notion that the similarity between phonemes can be expressed by the number of distinctive features they share or do not share. Now, there have been a number of different sets of distinctive features proposed by various writers. The set used by Dr. Blumstein is indicated in Table 1, where we see the percentage of errors with respect to each feature by diagnostic class. Here we see that there is only a rough similarity between patient groups. In fact we notice that Wernickes' aphasics differ in being considerably less likely than the others to lose the feature of 'continuancy.' It was anticipated that, to the extent that phonemic substitutions follow linguistic principles, they would tend to move towards the phonemes most similar, in terms of phonological distance. This means that we would expect misarticulations differing by only one distinctive feature to predominate over all others. From Figure 4 we see that, in fact, errors of one distinctive feature account for two thirds of simple phoneme substitutions in each of the three groups. This is rather strong evidence for a similar principal underlying phonemic errors in all aphasics—the bit of a surprise since our original position was that we were dealing with groups whose articulatory errors had different mechanisms. I will have more to say about that later.

A second phonological principle emerges from the distinctive feature system—namely the polar opposition between marked and unmarked features. Here again I will take time out for a short explanation. Distinctive features may be expressed as polar opposite pairs such as voiced-unvoiced and nasal-oral. However these opposites are not equal, because one is proposed as more basic and natural. This is the unmarked form. Thus, we say that in the contrast 'p' vs. 'b' that 'p' is unmarked for voice; similarly in the contrast d-n, d is unmarked for nasality. In each case, then, 'markedness' represents the addition of a complicating phonetic feature which modifies a more general or basic sound. For economy, it is sufficient to indicate the presence or absence of the marked pole of a contrast. That is when we designate a phoneme as unmarked for voice, that is the same as saying it is voiceless. When it is unmarked for continuance, it is a stop.

If Jakobson's hierarchy of marked vs. unmarked features is to be supported, we would expect that most phonemic errors which are off target by only a single feature should go from the more complex or marked version of a feature to the more basic or unmarked pole for that feature. In other words we should more
commonly hear errors such as "tinner" for "dinner" than the reverse, such as saying "dable" for "table". An analysis of direction of errors between marked and unmarked shows that this is again the case. The difference is again in the neighborhood of 2:1 for Broca and Wernicke aphasics, but not quite significant in the case of conduction aphasics. (Figure 5)

I could show you further parallels in the rules determining phonological errors; parallels which apply across diagnostic classes, in spite of the admittedly disparate number of errors that we are talking about in each group. For example, we find consistently that phoneme substitutions occur overwhelmingly in isolated consonants as opposed to consonant blends. Initial word positions for consonant substitutions are overwhelmingly more frequent for Brocas and Conduction aphasics, but equally distributed between initial and final for Wernickes. We do detect a trend towards a difference between groups when we tabulate the distance across which consonant assimilation errors are made. When a Broca's aphasic substitutes an earlier or later appearing consonant for a target phoneme, it is within the same word twice as often as across word boundaries. For example tape-pape is twice as likely as roast beef-rofe beef. Conduction aphasics are almost equal in intra-morphemic and cross-morphemic assimilation errors, while Wernickes aphasics actually reverse the proportion and make almost twice as many assimilations across word boundaries as within. Even though the actual numbers are too small for statistical significance tests, we may conjecture about this trend as follows. That greater facility in producing phonemic sequences reflects pre-programming of larger units, within which transpositions may be made. The laborious output of Brocas aphasia suggests that it is rare for them to go beyond the syllabic level of articulatory pre-planning. The contrasting pattern of Wernickes aphasia in which fluency is unimpaired suggests that these subjects' ability to anticipate multisyllabic runs permits the interaction between elements to extend over longer spans.

The major trend which emerges from Dr. Blumstein's study is the overriding similarity in the rules which determine phonemic errors, regardless of the clinical distinction among aphasics. This finding, I assure you gives us a great deal to think about, given the initial thesis that Broca's aphasics, with their predominantly phonetic disability, were disturbed via a different mechanism from the two posterior aphasial types--conduction and Wernicke aphasics.

There are two considerations which make it unnecessary to discard the notion that the phonetic disorders of Brocas aphasia invoke a different mechanism from the phonemic disorders of conduction and Wernicke's aphasia. First, is the fact that only the transcribable phonemic changes were used. This leaves out of consideration all the unclassifiable sounds produced by Broca's
aphasics. Second, it seems likely that the distinctive feature system can be restated in terms of different dimensions of motor activity involving the articulators. Thus there is considerable overlap between a description in terms of linguistically defined components and one in terms of motorically defined components—especially when one ignores minor deviations from normal articulation.

Summary

Perhaps after this rather rambling presentation it behooves me to relieve you of the job of extracting the main points of my talk. First I point out that articulatory errors can be assigned to the level of phonetic distortions or phonemic substitutions. I proposed that phonemic errors are errors in programming of entire well-articulated phonemes while phonetic failures are best conceived as involving the organization of motor commands. Noting that either of these two mechanisms may have the same error as an outcome, I propose that the total context of a patient's articulatory pattern be relied on to influence your judgment about the particular case. Next, I emphasized that the variation in performance between self-initiated propositional speech, automatized speech, and repetition is critical, not only for deciding between aphasic and dysarthric phenomena, but in categorizing the type of aphasic. In general Brocas (or anterior) aphasics show predominantly phonetic level deficits which improve in the presence of a model to repeat. Conduction aphasics (with a post-rolandic lesion) have predominantly phonemic errors, and may deteriorate rather than improve when required to repeat from a model.

Finally, we saw the results of a linguistic analysis of phonemic errors—an analysis that suggests that common linguistic principles enter into determining both phonemic and phonetic errors.
References


Table 1

Hierarchy of Feature Dissolution

<table>
<thead>
<tr>
<th>Broca</th>
<th>Conduction</th>
<th>Wernicke</th>
</tr>
</thead>
<tbody>
<tr>
<td>continuant</td>
<td>39.0%</td>
<td>continuant 26.2% compact</td>
</tr>
<tr>
<td>compact</td>
<td>19.8%</td>
<td>compact 25.4% continuant</td>
</tr>
<tr>
<td>voice</td>
<td>16.6%</td>
<td>grave 20.4% voice</td>
</tr>
<tr>
<td>grave</td>
<td>10.8%</td>
<td>voice 15.3% gráve</td>
</tr>
<tr>
<td>nasal</td>
<td>9.3%</td>
<td>strident 7.3% strident</td>
</tr>
<tr>
<td>strident</td>
<td>4.5%</td>
<td>nasal 5.4% nasal</td>
</tr>
</tbody>
</table>
FIGURE 1

DISTRIBUTION OF APHASIC ERRORS
FIGURE 2
PHONEME FREQUENCY DISTRIBUTION

PERCENT

aphasic
normal
FIGURE 3

% ERRORS ON PHONEME RELATED TO % FREQUENCY OF OCCURRENCE

- - - occurrence
- - ratio
FIGURE 4

PHONEME SUBSTITUTIONS:
DISTINCTIVE FEATURE ANALYSIS
FIGURE 5

MARKEDNESS ANALYSIS