NEURAL MODELS OF LANGUAGE PROCESSES

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Those papers and discussion papers marked * are drafts prepared by the editor on the basis of the oral presentation. All other papers were drafted by the author. The discussion sections were drafted by the editor on the basis of notes and tapes of the sessions, and have been submitted to all participants for review and revision.

1. Artificial Intelligence and Computational Models

2. Linguistic Perspectives

3. Neurological Perspectives

4. Brain Theory Models

HWIM: A Speech Understanding System on a Computer

by

William A. Woods

1. INCREMENTAL SIMULATION OF HUMAN SPEECH UNDERSTANDING

This paper was presented at the conference on "Neural Models of Language Processes" in the belief that the space of hypotheses through which the computer searches serially in seeking to provide an interpretation for an utterance is similar to that through which the brain searches, presumably in parallel. In this section, we motivate this claim by looking at a number of experiments on human speech understanding, and the way in which these led to the design of our own computerized speech understanding system called HWIM.

A convenient graphical representation of acoustic information is given by a spectrogram. This is a plot of the spectral energy in a signal over time in which the horizontal axis is time, the vertical axis is frequency, and the grey level at each point of the graph indicates how much energy there is at a given frequency at a given time. In a spectrogram, voiced speech separates into a number of distinct horizontal bands called formants, with the locations of the formants over a short time interval being roughly indicative of vowel quality, and with the shape of the formant transitions into non-vocalic regions

providing information about consonants. Denes and Pinson [1963] gives a good general introduction to the acoustic-phonetics of speech and the information that is available in spectrograms. An expert can look at short time intervals extracted from a spectrogram, and can come up with a number of alternatives as to the characteristics of the phonemes which are present, rather than unequivocal judgments as to which are indeed present. For example, an expert might judge that an initial segment is an l or a w; that the next segment is a front vowel, consistent with the hypotheses iy, ih, ey, eh, or ae; that the third segment is an s or a z; that the fourth is unvoiced and plosive, which could be consistent with p, t, k, or ch, and so on. In fact, such a judgment was made on a spectrogram of the word "list", and the reader can see that this is one of the words compatible with the above sequence of alternatives.

In a classic study of this phenomenon, Klatt and Stevens [1972] looked at samples from a spectrogram through a narrow window, and attempted to transcribe the phonemes based solely on objective acoustic evidence.

Stevens, for example, looked at 80 words comprising 299 phonetic segments in all. He was able to correctly (and uniquely) transcribe 24% of the phonetic segments, and gave a correct but incomplete specification of an additional 50%. In 15% of the cases, he made a wrong transcription of the segment, and actually missed 11% of the segments that were in the utterance. Thus, even when he was allowed to 'hedge' on 50% of his judgments, he still had an error rate of 25%. Klatt's performance was comparable. However, if they were allowed to look at more than a single segment, to look at earlier segments and following segments and to use constraints of syntax, semantics and vocabulary, they were able to recognize 96% of the words that were presented to them -- which is far better than getting 96% of the phonetic segments right. Moreover, most of the remaining 4% of errors were confusions between 'a' and 'the', which are hard to disambiguate on local cues, especially if they follow a weak fricative, and which are also hard to separate on the basis of the limited context within a single sentence.

The above experiments indicate that people are relatively poor at (visually) recognizing isolated phonetic segments, and must bring syntactic, semantic, and pragmatic knowledge to bear. To this it might be objected that people are far more accurate at *hearing* phonemes than experts are at *reading* spectrograms. For example, people usually speak isolated words sufficiently clearly for them to be correctly heard without supporting syntax and semantics.

Nonetheless, when speaking in a continuous stream of words, people seldom articulate clearly enough for all individual words to be highly intelligible. (Even the case of isolated words depends on a knowledge of the lexicon and phonology of the language, as anyone with an unusual name knows when trying to get it recognized over the telephone!). However, if such words are embedded in as little as four words of context, the

intelligibility rate becomes quite high. In other words, our claim is that when people speak, they rely on the redundancy of the language to economize their articulation, and that when people understand speech they take advantage of their knowledge of the language to make up for this lack of local clarity.

In building upon the Klatt and Stevens work to provide the basis for our design of HWIM, we analyzed a speech understanding system in terms of the components shown in Figure 1. *Feature extraction* extracts basic features of the kind we discussed above. *Lexical retrieval* then searches for words that match these low-level features. Given word hypotheses, *matching* checks the low-level acoustic data to determine the degree to which the hypothesis is supported by the evidence. Once a string of words have been hypothesized, *syntax* checks whether it is well formed; *semantics* checks whether it is meaningful; and *pragmatics* checks whether it is relevant in the present context. As we shall see in more detail below, there is no simple process which can directly yield an unequivocal string of words which through syntactic, semantic, and pragmatic processes yields the unique interpretation. Rather, *control* is required to distribute attention among different subproblems, deciding when to switch from looking at one hypothesis or part of an utterance to another, and when the time has come to go back and re-examine an earlier hypothesis in the light of new data. *Bookkeeping* keeps track of what hypotheses have already been considered.

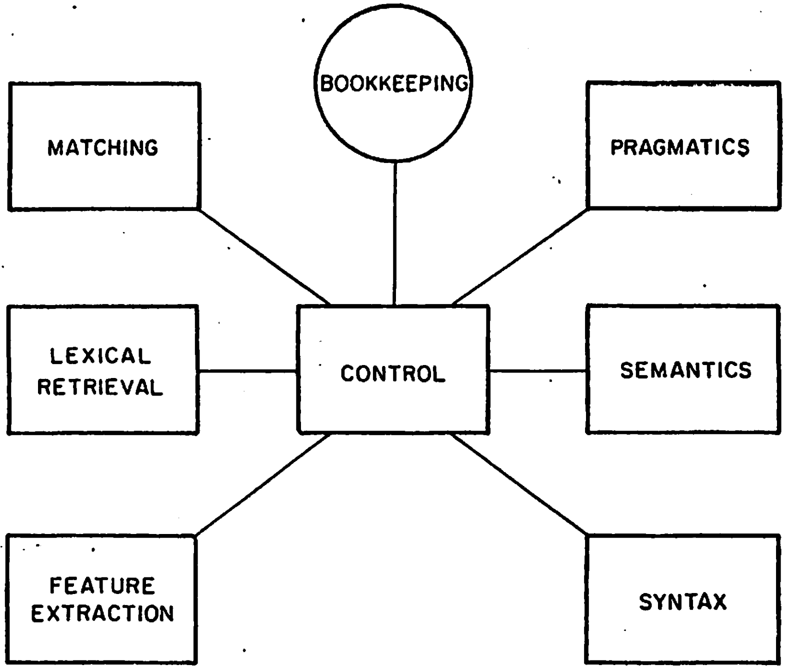

Figure 1. Components of a Speech Understanding System.

To get a better handle on how these different components might be implemented as computer programs, we carried out a number of experiments in what we call incremental simulation [Woods and Makhoul, 1974].

In each case, we 'implement' the system as a combination of human activity and computer programs, and 'run' it to discover and test algorithms and to develop an intuition for the problem. In our first experiment of this kind, we had a human carry out all the tasks of Figure 1 except that of lexical retrieval, which was handled by a machine. In this way, we could monitor the questions that the human asked, and begin to make hypotheses about his problem-solving strategy. In a second experiment, we had one human carry out the task of feature extraction, another human carry out the task of control, syntax, semantics, and pragmatics; while a machine handled the tasks of lexical retrieval, matching, and bookkeeping, and provided the communication channel between the two humans. In this way, we could get a better idea of the strategies that a human uses in feature extraction when he does not have access to higher level knowledge. The human carrying out feature extraction looked at the spectrogram through a narrow window — about 1-1/2 phonemes wide -- and his error rate suggested that he was indeed not using higher-level cues. By having the machine keep track of the communication between the two humans, we were able to form explicit hypotheses about the way in which feature hypotheses are mobilized in forming word-level and utterance-level hypotheses.

On this basis, we reached a number of conclusions which formed the basis for the design of the HWIM system to be described in the next section:

1. We learned that small function words are highly unreliable anchors. For example, the sound in 'a' is usually the same as that of the vowel in 'the', and it also occurs in many multi-syllable words. Thus, we feel that a speech understanding system cannot utilize the strategy used by several text understanding systems in which the function words provided the framework for determining the syntactic role of other words in the sentence.

2. We found that accidental word matches outnumber the correct ones. Good acoustic matches may in fact be misleading. For example, there is quite a good match for 'new' in the middle of 'anyway', and this false local hypothesis must be eliminated on the basis of further processing.

3. Computer scientists distinguish stochastic processes -- in which there is a probabilistic element in what will happen next -- from nondeterministic processes which are in no way unpredictable, but where at each stage of the process more than one hypothesis may have to be explored. In this sense, we came to the conclusion that speech understanding is an inherently nondeterministic process, and that it is necessary to make tentative hypotheses and systematically explore the consequences. For example, once a hypothesis has been made as to several words in the utterance, one should test for syntactically plausible hypotheses which cover the rest of the utterance in a consistent manner, and only maintain the original hypothesis if such an extension proves possible.

4. We found that sequential left-to-right scanning has problems. It is often necessary to provide the ability to recover from a garbled word. The first word of the sentence is often garbled due to high subglottal pressure. But right-to-left scanning is not a viable alternative, since the last word of a sentence can be garbled due to low subglottal pressure which, for example, lengthens phonemes. We thus often find it expedient to work out from those 'islands of reliability' provided by the stressed syllables away from the ends of the utterance.

5. When one is dealing with a vocabulary of upward of a thousand words, there are 10 to the 12th sequences of length 4. We thus cannot afford to list all possibilities and search them exhaustively, but must rather explore a limited but open-ended space of alternatives based on the data at hand.

6. Even limiting the alternatives in this way, we find that the search space can get too large unless we use merged representations: many similar hypotheses share common parts, and we can merge these to reduce the search space.

2. THE HWIM SYSTEM

In this section, we describe the HWIM speech understanding system [Woods et al., 1976]. This was designed to handle a vocabulary of 1,000 words, and had a syntactic/semantic/pragmatic grammar which enabled it to handle sentences such as

> How much is in the speech understanding budget?
> Show me a list of the remaining trips.
> What is the one-way air fare from Boston to London?
> Who went to IFIP?
> Show me Bill's trip to Washington.
> When did Craig go to Utah?
> Enter a trip for Jack Klovstad to San Francisco.
> The registration fee is twenty dollars.

which have to do with contracts, budgets, and conference travel.

The overall system organization of HWIM is shown in Figure 2. The speech signal is transduced by a microphone and then processed to yield parameters much like those that can be read from the spectrogram. An acoustic phonetic recognizer (APR) then processes these parameters to form a segment lattice such as that shown in Figure 3 corresponding to the spoken input "total budget". Actually, the segment lattice is richer than shown in the figure, since for each segment we list a number of alternatives together with their confidence levels. What is worth noting in Figure 3 is that not only do we offer alternative

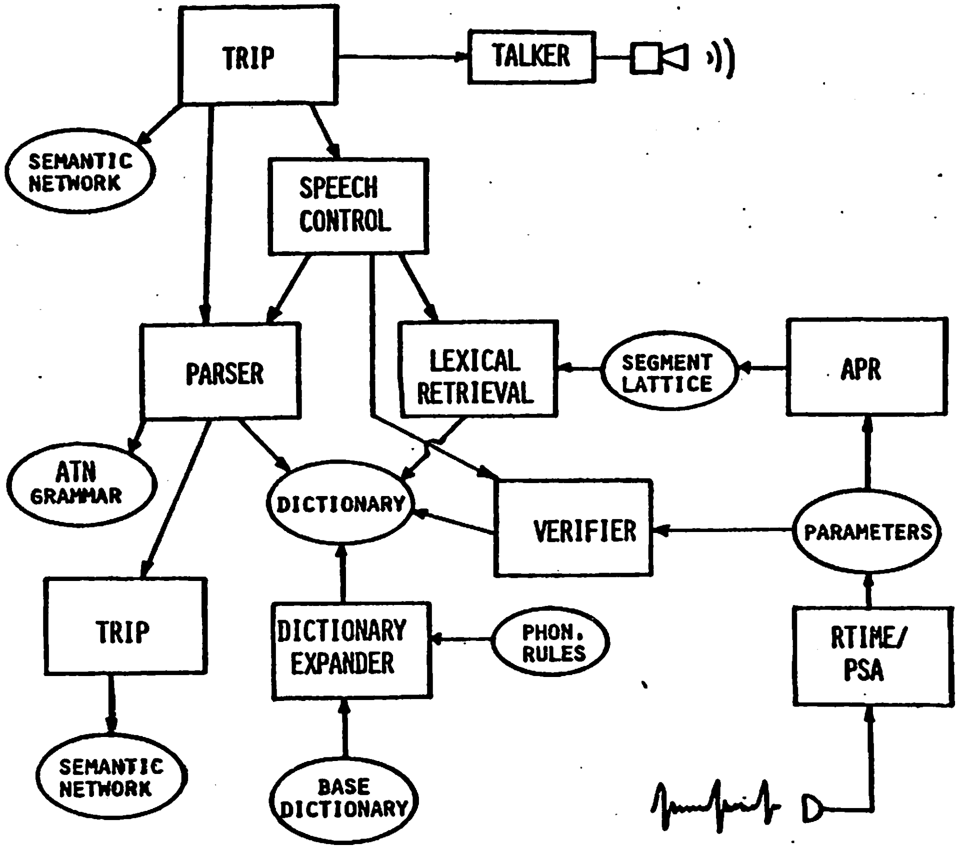

Figure 2. HWIM System Organization.

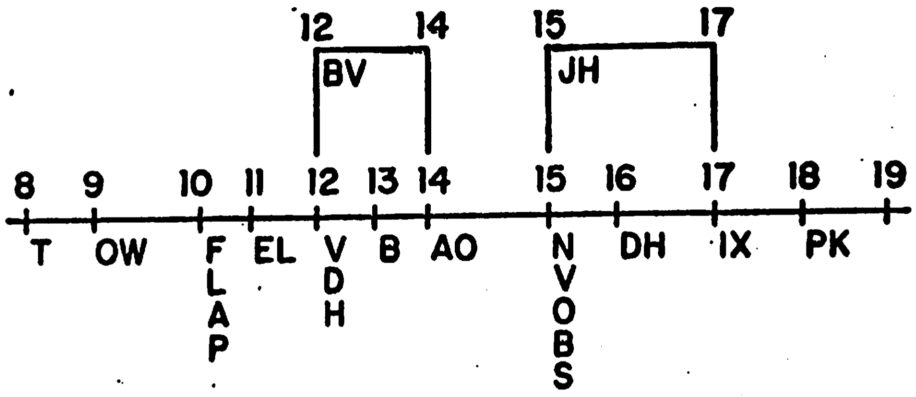

Figure 3. A segment lattice obtained from acoustic-phonetic recognition of a speech signal for "total budget".

phonemes for a given segment, but we also offer alternative segmentations, as for example in the case of 12-14 and 15-17.

Given a segment lattice and a specified sequence of intervals within that lattice, lexical retrieval can return the n best words falling in a given syntactic category for that region. The verifier can proceed through an analysis-by-synthesis process to check the extent to which the parameters support a given lexical hypothesis. Clearly, these processes of lexical retrieval and the verifier must be based on a specified vocabulary stored in the dictionary. In HWIM, this dictionary is produced by expansion from an ordinary pronouncing dictionary and a set of formal phonological rules. Although we will not go into them here, the mechanisms sketched at the top of Figure 2 are components that actually answer questions, compare synthetic spoken utterances for these responses. We shall now turn to the linguistic component, represented by parser and trip in the lower left-hand portion of Figure 2. In the current implementation, the parser contains an ATN grammar that expresses syntactic, semantic, and pragmatic constraints on possible sentences and which interfaces to a manifestation of the TRIP system for specific factual knowledge such as who has given first and last names, who is taking what trips, etc. The important feature of this linguistic component is that it has to work with sequences of words which have reached a certain confidence level whether or not they form a complete well-formed constituent of a sentence. Thus, the job of the parser is to take an arbitrary sequence of words and judge whether it could be a subsequence of a complete syntactically/semantically/pragmatically appropriate sentence.

While acoustic phonetic recognition is driven bottom-up by the input from the microphone, the verifier, lexical retrieval, and parser are invoked at appropriate times by the speech control system. We shall have more to say of this control below, after looking at some of these systems in more detail.

Lexical retrieval makes use of a nondeterministic discrimination net. The basic form of such a net is shown in Figure 4, where we see that a branch is taken for each possible initial phoneme, from which there follows a branch for each possible phoneme which can follow it, and so on and so on. In this way, we could -- given completely accurate information about the sequence of phonemes -- reach any word in a number of steps proportional to the length of the word, irrespective of how many words are contained in the vocabulary. Of course, when we do not have single phoneme hypotheses but instead have likelihoods, we can work sequentially through the words, pushing down the stack those initial segments that have a low overall likelihood, while pursuing further through the net those with a high likelihood, until a set of high likelihood words is obtained which covers regions of the segment lattice.

One catch with this is that the pronunciation of a phoneme is highly context dependent. One can certainly handle this for within-word context by designing the tree appropriately. But inter-word effects seem to pose a different problem, given that

one does not know what the adjacent words are when lexical retrieval is being applied. Jack Klovstad, a member of the HWIM project [Woods et al., 1976], discovered an elegant solution to this problem indicated in Figure 5. Here we see that the net is 'wrapped around' so that inter-word effects can be represented. For example, 'hand' usually ends with a 'd', but if it is followed by another word, that 'd' may be dropped. Thus, the two-word sequence 'hand label' may be pronounced as 'hanlabel', and we see this indicated in Figure 5 by the fact that the final d in 'hand' is recognized by the 'wrap around' network numbered 2 at the left-hand side of the figure, which can alternatively accept an 'l' and jump to the place in the main discrimination net corresponding to having matched an initial 'l' of a subsequent word. The reader may wish to follow through other 'wrap-arounds' to see how this convention is used in other cases.

The verification component is based on an analysis-by-synthesis approach to word and phrase matching. The pronunciation templates are generated by synthesis-by-rule, which allows a large vocabulary to be generated with relatively low storage, taking into account the contextual dependence in continuous speech, and the speaker dependence. The templates thus generated are given via a spectral model which is then compared with the parametric version of the signal to produce a spectral distance measure. More details of the verifier and the other components of the system can be found in Woods et al. [1976].

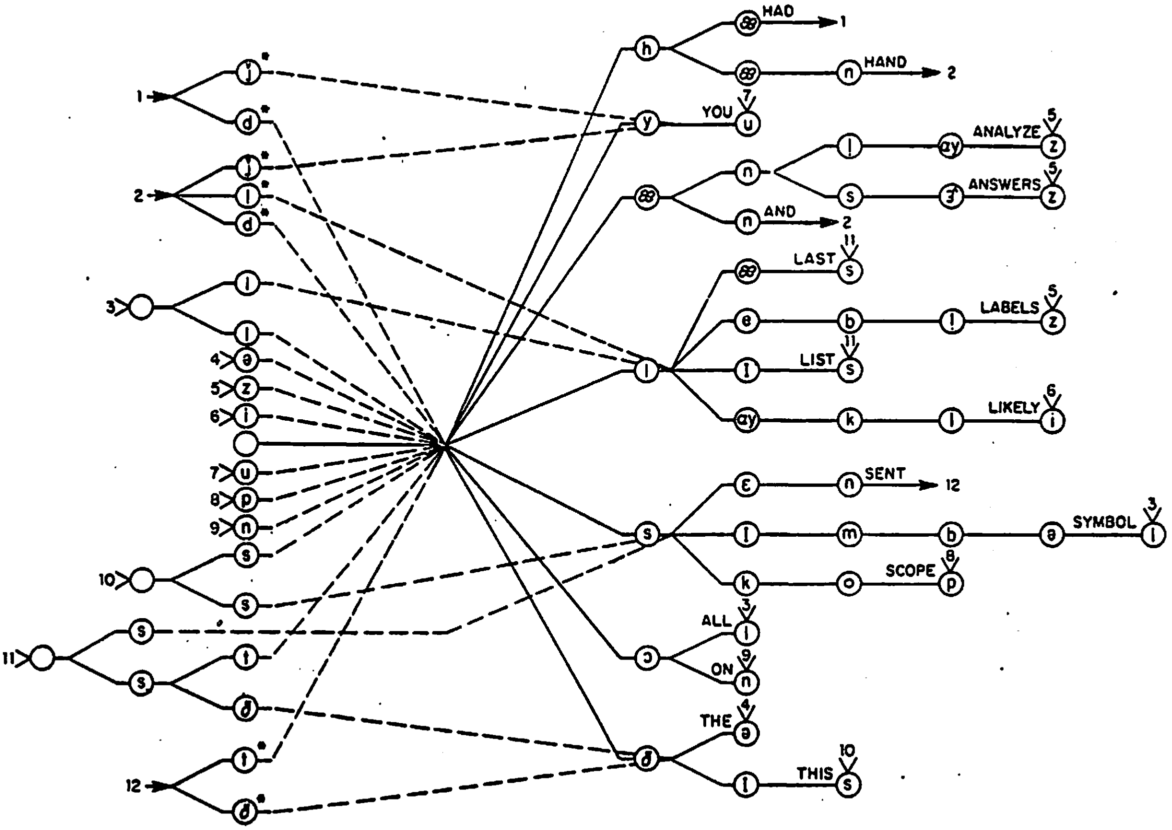

Figure 5. A "wrap-around" discrimination net which takes account of between-word context.

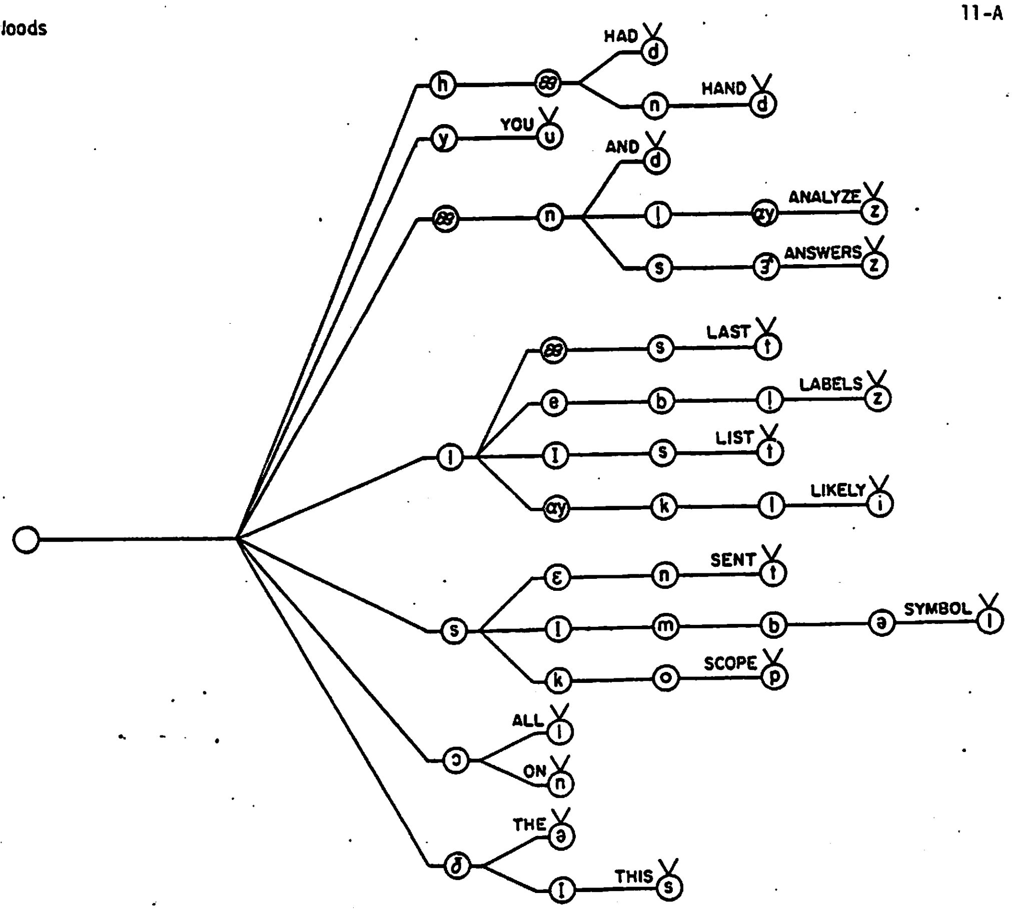

Figure 4. A discrimination net for word recognition.

Before going on to see the ways in which hypotheses are formed for various sequences of words within the utterance, and how these hypotheses are grown until one finally has an acceptable hypothesis for the utterance as a whole, let us briefly see what the parser does. Consider the output of the parser given the sequence of words "What is the plane fare to San Diego". The parse tree that will be produced looks as follows:

```
S Q
  SUBJ NP DET ART THE
          ADJ PLANE
          N FARE
          PP PREP TO
             NP NPR LOCATION CITY SAN@DIEGO
          FEATS NU SG
  AUX TNS PRESENT
      VOICE ACTIVE
  VP V BE
     OBJ NP PRO WHAT
            FEATS NU SG/PL
```

The form of the figure is to be interpreted as a branching tree laid on its side with its root at the top left. The Q of the first line indicates that the overall sentence is taken to be a question. We then see that the subject is a noun phrase which is "the plane fare to San Diego". Note that the syntax here already contains semantic information, since it indicates that San Diego is actually a location and a city. The expressions FEATS NU SG and FEATS NU SG/PL indicate that their respective noun phrases have syntactic features Number: Singular and Number: Singular/Plural.

The semantic interpretation provided by the parser takes the following form:

```
(FOR:  THE A0007 / (FINDQ: LOCATION (CITY SAN@DIEGO))
     : T ; (FOR: THE A0009 / (FINDQ: DB/FARE (DESTINATION A0007)
                                      (STARTING/POINT BOSTON)
                                      (MODE/OF/TRANSPORT PLANE))
               : T ; (OUTPUT; A0009)))
```

This form has rephrased the question as a program to retrieve the appropriate answer. The first line instructs the question answering system to set variable A0007 to the location of the city San Diego. The next line tells the system to set variable A0009 to the plane fare for travel to this destination (San Diego) from the starting point of Boston (note the standing assumption based on the fact that the HWIM system was designed in Cambridge, Massachusetts). The last line instructs the system to provide as answer to the question the value so computed for variable A0009.

It would burden the paper unduly to describe in detail the ATN (Augmented Transition Network) grammar and semantic net used by the parser. However, their content can be indicated by a brief look at Figures 6 and 7. Figure 6 indicates the ATN grammar fragment for date expressions in HWIM. (For details on ATN grammars, see Woods [1970].) The grammar will recognize a sequence of words as being a date expression if it can use these words to sucessfully move from the initial node marked DATE to the final node marked DATE/DATE at which the final popping out of that level of analysis can take place. For example, 13 May will be accepted by following the path PUSH NUMBER (must be less than 32) from DATE to DATE/DAY, by following the path CAT MONTH (recognizing that May falls in the category of a 'month') to then pass to the node DATE/MO, and then taking two jumps that require no further input to get to the final node. On the other hand, 13 May 23 will not be accepted, because the PUSH NUMBER transition from DATE/MO to DATE/NUM can only be done if it was not done during the first transition. To get some idea of the nondeterminism of the graph, note that 'Wednesday' could be a complete date expression, or could be the first word of a compound expression such as 'Wednesday the third of May, 1979'. The reader should check that for each of a wide range of date expressions, there is a valid path through the net, while illegal perturbations of such expressions cannot be accomplished through that net. Thus, indeed, passage through this ATN does provide an appropriate representation of the syntax of date expressions.

Turning now to Figure 7, we see that basic 'meaning relations' are embodied within this net. For example, we may see that Bill is a member of the set of people, that people can

provide one of the arguments to 'the concept of requesting', for which another argument is 'goodies', of which 'resources' form a subset, and amongst the properties of 'resources' are that they have an 'owner'. This figure represents the kinds of factual information of which HWIM is aware and which are used to answer questions and to perform certain tests invoked by conditions on the arcs of the ATN grammar. For example, the semantic network is consulted by the ATN when it recognizes a person's name to see if it knows anyone by that name. See Woods [1975] for a general discussion of semantic networks.

With this background we can now briefly sketch the <u>control strategy</u> embodied in the HWIM speech understanding system. Briefly, the stagegy is one of incremental development of the most likely interpretation of a speech signal as a result of stimulus-suggested word hypotheses, refined by the addition of new words, subject to the constraints of a formalized model (grammar) of possible interpretations. Initially, the segment lattice is generated, and on this basis lexical retrieval will provide an initial set of high-likelihood word hypotheses. A control system can then call upon the linguistic component to use both its pragmatic grammar (as exemplified in Figure 6) and its knowledge base (as exemplified in Figure 7) to come up with hypotheses which incorporate some of the words previously suggested by the lexical retrieval, but which may also hypothesize further words. On this basis, control can then call the verifier to see whether in fact the newly hypothesized words

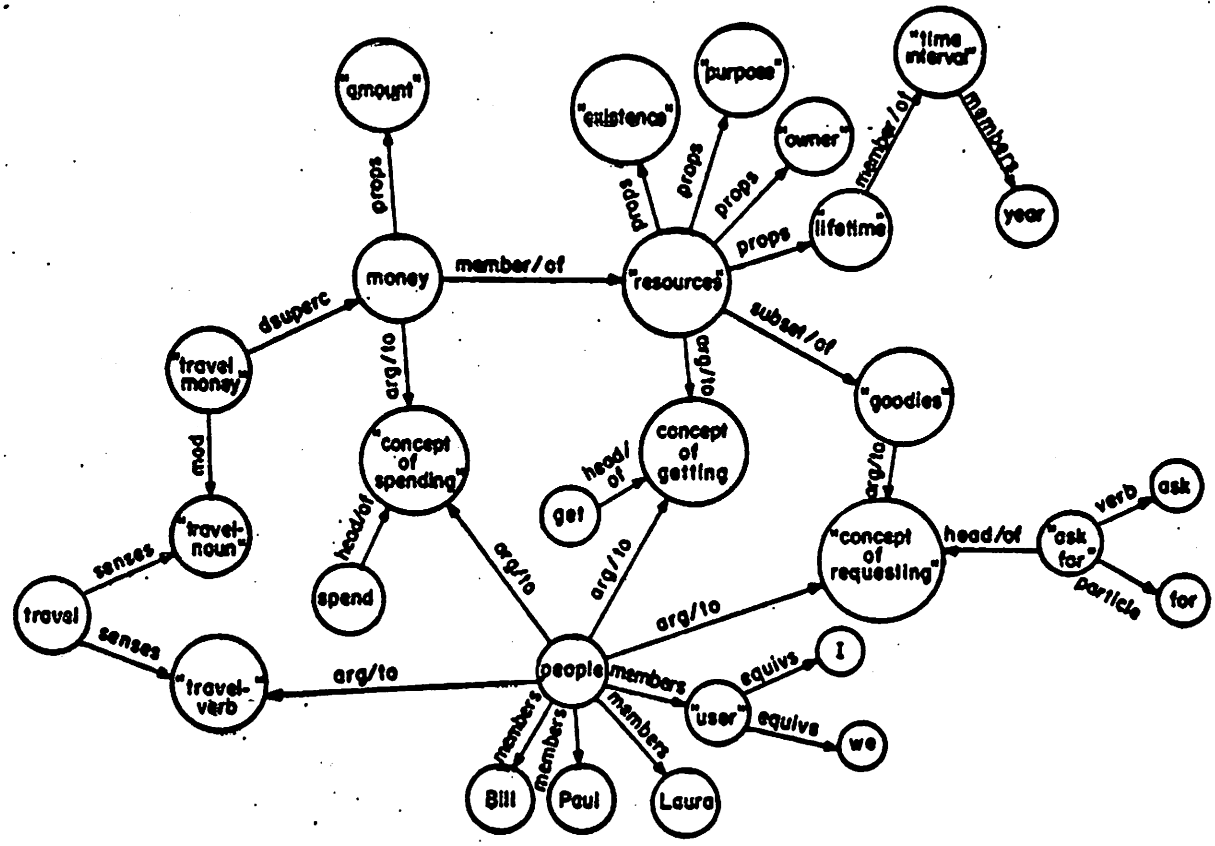

Figure 7. A fragment of "Travelnet", the semantic net for representing HWIM's knowledge of the travel microworld.

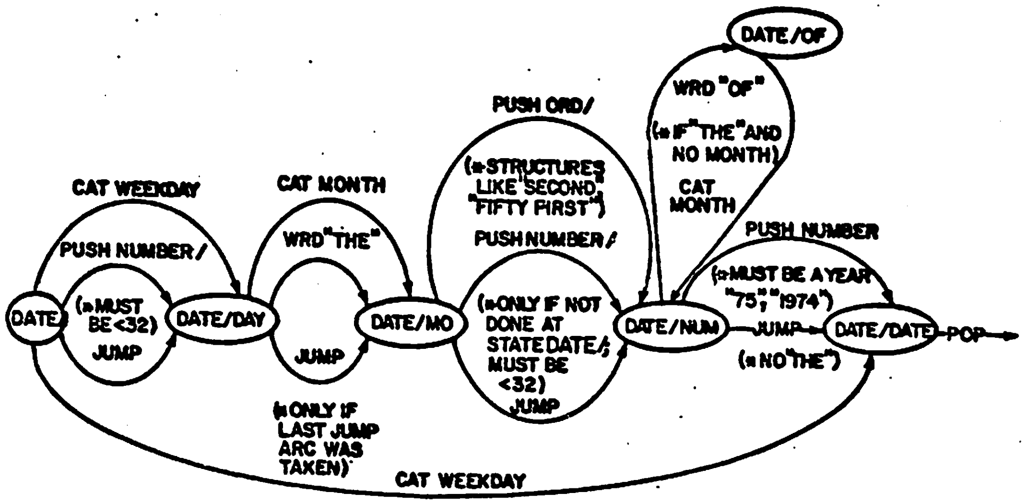

Figure 6. The ATN Grammar Fragment for Time Expressions.

are supported by the segment lattice. And so the process iterates.

The task of the system may be characterized as being high-level perception, in distinction from low-level perception in which one of a small number of stimuli must be recognized. In the present task environment, the system must recognize any member of a potentially immense class constructed from elementary objects via well-formedness rules. Thus, as we have already stressed before, then, analysis must proceed not by enumeration, but by <u>successive</u> <u>refinement</u> of partial hypotheses.

We use the term <u>theory</u> to refer to a partial hypothesis together with its current evaluation or likelihood level. The control strategy for the growth of such theories, working towards a complete spanning theory to provide the interpretation of the entire utterance, is sketched in Figure 8. On the basis of the initial scan, the segment lattice is formed, and lexical retrieval provides 'seed theories' which are the initial, stimulus-driven, hypotheses based on the purely bottom-up analysis. The resultant seed events are placed on the event queue, ordered by 'priority', where they will be joined by other events as processing continues. Events on this queue may be viewed as incipient theories. They consist of either 1) seed events, 2) theories together with adjacent words hypothesized to extend them, or 3) a combination of two 'islands' with a word that joins them. The priority of events on the queue can be determined by a number of methods all based on a semi-Bayesian

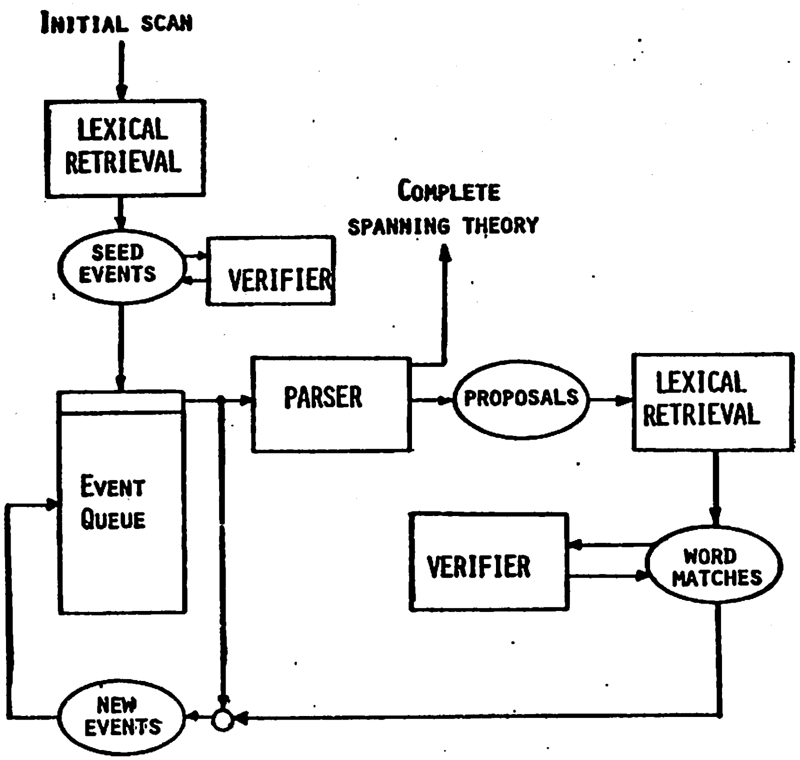

Figure 8. Control Strategy of the HWIM System.

analysis of the likelihoods of the alternative hypotheses given the evidence considered. One of the most interesting methods, called 'shortfall density', when combined with an appropriate search strategy, can guarantee that the first complete spanning theory found will have the highest likelihood of being correct [Woods, 1977]. The events on the queue are maintained in priority order, and the parser at each stage will take the event of highest priority, and check whether in fact it could be part of a syntactically/semantically/pragmatically correct sentence. One way of forming a new event is then to hypothesize that a new word be added to one end or the other of an existing theory. Another process, called 'island collision', occurs when theory1 is posited to be extended to the right by a word which is also posited to extend theory2 to the left, thus suggesting as a new event the concatenation theory1-word-theory2.

This control, based as it is on an event queue ordered by some priority score, does not work necessarily from left to right, but instead tends to work out from 'islands of reliability'. The algorithm does not guarantee that those hypotheses which are processed early on will prove to be correct, but some strategies (e.g. 'middle-out shortfall-density with island collisions') do guarantee that the search will eventually find the best complete spanning theory which provides an interpretation of the entire utterance.

Finally, we note that our serial computer strategy uses the priority ordering to tell what to do next; we would speculate

that analogs of such rankings may be used within the parallel computations of the brain to determine how resources are to be allocated among the various hypotheses.

3. A SAMPLE ANALYSIS OF AN UTTERANCE

In this final section, we shall briefly look at the analysis of an utterance to get a better feel for the control strategy indicated in Figure 8. The utterance to be analyzed is "Do we have a surplus?", and the analysis that we shall follow is fairly typical, save that it is much shorter and has less branching than what one would encounter on the average. Figure 9 appears to be overwhelming, but we hope to convince the reader that it is quite comprehensible. Look, first, at the first panel, in which we see 15 theories. For each theory we list its priority score, and the region of the utterance that supports it. In this example, the search strategy used is called 'left-hybrid shortfall density' and consists of using seed events near the left end of the utterance with a shortfall-density priority score.

You will note that the theories are arranged in descending order of their score, and we have shown the top 15 of the total of 54 seed theories. To make it easier for you to keep track of the processing, we have marked the correct theories with an exclamation mark, and you will note that many incorrect seed theories have higher priority than the correct seed theories

①

```
 #    SCORE   REGION   THEORY
 1     3.53    1-5     WHO
 2     1.92    3-6     WE !
 3     0.0     0-1     -PAUSE- !.
 4    -2.43    2-3     A
 5    -3.24    5-10    ELEVEN
 6    -4.32    5-9     IRAQ
 7    -5.36    1-3     HER
 8    -6.00    1-4     WHOLE
 9    -6.18    1-5     DO !
10    -6.21    3-6     WERE
11    -6.53    3-7     WORK
12    -6.85    1-4     HIS
13    -7.00    1-5     HOW
14    -7.12    1-6     HAWAII
15    -7.21    3-6     WHERE
          + 39 ADDITIONAL EVENTS
```

②

```
 #    SCORE   REGION   THEORY
 1     1.92    3-6     WE !
 2     0.0     0-1     -PAUSE- !
 3    -2.43    2-3     A
 4    -3.24    5-10    ELEVEN
 5    -4.32    5-9     IRAQ
 6    -5.36    1-3     HER
 7    -6.00    1-4     WHOLE
 8    -6.18    1-5     DO !
 9    -6.21    3-6     WERE
10    -6.40    1-7>    [WHO IS   ←
11    -6.53    3-7     WORK
12    -6.85    1-4     HIS
13    -7.00    1-5     HOW
14    -7.12    1-6     HAWAII
15    -7.21    3-6     WHERE
          + 41 ADDITIONAL EVENTS
```

③

```
 #    SCORE   REGION   THEORY
 1     1.82   <1-6     DO WE !    ←
 2     1.12    3-6     [WE        ←
 3     0.0     0-1     -PAUSE- !
 4    -1.13   <2-6     ARE WE     ←
 5    -1.96   <2-6     WILL WE    ←
 6    -1.96   <2-6     WERE WE    ←
 7    -2.43    2-3     A
 8    -3.20   <2-6     CAN WE     ←
 9    -4.03    5-10    ELEVEN
10    -5.26    5-9     IRAQ
11    -5.36    1-3     HER
12    -6.00    1-4     WHOLE
13    -6.18    1-5     DO !
14    -6.21    3-6     WERE
15    -6.43    1-7>    [WHO IS
          + 47 ADDITIONAL EVENTS
```

④

```
 #    SCORE   REGION   THEORY
 1     1.12    3-6     [WE !
 2     1.10    1-9>    [DO WE HAVE !  ←
 3     0.0     0-1     -PAUSE- !
 4    -1.13   <2-6     ARE WE
 5    -1.96   <2-6     WILL WE
 6    -1.96   <2-6     WERE WE
 7    -2.43    2-3     A
 8    -2.83   <0-6     WHAT DO WE   ←
 9    -3.20   <2-6     CAN WE
10    -4.03    5-10    ELEVEN
11    -5.26    5-9     IRAQ
12    -5.36    1-3     HER
13    -6.00    1-4     WHOLE
14    -6.18    1-5     DO !
15    -6.21    3-6     WERE
          + 48 ADDITIONAL EVENTS
```

⑤

```
 #    SCORE   REGION   THEORY
 1     1.10    1-9>    [DO WE HAVE !
 2     0.0     0-1     -PAUSE- !
 3    -1.13   <2-6     ARE WE
 4    -1.96   <2-6     WILL WE
 5    -1.96   <2-6     WERE WE
 6    -2.43    2-3     A
 7    -2.65    3-9     [WE WENT   ←
 8    -2.83   <0-6     WHAT DO WE
 9    -3.20   <2-6     CAN WE
10    -4.03    5-10    ELEVEN
11    -5.26    5-9     IRAQ
12    -5.36    1-3     HER
13    -6.00    1-4     WHOLE
14    -6.18    1-5     DO !
15    -6.21    3-6     WERE
          + 51 ADDITIONAL EVENTS
```

⑥

```
 #    SCORE   REGION   THEORY
 1      .99    1-10>   [DO WE HAVE A        ←
 2     0.0     0-1     -PAUSE- !
 3    -1.13   <2-6     ARE WE
 4    -1.55    1-10>   [DO WE HAVE EIGHT    ←
 5    -1.96   <2-6     WILL WE
 6    -1.96   <2-6     WERE WE
 7    -2.31    1-11>   [DO WE HAVE LESS     ←
 8    -2.43    2-3     A
 9    -2.50    1-13>   [DO WE HAVE OVER     ←
10    -2.65    3-9     [WE WENT
11    -2.83   <0-6     WHAT DO WE
12    -2.84    1-13>   [DO WE HAVE UNDER    ←
13    -3.20   <2-6     CAN WE
14    -3.67    1-10>   [DO WE HAVE MORE     ←
15    -4.27    5-10    ELEVEN
          + 56 ADDITIONAL EVENTS
```

⑦

```
 #    SCORE   REGION   THEORY
 1     0.0     0-1     -PAUSE- !
 2    -1.13   <2-6     ARE WE
 3    -1.27    1-17>   [DO WE HAVE A SURPLUS !  ←
 4    -1.55    1-10>   [DO WE HAVE EIGHT
 5    -1.96   <2-6     WILL WE
 6    -1.96   <2-6     WERE WE.
 7    -2.31    1-11>   [DO WE HAVE LESS
 8    -2.43    2-3     A
 9    -2.50    1-13>   [DO WE HAVE OVER
10    -2.65    3-9     [WE WENT
11    -2.83   <0-6     WHAT DO WE
12    -2.84    1-13>   [DO WE HAVE UNDER
13    -3.20   <2-6     CAN WE
14    -3.30    1-15>   [DO WE HAVE A THOUSAND  ←
15    -3.67    1-10>   [DO WE HAVE MORE
          + 57 ADDITIONAL EVENTS
```

⑧

```
 #    SCORE   REGION   THEORY
 1    -1.13   <2-6     ARE WE
 2    -1.27    1-17>   [DO WE HAVE A SURPLUS !
 3    -1.55    1-10>   [DO WE HAVE EIGHT
 4    -1.96   <2-6     WILL WE
 5    -1.96   <2-6     WERE WE
 6    -2.31    1-11>   [DO WE HAVE LESS
 7    -2.43    2-3     A
 8    -2.50    1-13>   [DO WE HAVE OVER
 9    -2.65    3-9>    [WE WENT
10    -2.83   <0-6     WHAT DO WE
11    -2.84    1-13>   [DO WE HAVE UNDER
12    -3.20   <2-6     CAN WE
13    -3.30    1-15>   [DO WE HAVE A THOUSAND
14    -3.67    1-10>   [DO WE HAVE MORE
15    -4.21    0-3>    [-PAUSE- HER   ←
          + 64 ADDITIONAL EVENTS
```

⑨

```
 #    SCORE   REGION   THEORY
 1    -1.27    1-17>   [DO WE HAVE A SURPLUS !
 2    -1.37    2-6     [ARE WE   ←
 3    -1.55    1-10>   [DO WE HAVE EIGHT
 4    -1.96   <2-6     WILL WE
 5    -1.96   <2-6     WERE WE
 6    -2.31    1-11>   [DO WE HAVE LESS
 7    -2.43    2-3     A
 8    -2.50    1-13>   [DO WE HAVE OVER
 9    -2.65    3-9>    [WE WENT
10    -2.83   <0-6     WHAT DO WE
11    -2.84    1-13>   [DO WE HAVE UNDER
12    -3.20   <2-6     CAN WE
13    -3.30    1-15>   [DO WE HAVE A THOUSAND
14    -3.67    1-10>   [DO WE HAVE MORE
15    -4.21    0-3>    [-PAUSE- HER
          + 64 ADDITIONAL EVENTS
```

Figure 9. Nine successive views of the top of the event queue in recognizing the utterance "Do we have a surplus?"

before higher level syntactic and semantic processes are brought into play. Now, as we invoke these higher level processes, new theories will be developed, and these theories are to be inserted into the event queue on the basis of their priority ordering. For example, we see that panel 2 of Figure 9 is obtained from panel 1 by inserting the theory "[WHO IS" between "WERE" and "WORK" on the basis of its score which is intermediate between theirs. Arrows single out the new theories in each panel. Note that "WHO" has disappeared from the event queue after its use to construct this new theory "WHO IS". We shall describe in more detail the process whereby the new theories are created, but for the moment let us just make sure that we understand the way in which these theories, once created, are inserted into the event queue. The transition from panel 2 to panel 3 is based on the creation of at least 6 new hypotheses together with their scores, and their insertion in the event queue in the appropriate priority ordering. Note that the hypothesis "WE" has been removed, but that amongst the new hypotheses which subsume it is "[WE", namely the hypothesis of "WE" not simply as a word somewhere in the sentence, but of "WE" as the initial word of the sentence. The reader should now be able to follow the way in which the remaining panels are formed from their predecessors. The arrows indicate the items that are added. Items are removed from the queue as they are used to form new hypotheses, and some are pushed down the queue by new higher priority events. The process continues until, in this case in panel 9, we come up with

a theory that not only reaches the top of the stack but also covers the entire utterance. When the top event of panel 9 is evaluated and determined to produce a complete spanning theory, it is produced as the interpretation and no new events are added to the queue. (Clearly, the process could be continued to produce second best interpretations and so on.)

In growing new events from old, the system has the following options:

> 'Doing left-end event' forms the hypothesis that the event comes at the beginning of an utterance, and indicates this by placing a '[' in front of the event; similarly 'doing right-end event' places a ']' after the event.
>
> 'Noticing on the right' adds a new word at the right end of an event; while 'noticing on the left' adds a new word at the left end of the event.

In what follows, we shall simply indicate what new events are proposed at each stage.

In getting from panel 1 to panel 2, the system starts from the top hypothesis "WHO" of panel 1; does a left-end event to create the proposal "[WHO", and then performs noticing on the right to create "[WHO WILL", "[WHO WENT", and "[WHO IS". Of these, only "[WHO IS" has sufficiently high priority rating to enter the top 15 items of the stack as shown in panel 2.

The system then takes the event "WE" from the stack as shown in panel 2 and, by noticing on the left, creates 7 new hypotheses of which 5 enter the top 15 items of the stack as shown in panel 3; and by noticing left-end event, forms the new theory "[WE" which enters second place of the stack at that time. Note that "WE" has now been subsumed in the other hypotheses and is removed from the stack. The stack contains only a list of hypotheses that have not yet been fully considered.

At the next stage, "DO WE" is removed from the top of the stack and the system provides 3 new theories: "WHAT DO WE" by noticing on the left; "[DO WE" by doing a left-end event; and then "[DO WE HAVE" by noticing on the right. "[DO WE" is extended to [DO WE HAVE without creating an intermediate event on the event queue since its score is unchanged by the addition of the left-end hypothesis.

By now, the reader should be able to see the processes that led to the hypotheses which enter the stack in each succeeding panel, and to understand why other hypotheses have been removed from the stack. It's worth noting that the correct, but rather unilluminating, "-PAUSE-" comes to the top of the stack at panel 7, but that an incorrect hypothesis temporarily bubbles to the top of the stack in panel 8.

The correct hypothesis "[DO WE HAVE A SURPLUS" enters the stack in almost final form in panel 7, when "[DO WE HAVE A SURPLUS" is formed from "DO WE HAVE A" by noticing on the right. However, it is not until "[DO WE HAVE A SURPLUS" comes to the top of the stack as shown in panel 9 that we can finally do a right-end event to form "[DO WE HAVE A SURPLUS]", which at last provides a complete parse and thus the spanning theory which provides the interpretation of the entire utterance.

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From AI to Neurolinguistics[1]

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Abstract

We start with a discussion of the status of the methodology of cooperative computation in neural modelling, suggesting that it will prove valuable both for pencil-and-paper analysis and for detailed computer implementation. We then complement Wood's discussion of HWIM in Chapter 1 by discussing HEARSAY, and AI speech-understanding system based on the cooperation of multiple processes, and a set of different levels of representation of linguistic information. We then discuss several important conceptual issues involved in adapting this purely AI model to provide a cooperative computation methodology adapted to the needs of neurolinguistic theory.

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1. Introduction

My aim in this paper is to suggest that certain concepts from computer science may come to enrich neurolinguistics. At the outset, then, let me stress that the brain is not a serial computer, with some executive knot of control bringing in one subsystem at a time. Rather, the brain is made up of a number of continually active regions which constrain each other by passing signals back and forth. In Section 1, we briefly sketch out what we mean by cooperative computation as the embodiment of this style of the brain. Unfortunately, there are no thoroughgoing analyses of neurolinguistics based on this style. To build a bridge, we analyze HEARSAY, a speech understanding system which is designed for serial computation but which does, nonetheless, exhibit "a multi-level organization for problem-solving using many, diverse, cooperating sources of knowledge" [Erman and Lesser, 1975]. We shall explore the commonalities of HEARSAY with the HWIM system described by Woods in this volume. More importantly, we shall analyze the extent to which HEARSAY falls short of both psychological and neurological validity to outline what a "neurological HEARSAY" might look like as a goal for computational neurolinguistic modelling. Warren McCulloch often urged his audience, "Don't look at the end of my finger, look where I'm pointing." HWIM and HEARSAY are not neurolinguistic models, but they do enable us to point to certain features which such models should embody in the future. In the companion paper in this volume, "Sensorimotor Processes and the Neural Basis of Language", we stress commonalities between language and other forms of

perception and motor control to suggest how neurolinguistics may make contact with the synapse-cell-circuit analysis that lies at the heart of modern neuroscience.

2. Cooperative Computation as the Style of the Brain

We must distinguish AI (artificial intelligence) from BT (brain theory), where we go beyond a model of the overall input-output behavior of a system to one in which various processes are mapped onto anatomically characterizable portions of the brain. Such regions of the brain continually exhibit neural activity. We thus cannot use models of serial computation in Brain Theory if these would ascribe activity to a single region at any time. Rather, we must seek to model cooperative computation in which concurrent activity in many subsystems is so constrained by the interaction of these subsystems as to yield the overall attainment of a global goal of the system as a whole despite the "local view" of each subsystem.

Warren McCulloch's concept of "redundancy of potential command" led to the Kilmer-McCulloch model of mode selection in reticular formation (1969), and the Didday model of frog visuomotor activity (1960, 1976) which is discussed in Chapter X. These are both models of cooperative computation in the brain, and are summarized in Arbib (1972, Chapter 7). We may refer to these models as examples of the C^2-style of modelling (C^2 for "Cooperative Computation"). In Section 4, we shall introduce a "neurologized HEARSAY" model which attempts to fit the linguistic aspects of HEARSAY into the pre-existing (albeit still elementary) framework of cooperative computation in Brain Theory. To give a further example of the C^2-style of modelling, this time in a hypothetical visual system, the reader may consult the evolutionary account in Chapter X of a system for computing optic flow. Some general issues of cooperative computation in both AI and BT are taken up in Section 5 of (Arbib, 1979).

It is important to distinguish the use of the computer as a <u>tool</u> for data-processing and simulation from the use of the <u>concepts</u> of computation for the analysis of complex systems. A C^2-model of brain may be analyzed by pencil-and-paper without computer implementation, but can still provide valuable insights as the subsystem interactions are constrained to yield a model which details mechanisms for neurological processes, and which goes on to define ways in which disruptions occur such that the distorted performance maps onto the clinical data. What all this says is that when we advocate the cooperative computation style for Brain Theory, we accept and even encourage that much of that analysis will be of the pencil-and-paper kind, without recourse to computer simulation to explore the implications of the model.

Marr and Poggio's (1977) notion of the "near independence" of different levels of analysis and the notion of top-down design in computer programming (Alagić and Arbib, 1978) both suggest that it is fruitful to describe subsystems in a language which is relatively independent of the details of implementation -- whether the latter be in terms of neural circuitry in the brain, or the machine language of some computer. Thus a brain-theoretic analysis at the regional level may, to a first approximation, be formalized in an abstract language of cooperative computation. The interactions and internal state transitions posited in this model can then be implemented within some computer programming language. But if the program is properly designed, runs of the program test the high-level model, not the details introduced in the implementation. In the same way, a simulation of a dynamical system described by Newton's laws may involve the choice of a Runge-Kutta method for numerical integration of the differential equations, but the resultant trajectories should not depend, save perhaps in fine detail, on the numerical analysis chosen. If distinctions of this kind are rigidly maintained, we can clearly distinguish the use of computation concepts as content in a theory from the concepts used in implementing the theory in a computer program.

3. The HEARSAY Speech Understanding System

This section provides an exposition of HEARSAY (Erman and Lesser, 1975, 1980; Lesser et al., 1975), an AI speech-understanding system which is explicitly based on the cooperation of multiple processes. It has not yet fed into psycholinguistic, let alone neurolinguistic, theory but we shall indicate in Section 4 a number of ways the model could be modified to better fit it as a test-bed for simulating the neural basis of language.

The HEARSAY-II system has a very explicit representational structure, based on a set of different levels of representation. The raw data, whose interpretation is the task of the system, are represented at the "parameter" level as a digitized acoustic signal. The system will, via intermediate levels, generate a representation at the "phrasal" level of a description in terms of a grammar which contains both syntactic and semantic constraints. The combination of phrasal and lexical information can then be used to generate the appropriate response to the verbal input, e.g., by retrieving the answer to a question from some data-base stored within the computer.

HEARSAY uses a data structure, called the "blackboard", which is partitioned into the various levels. At any time in the system's operation, there are a number of hypotheses active at the various levels, and there are links between hypotheses at one level and those they support at another level. For example in Figure 1 we see a situation in which there are two surface-phonemic hypotheses 'L' and 'D' consistent with the raw data at the parameter level, with the 'L' supporting the lexical hypothesis "will" which in turn supports the phrasal hypothesis "question", while the 'D' supports 'would' which in turn supports the 'modal question' hypothesis at the phrasal level. Each hypothesis is indexed not only by its level but also by the time segment over which it is posited to occur, though this is not explicitly shown in the figure. We also do not show the "credibility rating" which is assigned to each hypothesis.

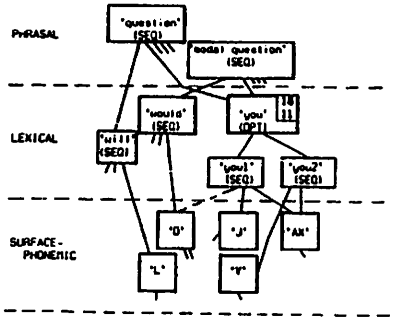

Fig. 1. Multiple hypotheses at different levels of the HEARSAY blackboard (Lesser et al., 1975).

HEARSAY embodies a strict notion of constituent processes, and provides scheduling processes whereby the activity of these processes and their interaction through the blackboard data base is controlled. Each process is called a knowledge source (KS), and is viewed as an agent which

embodies some area of knowledge, and can take action based on that knowledge. Each KS can make errors and create ambiguities. Other KS's cooperate to limit the ramifications of these mistakes. Some knowledge sources are grouped as computational entities called modules in the final version of the HEARSAY-II system. The knowledge sources within a module share working storage and computational routines which are common to the procedural computations of the grouped KS's.

HEARSAY is based on the "hypothesize-and-test" paradigm which views solution-finding as an iterative process, with each iteration involving the creation of a hypothesis about some aspect of the problem and a test of the plausibility of the hypothesis. Each step rests on *a priori* knowledge of the problem, as well as on previously generated hypotheses. The process terminates when the best consistent hypothesis is generated satisfying the requirements of an overall solution.

The choice of levels and KS's varies from implementation to implementation of HEARSAY, which is thus a class of models or a modelling methodology rather than a single model. (In fact, the HEARSAY methodology has been used in computer vision with picture point/line - segment/region/object levels replacing the acoustic/phonetic/lexical/phrasal levels of the speech domain (Hanson and Riseman, 1978).) The C2 configuration of HEARSAY-II is shown in Figure 2. We see that each KS takes hypotheses at one level and uses them to create or verify a hypothesis at another (possibly the same) level. In this particular configuration processing is bottom-up from the acoustic signal to the level of word hypotheses, but involves iterative refinement

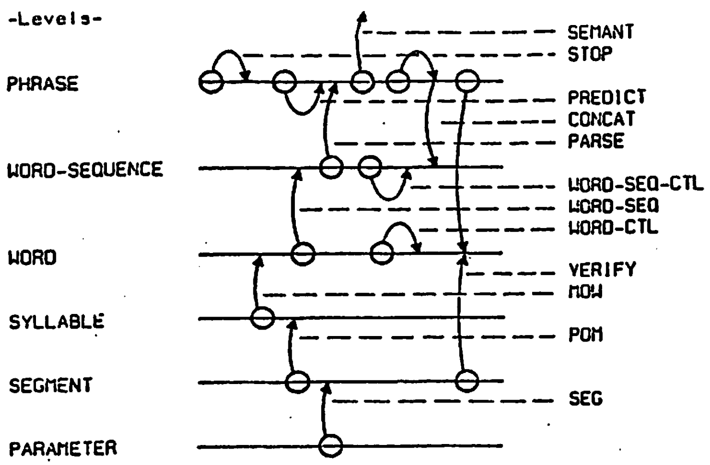

Fig. 2. The C2 configuration of HEARSAY-II. The levels are represented by the solid lines, labelled at the left. The KS's are represented by the circle-tailed arrows, and are linked to their names by the dashed lines. Each KS uses hypotheses at the tail-end level to create or verify hypotheses at the head-end level. (Erman and Lesser, 1979).

of hypotheses both bottom-up and top-down before a phrasal hypothesis is reached which is given a high enough rating to be accepted as the interpretation of the given raw data.

As we have seen, the KS's cooperate in this iterative formation of hypotheses via the blackboard. In HEARSAY, no KS "knows" what or how many other KS's exist. This ignorance with respect to other KS's is maintained to achieve a completely modular KS structure which enhances the ability to test various representations of a KS as well as possible interactions of differing combinations of KS's.

The current state of the blackboard contains all current hypotheses. Subsets of hypotheses represent partial solutions to the entire problem. A subset of hypotheses is defined relative to a contiguous time interval. A given subset may compete with other partial solutions, or subsets having time intervals which overlap the given subset.

We thus regard the task of the system as a search problem. The search space is the set of all possible networks of hypotheses that sufficiently span the time interval of the utterance connecting hypotheses directly derived from the acoustic input to hypotheses which describe the semantic content of the utterance. The state of the blackboard at any time, then, comprises a set of (possibly overlapping) partial elements of the search space. No KS can singlehandedly generate an entire network to provide an element of the search space. Rather, we view HEARSAY as an example of "cooperative computation": the KS's cooperate to provide hypotheses for the network providing an acceptable interpretation of the acoustic data. Each KS may read data; add, delete, or modify hypotheses and attribute values of hypotheses of the blackboard. It also may establish or modify explicit structural relationships among hypotheses. The generation and modification of hypotheses on the blackboard is the exclusive means of communication between KS's.

Each KS includes both a precondition and a procedure. When the precondition detects a configuration of hypotheses to which the KS's knowledge can be applied, it invokes the KS procedure, i.e., it schedules a blackboard-modifying operation by the KS. The scheduling does not imply that the KS will be activated at that time, and/or that the KS will indeed be activated with this particular triggering precondition since HEARSAY uses a "focus of attention" mechanism to stop the KS's from forming an unworkably large number of hypotheses. The blackboard modifications may trigger further KS activity -- acting on hypotheses both at different levels and at different times. Any newly generated hypothesis would be connected by links to the seminal hypothesis to indicate the implicative or evidentiary relationship between them.

There are essentially two operations which generate hypotheses: Synthesis or abstraction which results in additional hypotheses at a higher level based on conjoined lower-level hypotheses which have already been substantially validated; and analysis or elaboration resulting in the creation of lower-level hypotheses which, when verified, tend to confirm or refute a higher-level hypothesis. Analysis or elaboration can also decompose hypotheses from a higher level into more explicit hypotheses whenever stagnation occurs in the system.

A jump over several levels is the equivalent of constructing a major step in the plan, helping to significantly prune the search space. Partial solutions can be combined to create hypotheses which skip several intermediate levels on the blackboard. This concatenation of contiguous partial solutions is validated by consideration of the desirability of the conjoined partial solution at a higher level.

Each hypothesis has an associated set of attributes, some optional, others required. Several of the required attributes are: (i) the name of the hypothesis and its level; (ii) an estimate of its time interval relative to the time span of the entire utterance; (iii) information about its structural relationships with other hypotheses; and (iv) reliability ratings.

The reliability measure reflects the plausibility of a hypothesis. It is calculated by a weighted functional composition of the validity of the hypothesis (an integer between -100 and 100: from maximally implausible to maximally plausible); and the conditional strength of the hypothesis as an inference represented by its implication value (ranging from 100, maximally confirming evidence, to -100, maximally disconfirming evidence). The validity of a hypothesis is a function of the validity of the hypotheses directly supporting it via implicative links and the implicative strengths associated with those links. Changes in validity ratings reflecting creation and modification of hypotheses are propagated automatically throughout the blackboard by a rating policy module called RPOL.

The actual activation of the knowledge sources occurs under control of an external scheduler, the "schedule KS" (Figure 3). The schedule KS constrains KS activation by functionally assessing the current state of the blackboard with respect to the solution space and the set of KS invocations that have been triggered by KS preconditions.

The focusing strategy module, in a manner consistent with the desired search policy (bottom-up, breadth-first, best-first -- determined heuristically and by current state considerations), calculates a weighted functional value based on: (i) validity of the hypothesis on which an inference is based; (ii) the implicative strength of the inference (that of the permanent knowledge on which the inference is based); and (iii) the estimated validity of the results.

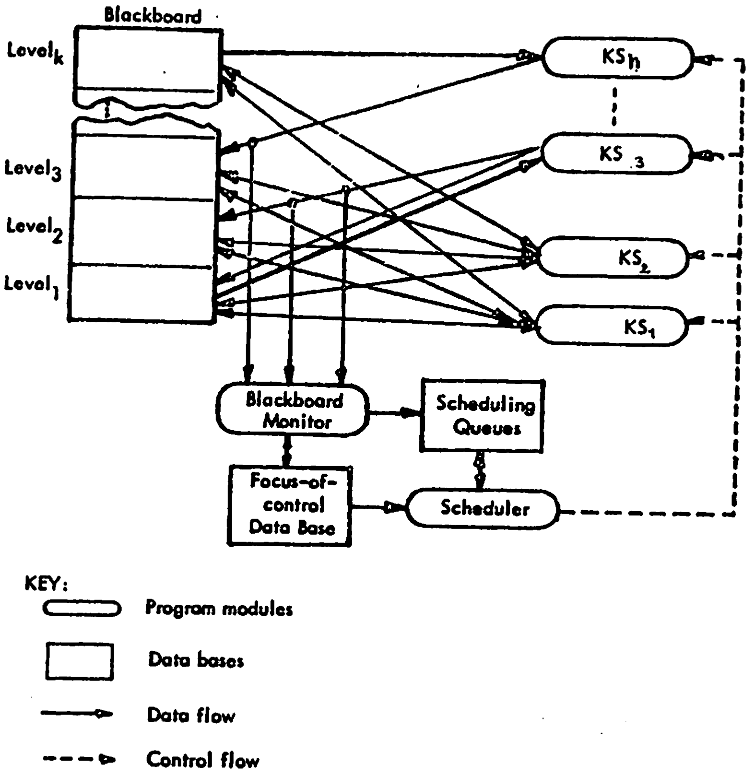

Fig. 3. HEARSAY-II Architecture. The blackboard is divided into levels. Each KS interacts with just a few levels. A KS becomes a candiate for application if its precondition is met. However, to avoid a 'combinatorial explosion' of hypotheses on the blackboard, a scheduler is used to restrict the number of KS's which are allowed to modify the blackboard. (Lesser & Erman, 1979)

The KS having the highest functional value becomes the best-first knowledge source and is the one that is activated by the scheduler (Hayes-Roth & Lesser, 1977). The highest overall rated KS reflects the activation level of the hypotheses relevant to the KS instantiations. The scheduler supplies the external input which actually activates the specific KS most likely to aid in attaining the complete parse of the utterance.

We now give a more detailed account of the syntactic and semantic components of HEARSAY-II. The C2 configuration (September 1976) of the HEARSAY-II system will be used throughout the subsequent description. In HEARSAY-II, both syntactic and semantic operations are implemented via the SASS module. The SASS module is comprised of three independent KS's, PARSE, PREDICT, and CONCAT. It utilizes a semantic template grammar to effect the parse of the utterance. Efficiency considerations led to much of the partial working results being stored internally to SASS. The only information placed on the blackboard at the phrasal level is the recognized and extended word sequences, with their maximal partially-matched template and missing constituents as attributes. Note then that there is much data flow and updating within SASS which is not mediated via the blackboard -- a departure from the initial HEARSAY design procedure. SASS seems to correspond to the input side of Winograd's (1972) system -- if one equates a SASS parse with Winograd's Planner representation. Once the best parse is achieved, KS's SEMANT and DISCO operate to produce the required interpretation and initiate the desired response.

The PARSE KS of the SASS module relies heavily on an ACORN (Automatically Compilable Recognition Network) representation of the semantic template grammar (to be described later). A parse is effected through cooperation in a top-down and bottom-up manner, by extensive interaction among the KS's of the HEARSAY-II system.

The language recognized by HEARSAY-II is described by a semantic template grammar. Each template describes a context-free language of expressions with a common interpretation class. For example, the template $REQUEST ($ precedes a non-terminal) is defined as the sequence

TELL $ME $RE $TOPICS

and describes a set of typical requests which might be made to an information retrieval system.

TELL is a hypothesis at the word level, while $ME, $RE, $TOPICS, and $REQUEST are hypotheses at the phrasal level. Here is an example of data at one level being used to confirm hypotheses at the same level. The system (via the KS's SEMANT and DISCO) must have an explicit access to the hypotheses confirming $REQUEST if the request is to be understood.

A template combines syntax and semantics. It consists of ordered, linked terminal and non-terminal nodes. Each terminal node has associated with it a description of all possible instances. Node names representing networks, i.e. the non-terminal nodes, can occur within a template network. Figure 4 provides an example.

The partial hypotheses which match the terminal elements are combined into phrasal node hypotheses and each has associated attribute values. The grammar is "speech act" based in that in its definition, the frame of an inquiry discourse is explicitly and restrictively utilized. The pragmatics of the discourse permit evaluation of possible sentence types which are likely to occur. This limits the number of possible high-level templates. (The DISCO KS network contains these as its nodes -- there are 13 of them in

```
<SENT> ::= [<SS>]

<SS> ::= <$WHATIS>  THE SIZE OF THE DATABANK
         <$QUANTITY> PLEASE

<$WHATIS> ::= WHAT IS
              WHAT'S

<$QUANTITY> ::= THE <LAST> <$NUMBER 1-99>
                THE <LATEST> <$NUMBER 1-99>
                THE <LATEST>
                <$NUMBER 1-99>

:.

<LAST> ::= LAST
           NEXT
           FIRST

<LATEST> ::= EARLIEST
             LATEST
             NEWEST
             OLDEST
             MOST RECENT

<$NUMBER 1-99> ::= <NUMBER2>
                   <TEENS>
                   <DIGITS>
```

Fig. 4. Part of the template of the AIX15 Semantic Template Grammar. Note that alternatives are represented on separate lines. TEENS, DIGITS, AND NUMBER2 are subsequently rewritten.

addition to the semantic registers.) Each template is expanded with respect to all sentences possible within the narrowly defined problem space of 1011 words in an information retrieval frame. The definition of subsequent nodes and their related substructures is thus determined by grouping like sub-phrases and creating additional subnodes. Ultimately, all possible meaningful combinations of word sequences are evaluated to create tables used by the word sequence KS to produce 'islands of reliability' from the bottom-up acoustic phonetic KS's.

Parsing with a semantic template grammar is used to produce the most consistent network of hypotheses spanning the entire utterance. Once this occurs the SEMANT KS reparses the blackboard information using the same grammar, but incorporating semantic information at the nodes that are parsed. This second parse produces a semantically-tagged representation of the utterance which the DISCO KS, a finite state network of the solution space, decomposes into appropriate actions. DISCO also maintains a pragmatic, current semantic register list to determine pronoun reference and current frame reference in a manner similar to the Winograd system.

In the HEARSAY-II environment, each instantiation of a template is accomplished by a separate execution of the SASS knowledge source. The scheduling execution is controlled by the focussing strategy of the system -- the template supported by the highest rated information will be instantiated first.

The omission of a symbol at word junctures, background noise or poor speaker enunciation result in incorrect words being hypothesized with high ratings, while the correct word may be hypothesized with low ratings, or not at all. Because of the scheduler focussing strategy, a correct word with low validity may never be considered unless the alternatives are exhausted.

According to Hayes-Roth et al. (1977), "present statistics" show that only 80% of the words in the utterance are supplied to the ACORN. Techniques of prediction, partial matching and filtering are used to supplement the bottom-up processes.

This account by no means exhausts the details of HEARSAY-II, but it does make explicit a number of features which suggest that it contains the seeds for a C^2-style of neurolinguistic modelling. First, we see in it the explicit specification of different levels of representation, and an interpretive strategy wherein components interact via the generation and modification of multiple tentative hypotheses. This process yields a network of interconnected hypotheses which supports a satisfactory interpretation of the original utterance. Second, through data-directed activation, KS's can exhibit a high degree of asynchronous activity and parallelism. HEARSAY explicitly excludes an explicitly predefined centralized control scheme though it does use blackboard-driven "priority scheduling" of KS's. The multi-level representation attempts to provide for efficient sequencing of the activity of the KS's in a fashion which provides a serial emulation of computation distributed across a number of concurrently active processors. The decomposition of knowledge into sufficiently simple-acting KS's is intended to simplify and localize relationships in the blackboard.

4. Towards a Neurological HEARSAY

The HEARSAY model lays claim neither to psychological nor to neurological validity as a model of language comprehension. We now observe that incorporating it into a C^2-analysis of neural organization pertinent to language raises several important conceptual questions. We present four of these, along with a brief discussion which sheds light on possible neural mechanisms at this regional level.

First, in HEARSAY, changes in validity ratings reflecting creation and modification of hypotheses are propagated throughout the blackboard by the rating policy module, RPOL. These ratings are the basis for the determination, by a single schedule KS, of which blackboard-manipulating KS will next be activated. This use of a single scheduler seems "undistributed" and "non-neural" -- in a brain region, one may explore what conditions lead to different patterns of activity, but not of scheduling. However, the particular scheduling strategy used in HEARSAY is a reflection of the exigencies of implementing the system on a serial computer. Serial implementation requires us to place a tight upper bound on the number of activations of KS's, since they must all be carried out on the same processor. The HEARSAY methodology can, however, be extended to a parallel "implementation" in which we may view each KS as having its own "processor". In a neural model, each such processor would be located in a different portion of the brain. Such a modufication would require that RPOL be represented as a subsystem within each KS, so that propagation of changes in validity ratings can be likened to relaxation procedures (see, e.g., Arbib, 1979).

Second, we have seen that the processes in HEARSAY are represented as KS's, and that certain KS's may be aggregated into modules. It would be tempting, then, to suggest that in HEARSAY-style implementations of process models such as that of Luria (cf. Arbib and Caplan, 1979, Figure 9), each brain region would correspond to either a KS or a module. Schemas -- the representations of particular words or objects -- would then correspond to much smaller units both functionally and structurally -- perhaps at the level of application of a single production in a semantic template grammar (functionally), or the activation of a few cortical columns (neurally). But a major conceptual problem arises because in a computer implementation, a KS is a program, and it may be called many times -- the circuitry allocated to working through each "instantiation" being separate from the storage area where the "master copy" is stored. But a brain region cannot be copied ad libitum, and so if we identify a brain region with a KS we must ask "How can the region support multiple simultaneous activations of its function?" We may hypothesize that this is handled by parallelism (which presumably limits the number of simultaneous activations). Alternatively we may actually posit that extra runnable copies of a program may be set up in cortex as needed.

The third "non-neural" feature of current HEARSAY implementations is the use of a centralized blackboard. But this is not, perhaps, such a serious problem, for examination shows that the levels of the blackboard are really quite separate data structures, and that they are only linked via KS's. For each level, we may list those KS's that write on that level ("input" KS's) and those that read from that level ("output" KS's). From this point of view, it is quite reasonable to view the blackboard as a distributed structure, being made up of those pathways which link the different KS's. One conceptual problem remains. If we think of a pathway carrying phonemic information, say, then the signals passing along it will encode just one phoneme at a time. But our experience with HEARSAY suggests that a memoryless pathway alone is not enough to fill the computational role of a level on the blackboard; rather the pathway must be supplemented by neural structures which can support a short-term memory of multiple hypotheses over a suitable extended time interval.

Finally, we note that the HEARSAY methodology requires that "no KS knows what or how many other KS's exist" -- a KS is simply activated whenever data meeting its precondition appear on the blackboard, and it writes the result of its procedures at some appropriate level. In particular, this approach is said to preclude one KS directly activating another. But this restriction would seem to vanish once we "neuralize" HEARSAY, for if we decentralize scheduling, and reconceptualize levels as pathways from "input" to "output" KS's, we may certainly view an "input" KS as sending a signal along the pathway to directly activate an "output" KS.

An immediate research project for computational neurolinguistics, then, might be to approach the programming of a truly distributed speech understanding system (free of the centralized scheduling in the current implementation of HEARSAY) with the constraint that it include subsystems meeting the constraints such as those in the reanalysis of Luria's data in Figure 9 of Arbib and Caplan (1979). To date, there seems to be no detailed simulation of this kind, though Patrick Hudson's Ph.D. thesis (1977) contains a (non-implemented) analysis of neurolinguistic/psycholinguistic data which is in the spirit of the present paper. Hudson's Chapter XI offers a somewhat ad hoc flow diagram for normal performance without reference to neurological data and then, in Chapter XII, he defines the effects of neural damage as "buffer threshold alterations", relating these to brain regions to see if his model predicts the clinical effects. One can expect that psycholinguistic tests will be increasingly integrated into the study of aphasic and other patients so that our neurolinguistic models can fully exploit psycholinguistic cues to 'mental structure'. But we must expect that our localizations will evolve

with our concepts -- we may localize a function in a region only to later conclude that there we can only localize some aspect essential to that function.

5. The Status of the Cooperative Computation Methodology

We did not offer the C^2 configuration of HEARSAY II as a model of language processing in the brain. Rather, we outlined in Section 4 how the methodology instantiated in Figure 1 might plausibly be adapted to contribute to neurolinguistic modelling. A valid criticism of this paper is that it contains no example of a model within the new methodology. Since we have not stated what the KS's might be, or what the relevant anatomical regions would be, in a neurolinguistic HEARSAY, we do not yet offer testable hypotheses about anatomical localization of linguistic processes. We predict, however, that future modelling will catalyze the interactive definition of region and function which will be necessary in neurolinguistic theory no matter what the fate of our "neurological HEARSAY" may prove to be.

It is my view (cf. Arbib, 1979) that neurolinguistics should not be restricted to the construction of purely linguistic models. I think it is a virtue of the C^2-style that it provides a framework for the future marriage of a parsing system with a cognitive system and an intentional evaluation system. A speech understanding model can thus provide a "growth-node" for a comprehensive model incorporating the whole range of human language abilities such as answering questions, producing stories, describing the perceptual world, and holding conversations, as well as understanding speech. While the implementation described in Section 3 only addresses speech understanding, the C^2-methodology espoused in Section 4 is meant to accommodate such a general model. As a matter of practicality, one must pick a subtask, whether for modelling or for experimentation, and one cannot know a priori whether this subtask is "neurologically valid". One will model it as well as one can, adjusting the subsystems as one seeks to accommodate psychological and neurological data. The real interest comes when we look up from our own subtask to look at a cooperative computation model of another subtask. My expectation is that certain "knowledge sources" (KS's) in the two models will have major similarities; and that it will take relatively little work to adjust these to have a single definition of each KS compatible with each model. As a result, by identifying the instances of each KS in the two models, we may obtain a single model applicable to both subtasks. (For a hint of this cf. Figure 7 of (Arbib and Caplan, 1979) which offers a block diagram of subsystems involved in Luria's analysis of repetitive speech. All the blocks occur in analyses of other language tasks.)

It seems accepted by workers in speech understanding systems that HEARSAY (the actual implementation of Section 3) would perform better were the initial acoustic analysis not so crude, sketchy and error-prone. In particular, this would reduce the extent to which the hypothesize-and-test style of both HWIM and HEARSAY (the general methodology, this time) is required to analyze a given utterance. Does this mean that the methodology is irrelevant for neurolinguistics? We have not, indeed, proven the neurological validity of the hypothesize-and-test style of cooperative computation -- that the brain is modular in a disciplined way, and that it takes hypotheses at one "level" of description and submits them for test against other levels of analysis, with multiple hypotheses active at any time. Perhaps the brain can simply switch in the right process at the right time in a smooth progression from input to output without the "competition and cooperation" of multiple hypotheses. However, such anatomical data as the fact that there are more fibers going 'the wrong way' from visual cortex to the lateral geniculate than those going 'the right way' do seem to run counter to straight-through processing (and compare the discussion in Arbib (1972, pp. 109-112)). Despite the arguments on both sides, the status of the hypothesize-and-test style is an open question for future research.

We noted in Section 2 that there will be much 'pencil-and-paper' analysis of C^2-models. Nonetheless, we do expect computer simulation to be a useful part of computational neurolinguistics, and so I turn to a few observations on the style of such simulations. HEARSAY-II took many man-years to program and does not perform very well. Development of programming methodology will make many aspects of programming such systems relatively routine and efficient -- this has certainly been the case with the writing of compilers and operating systems. However, the poor performance of HEARSAY does raise serious concerns. HEARSAY-II achieves less than 90% recognition of sentences based on a 1,000-word vocabulary. What is to be gained by degrading a model like that to explain the data of aphasiology? My initial, somewhat feeble, answer is that models are not to explain everything. We should not aim to predict the exact sentences produced or comprehended by each individual patient with brain damage. Rather, we should seek to understand the pattern of abilities which survive certain repeated patterns of brain damage -- where both kinds of patterns are abstracted from a variety of individual cases. At this level of approximation, we might test whether or not the pattern of performance of a model degrades in a fashion consistent with experiment whether or not the base of "normal performance" is the same for model and human.

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Computational Models and Psycholinguistic Validity in Speech Understanding

William Marlsen-Wilson

The first half of these comments address three concepts raised by computational models of speech understanding: computation, knowledge types and control structures.

__Computation__: Arbib and Caplan [1979], arguing that 'Neurolinguistics must be Computational', make the point that to progress in neurolinguistics we need a properly specified information-processing model, and that the attendant specification of processing stages and the communication between them entails specification of the precise representations within each. In this sense, it would seem that a psychological process model is inherently 'computational'. The HWIM and HEARSAY models, being implemented on computers, offer examples of systems with this precision of specification. But it is not clear that they are psychologically valid, or that they offer models of human processes to the psycholinguist.

__Knowledge Types__: Given a decomposition of, say, speech understanding into processing stages, the psychologist must ask

where this segmentation comes from. What evidence do we have that the decompositions in HWIM or HEARSAY are psychologically real, rather than the outcome of decisions made for ease of programming a system? It is a deep question to ask if distinctions of knowledge type made in discussing some performance carry over into distinct processes with a system that embodies that performance. Marslen-Wilson and Tyler (Cognition, in press) argue that the notion of distinct processing stages is not strictly essential.

We would like to know what knowledge structures the different systems are cooperating to build. The hope that neurolinguistics will be a source of information on what distinctions to draw is not encouraged by such controversies as that over whether the deficits in Broca's aphasia are syntactic or phonological.

__Control Structures__: We need to specify the way in which interactions between the different parts of the system are controlled. But what is the psychological validity of a Rating Policy Module in HEARSAY; and what is the real neurology of the "neurologized HEARSAY" of Arbib and Caplan's (1979) Section 5? Note that a one-way flow of information from input to output in a purely "bottom-up" psychological model provides an implicit control structure; and that putting in "all the arrows" provides no useful clues as to the actual flow of control. HWIM and HEARSAY have focused the issue by providing explicit examples of control structures, but the need remains to characterize psychologically valid knowledge types and control structures.

The latter half of these comments briefly outline the characteristics of "on-line" human recogniton of a normal utterance in a normal context. I claim that speech is recognized as it is heard, with words recognized within 200 msec. and mapped onto an interpretive structure which affects recognition of later words. This is *contra* Woods' theory, expressed in the design of HWIM, of working out from "islands of reliability" (a Velikooskian view of "islands of collision"!). The problem in current AI systems seems to be that the initial portion of utterances is not reliable enough to provide a basis for processing of this type, but is it that the signal is underdetermined, or rather that the front ends of present computer systems are inadequate? In any case, the process of understanding involves cooperation between knowledge sources. But are HEARSAY and HWIM reasonable models of this cooperation? In any case, what is the role of "top-down knowledge" -- how is high-level knowlege used to guide the lower-level processes in the system? Do humans use this high-level knowledge to predict what is to come? Our own psychological models exclude this. They are optimally effective without explicit predictions.

I believe that most biological systems are optimal solutions to the problem they solve, and that human speech understanding is an optimal solution, resulting from long evolution, to the problem of communication via sound waves. The evolutionary problem is that we have but one vocal tract and that sound waves

can only be modulated slowly, so that speech must comprise a single slowly modulated series of articulatory signals. I take it that the task of the recognition system is to optimize the speed with which a speech signal is analyzed. Overall, the system must maximize the speed with which messages are transmitted and received. The main strategy, then, is to move the analysis as rapidly as possible to a domain where all available sources of knowledge are brought to bear. The essential link is the word recognition system, which provides the basis for invoking the structural and interpretive context. And that's why the system ought to be interactive, even if it is not. The question remains: how are these interactions brought about to yield a true interpretation, rather than a hallucination?

Mitch Marcus: Differential Diagnosis and Least Commitment in Parsing Strategies

When the ARPA speech understanding projects were initiated around 1970, the workers assumed that available phonological and linguistic knowledge was adequate to the task, and that the real problem was that of control: searching a huge space of combinatoric possibilities. Let me start, then, by citing psycholinguistic experiments that suggest that humans can extract far more information "bottom-up" than seems to be available via current computer front ends.

Marslen-Wilson's shadowing experiments show word recognition within 250 msec. Doddington et al. (Acoustical Society of America, 1979) recorded 26,000 words spoken by a random sample of citizens of Dallas. The overall error rate for recognition of these isolated words was 2.3%, with an error rate of 1.2% for the top 1500 words. Phil Liebermann asked phoneticians to transcribe sentences of English-sounding nonsense words. With no higher-level knowledge to bring to bear, they got 91% of the phonemes correct -- but this was after 10 hearings. Sweeney's experiment on the semantic facilitation effect showed that judgement of whether or not a word is in the language seemed to be facilitated by presentation of an associated word, whether or not it occurs in a context which uses the related sense (e.g. 'bug' might relate to 'spy' or 'ant'). The effect is gone after three syllables. [Editor's note: Dr. Marcus said "effect's gone", but it sounded much more like "effects go on". Note that syntactic and semantic considerations could not disambiguate this, and that pragmatics was required to reject the acoustically preferred interpretation.]

These observations lead me to a "sawtooth theory of speech understanding" -- local expansion of possibilities is immediately constrained by following context. Thus rather than growing into a huge tree, the range of possible interpretations expand and contract over time, much like the teeth of a saw.

With this as background, let me make some suggestions for modelling speech understanding, based on David Marr's "Principle of Least Commitment": "Never say anything you are not completely sure of." This calls for richer representations to reduce the search space, using a rich enough vocabulary of knowledge types to specify whatever it is you know without overcommitting yourself.

Note that there is much acoustic information not included in AI systems. Dorthy Siegel, Mark Libermann and Alan Prince have shown, e.g. with the notion of the metrical grid of phonemes, that there is a rich conceptual structure of syllables and their interaction "South of the lexicon". This allows a richer structure of non-commitment below the word level. There is also much information in the intonational structure of an utterance.

My Ph.D. thesis offers a form of least commitment for parsing sentences (from text, though) called "differential diagnosis". Consider the two sentences:

Have the boys who you're auditioning sing the song
Have the boys who you're auditioning sung the song

The first is a command, the second a question, and "have" plays a very different syntactic role in the two sentences. Since the first six words are the same in each, this seems a blow to left-to-right parsing since it seems to inescapably require nondeterminism with the normal sequence of syntactic desicions. But if you clump it differently, aware of the various possibilities, and taking the right notion of context, you can recognize "the boys who you're auditioning" as a noun phrase to get the "least commitment" analysis

Have/NP/verb ...

and a knowledge source with a window just three items wide can then analyze the explicit alternatives to make the decision.

Admittedly, some of these decisions do need semantic interactions. For example, David Marr's work on vision provides an example where bottom-up processing must be supplemented. In the example of Figure 1, it is only "semantic information" about the shape of leaves that supplies the missing boundary to support the interpretation that the top-left and bottom-right regions belong to the same leaf. The point I am making, however, is that rather than delimit all possible interpretations in advance, we can and should generate an option only when the decision must be made.

To summarize: "differential diagnosis" and "least commitment" may help reduce the search space and thus simplify the control problem. However, these restrictions do not imply a retreat to pure competence linguistics. The design of search-limiting representations may well call on "knowledge about knowledge" based on a careful analysis of performance. The result will be a model which is computational, but in a sense which is very different from that of HEARSAY or HWIM models of the ARPA project.

Discussion of Artificial Intelligence and Computational Models

Levine: The argument on the use of knowledge in perception is an old one. For example, in vision we have Helmholtz and Bruner arguing for supplementation, while Gibson argues that "it's all out there". But is this a real argument? Could any experiment distinguish between them?

Woods: Our question is not a choice between 'top-down' and 'bottom-up', but rather one of how far to analyze the acoustic signal before introducing high-level constraints. Do we settle for the most plausible phoneme on local cues, or keep a list of alternatives which high-level knowledge can easily disambiguiate?

Marcus: If we cannot hope to distinguish top-down from bottom-up, then we would have to despair of telling how language is processed by the brain. In pure top-down "analysis-by-synthesis" one tries out the entire range of possibilities to find the best match; in pure bottom-up processing, the speech signal is unequivocally recorded at higher and higher levels to yield a single overall interpretation. No one here advocates either of these in their pure form. The issue of control structures is crucial because even a parallel system has time and resource limitations. The flow of data and control must be studied whether in the computer or in the brain.

Arbib: Our own work on optic flow shows that there is no easy line between 'top-down' and 'bottom-up'. For example, Gibson has noted how much useful information can be picked up from 'optic flow' — the pattern of movement of the retinal projections of environmental features. However, when we try to implement computer algorithms to compute the pattern of optic flow on the basis of a sequence of photographic images, we find that the use of contextual relations is absolutely critical to coerce local estimates into a globally coherent flow field.

Turning to Marcus' analysis of differential diagnosis, I want to suggest that his approch does not mark as radical a departure from the HWIM-HEARSAY approach as he would argue. His thesis work has dealt with text, while -- as examples throughout the conference have demonstrated -- the inference of words from poorly articulated speech provides a challenging new dimension to understanding. I think his point of using richer representations to reduce the search space is well-taken -- just as we can replace nondeterministic search of the state-space of an automaton by deterministic search through the state-space of a suitably structured automaton whose states correspond to sets of states of the original automaton. But -- as in Marr's leaf example -- there is still implicitly a threshold at which alternatives will be considered, and there will remain occasions in which large contexts will be required prior to resolution; and there contexts may well raise further alternatives en route. Thus, better representations will reduce the search space but, with normal human elocution, they will not eliminate it.

Woods: The ARPA workers were aware of the limitations of

phonological knowledge c. 1970. Nonetheless, they stressed that control issues for control strategies had, until then, been unexplored.

Marslen-Wilson: My position is not the same as that of Marcus. I admit that the front-end of computer systems provides degraded input, but I do not want to go so far as saying that all the information is in the signal. I want to invoke contextual and other information, but I claim that this can be done from left to right.

Halwes: Two observations contra bottom-up processing. a) Phoneticians transcribing from a language other than their own had only 40% success on phoneme recogniton. [Editor's note: Sub-lexical knowledge is absent here, as well as "high-level" knowledge.] b) P. J. Price of Haskins Laboratories recorded multi-word extracts from a conversation, and found examples where even the original speaker couldn't recognize them unless they were put back into context. They made responses that didn't seem to connect phonetically with the original.

Marcus: Taking a fragment out of context can destroy intonational cues that can aid recognition.

Marslen-Wilson: Phonetic transcription is not a test of understanding; and it is doubtful whether phonetic labelling is part of the speech understanding process.

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Kean: Woods suggests that function words are unreliable as anchor points. Yet Diane Bradley's work, which has been extended to agrammatic aphasics, shows that there is differential access for function words and content words. Perhaps this segregation compensates for low discriminability on a purely phonetic basis, providing cues to syntactic structure via differential lexical retrieval.

Woods: The classes do seem to be handled differently in speech. Content words seem to get the stressed syllables. The system may thus recognize content words, then devote special processes to analyzing the "stuff in between" as function words.

Kean: It seems to make more sense to work out from the function words.

Woods: Subjective criteria of simplicity are poor guides, especially when based on complexity measures tied to notions of serial processing. The brain is not structured that way. I would maintain that speech understanding involves neural decision between multiple hypotheses.

Marslen-Wilson: Close shadowing yields less than 5% error rate in word recognition, with no suggestion of poorer performance on function words. Admittedly, the speech was well-enunciated and well-recorded.

Arbib: How does the scorer confirm that the words are correctly repeated?

Woods: They may recognize that a function word has occurred and what it sounds like, without knowing which it is.

Arbib: In any case, it seems important to repeat the shadowing experiments with normal speech. The trouble I have getting telephone operators to repeat my name correctly seems powerful anecdotal evidence for the inadequacy of the low-level signal. And the fact that we often catch a speaker's production errors shows that top-down processing is constantly mobilizable, even if not always mobilized.

Zurif: Function words are used as input to the parser by a special retrieval mechanism. People without this mechanism treat these as content words and are agrammatic.

Caramazza: There are different interpretations of Bradley's data. Sweeney, Cutler and Zurif found that detection of function words is slower than for content words in recognition of speech. Of course they play a key role in speech understanding, but do they play an early role?

Schnitzer: In many sentences, the function words may not be particularly functional for analyzing that sentence. In other cases they may be crucial. [Examples from Spanish.]

Lavorel: If we study eye movements during reading, we see that function words may or may not be fixated -- depending on context and on position within the line. In some languages, prepositions appear as morphemic case markers, so the stress on function vs. content words may be misleading.

Three Perspectives for the Analysis of Aphasic Syndromes*

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1. Introduction

Until quite recently there has been a striking paucity of attention to theories of the structure of language and human linguistic capacity in behavioral analyses of aphasic deficits. Analyses have typically relied on naive notions of linguistic structure which would in many cases shock elementary school students brought up on Reed and Kellogg sentence diagrams.[1] The level of discussion took a marked turn upward with the work of Harold Goodglass in which, for the first time, there was a serious attempt made to pay attention to systematic taxonomies of linguistic structure in a broad body of aphasia research. With only sporadic and isolated exceptions (e.g., Whitaker, 1971), the contribution of Goodglass was allowed to remain static rather than being seen as suggesting an aphasia research program where theories of linguistic structure would be more and more incorporated into functional analyses of aphasic syndromes. Thus, in spite of significant advances in the last twenty years in both linguistics and psycholinguistics, for many years

there was little effort to bring those research results directly to bear on the analysis of linguistic deficits. Within the last few years, however, there has at last been a concentrated effort to follow just such a research program as was suggested by Goodglass's work. This is most notable in research which has recently been carried out on agrammatism in Broca's aphasia. At this point we now have relatively detailed analyses of agrammatism in terms of grammatical theory and in terms of processing theories of comprehension and production. The virtue of such analyses should be self-evident. For the first time we have empirically predictive hypotheses which not only aim toward an understanding of deficits at a considerably deeper level than previously known, but which also serve to open up the field to serious debate about the nature of deficits in the context of models of the structure of the linguistic capacity of normal mature human beings. One does not have to be sliding down the slippery slope of confusing lesion sites with function to recognize that such analyses are a necessary component of any attempt to understand the structure of the organism.

Agrammatism can be characterized for English as the selective loss of "function words" and various bound grammatical elements (e.g., tense markers on verbs). In considering the recent work on agrammatism three sets of studies come to mind. First, there is the work of Bradley and her colleagues on comprehension. On the basis of a series of lexical access studies, Bradley and Garrett (in preparation) argue that in the normal course of linguistic events there is a differential access for "function words" than for members of the major lexical categories (nouns,

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adjectives, verbs). These results suggested that it might be profitable to consider whether or not agrammatic aphasics demonstrated the same sort of differential access systems. The research of Bradley et al. (in preparation) addressed just this issue, and it was found that the normal pattern of access was indeed disrupted in the agrammatic subjects. This work served as the basis for developing processing in comprehension analyses of agrammatism (Bradley, 1978; Bradley, Garrett, and Zurif, 1980; Kean, 1980b).

The second case under consideration involves work on sentence production. In an extensive study of normal spontaneous speech errors, Garrett (1975, 1976, 1980) proposed a model of the successive levels of representation which a linguistic string would, minimally, have to pass through in the course of the processes of sentence production. Garrett's model was adopted in Kean (1977) and served as the basis for an attempt to account for aspects of variation in agrammatic production. Furthering the use of Garrett's model, Kolk (1979) attempted to provide a processing analysis of the production deficit of agrammatism; extending and revising Garrett's basic model. Kean and Garrett (1980) have also put forward a production analysis.

The third case to be considered here is the grammatical analysis of agrammatism. Traditionally viewed as a syntactic deficit, in work which attempted to analyze the linguistic structure of agrammatic language use in terms of grammatical theory, it has been argued that the deficit must be located at the representational interface of what are traditionally thought of as syntactic rules and phonological rules (Kean, 1977, 1979, 1980a, 1980b). Under this analysis the apparent grammatical variety of agrammatism--syntactic, morphological, semantic, phonological--is argued to be accountable for in terms of the interaction of various components of the grammatical model.

It will be argued here that each of the models of grammar and processing which figure in these accounts of agrammatism represent necessary components of any psychological theory of human linguistic capacity. Furthermore, it will be argued that the three models in question can, as currently constituted, be viewed as components of the same theory of human linguistic capacity. There is the superficial appearance of inconsistency in the analyses of agrammatism which have been proposed in the context of each of these models: In the case of comprehension the deficit seems to be in lexical access, in the case of production in syntax, and in the case of grammar phonological. Given the consistency of the models across consideration of normal linguistic function, this appearance of inconsistency in the deficit analyses each leads to seems curious, at best. Appearances to the contrary, when taken together the three analyses form a skeleton of a full and consistent analysis of the deficit which has ~~implicat~~ions for further research not only in the local domain of deficit studies, but also in the broader domain of the structure of (normal) human linguistic capacity.

Three issues will be the central foci of this paper:

(I) A minimal condition for the development of functional analyses of linguistic deficits is that there be a consistent use of technical terminology. Unless systematic definitions are given to theoretical notions such as "phonology" and "syntax," and those theoretical definitions rigidly adhered to, what are terminological divergences of no theoretical or analytic content can be too easily confused with those actual theoretical and empirical distinctions which warrant our closest attention. The recent history of aphasia research is frought with use-mention confusions of terminology which have served to shed no light on understanding the nature of linguistic deficits. In this paper there will be an effort to enforce an explicit terminology to the end of determining to what, if any extent the models being considered provide empirically inconsistent analyses of human linguistic capacity, in general, and of agrammatism, as a particular case in point. In section 2.1 the analytic motivation for this terminology will be outlined.

(II) It is an indespensible condition of the analysis of linguistic deficits that they be put forward to the context of models of the structure of normal linguistic capacity. There is no advantage to be taken from an analysis of a deficit which makes no contact with accounts of the structure of normal human linguistic capacity. One cannot analytically characterize any linguistic behavior, normal or deviant, in the absence of appeal to explicit characterizations of the structure of human languages and how knowledge of language is exploited in use. The usefulness of any analysis resides in its explicitness and its predictive capacity, that is, as a guide to future research. Thus, analyses based on grammatical theories which are clearly misguided as models of human linguistic capacity are of no utility--no one would, for example, attempt to put forward an analysis of deficits in terms of the grammatical model

of Reed and Kellogg or on the basis of Panini's grammatical theory, but when it comes to more recently proposed linguistic and psycholinguistic models, in much of aphasia analysis it would seem to be the case that it is taken as totally irrelevant whether or not the model in question has been discredited. The attempt to provide insightful analyses of deficits must be based on the best available models of linguistic capacity. In sections 2.1 and 2.2 synchronically plausible, if yet skeletal, models which attempt to characterize the central components of human linguistic capacity will be outlined.

(III) The combined grammatical-production-comprehension model to be discussed here provides a framework for the analysis of deficits. While the model is skeletal, it has the essential characteristics that one would surely find in more fully elaborated models. In particular, it does allow for serious and systematic analysis of deficit data, it suggests explicit lines of research, and it allows for the construction of falsifiable analytic hypotheses. It is, as far as I know, the only model which has both scope over the central components of a linguistic performance theory and is worked out in sufficient detail to allow for precise predictive analyses. A review of the aphasia literature quickly reveals that much of the experimental research reported has the following two properties: (a) the experimental design is not based on explicit accounts of the structure of language and thus the variables manipulated are arbitrary, and (b) the analyses of the data obtained are either pre-theoretic of <u>post hoc</u> appeals to whatever linguistic analysis is most convenient at the time. Neither of these characteristics permits a systematic research program in neurolinguistics. In outlining an analysis of agrammatism here (section 3), it is hoped that both the necessity and availability of a systematic approach to deficit studies will have been illustrated.

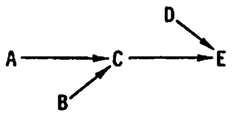
2. On Modeling Linguistic Capacity--some general considerations

2.0. It is taken here as an obvious and necessary assumption of the study of human linguistic capacity that a human being with normal mature command of his native language both knows something (his native language) and can use that knowledge to produce and comprehend sentences. A theory of human linguistic capacity which incorporates accounts not only of the knowledge of language but also the processes of the use of that knowledge in comprehension and production will be called a linguistic performance theory. Thus, each of the models to be considered here is a component of some performance theory. Beyond that rather weak relation among them, the three can be taken together as constituting an attempt to develop a complete linguistic performance theory, skeletal though that theory may currently be, that is, they are not to be viewed as competing models.

A central assumption of all three models is that linguistic capacity under any conceptualization must be viewed as a partially ordered set of autonomous components. Each component is autonomous in the sense that the substantive vocabulary which it operates over is a specific and limited subset of the universally available vocabulary of linguistic elements and that the formal devices available for any one component of the system are a proper subset of those available universally across the whole system. To take a concrete example: In the theory of transformational generative grammar there are two so-called syntactic components in any grammar, a phrase structure component and a transformational component. Both components operate over a categorial vocabulary of N (noun), V (verb), Adj (adjective), etc., and the phrasal projections of those elements (NP, VP, AdjP, etc.). Viewed as formal rule systems the two components are fully distinct; the phrase structure rules are context free rewrite rules whereas the rules of the transformational component are transformational rules which move constituent elements of a linguistic string, subject to a set of universal constraints on the application of such rules. As the phrase structure rules are ordered just 'before' the transformational rules, the representations of syntactic structure which they generate must be such that a proper syntactic derivation will be obtained in consequence of the application of the transformations. Needless-to-say, the formal theory of transformations must, by the same token, be so formulated as to function over the structures generated by the phrase structure rules. There is then an interaction among the components of the system. The output of any component of the system is a representation of a linguistic string with respect to the component of the system which generated it. Such representations serve as the interface of components. To the extent that any component may "directly" interact with any other component, that interaction is <u>modulo</u> their interface representation. Thus, pursuing our example, if there is a proposed phrase structure system consisting of 10 distinct rules, no transformation can demand reference to the structure of a string at any arbitrary intervening level of representation generated by those rules--e.g., no transformation may refer in particular to the output of the rules as they applied up through but not beyond the fifth phrase structure rule. The interaction of components in these models is therefore restricted to those which arise through their ordering relations; two components not linearly ordered with respect to each other or not both feeding a third component cannot in any way interact. Such interactions as there are captured in the systematic representations generated by the components. It is assumed that substantively and formally the components of each model are universally (i.e., language independently) specified, as are their ordering relationships.

The general structural parallelism among the models noted thus far hardly warrants the claim that they are to be viewed as components of the same performance model. However, this general structural parallelism plays a central role in how deficits are to be analyzed in the context of each of the models. Consider the partially ordered set (1).

1.



Assume that A, B, C, D, and E are autonomous components of some system in the same sense as are the components of the linguistic models. If C is impaired there will be a deviant output from the system. It should be evident that while the contributions of A and B to C will be well-formed that well-formedness will be obscured through the deviance of C; at the

same time, while D and E are unimpaired, the imparied contribution of C to E where it interacts with D may well have the attendant consequence of thoroughly obscuring the fact that both D and E are intact. Thus, the deviant output which arises solely from the impairment to C will not be transparently to trivially labelable as "deviant because C is imparied." One could only arrive at the conclusion that C is the functional culprit through an analysis of the data with respect to the particular contributions made by each component to the formal realization of the output function. Under such circumstances, it would then be more than misleading to characterize the output as showing, for example, a deficit in E; surely there would be a 'failure' in E but that failure would be caused entirely by factors extrinsic to E, that is, in no strict sense would we be justified in characterizing the realized deficit directly in terms of or with respect to E.

This rather obvious point has long been noted in the localization literature. While Jackson's (1882) injunction against confusing a lesion site with the locus of function is frequently cited in the literature, if analyses speak to attention paid that injunction, then it is little more than lip service which has been paid it in behavioral work. The confusion which Jackson noted in the context of functional localization work (diagram making) has been a chronic infection of the more restricted domain of functional analyses of deficits. In the case of the analysis of agrammatism the typical error in this regard is the claim that agrammatism is a syntactic deficit because agrammatic sentences are syntactically ill-formed. All realized linguistic strings, well-formed and ill-formed, are products of the full set of components of the linguistic system--the phonology, morphology, syntax, etc.; all strings are therefore *a priori* phonological strings, syntactic strings, morphological strings, etc. Pursuing this point for a moment, compare the strings in (2), where (2a) is well-formed and (2b) is ill-formed.

2. a. Fred and Ethel are living near the Ricardos
   b. Fred ... Ethel ... living near ... Ricardos

A priori one is as justified in claiming that (2b) is phonologically deviant (due to the lack of phonetic realization of certain necessary elements) as they are in claiming (2b) is syntactically deviant or morphologically deviant. For any ill-formed string it is impossible to have any preanalytic insight into the source of its deviance(s). In this regard, it seems appropriate to reiterate Goldstein (1948):

> The question of the relationship between the symptom complex and a definitely localized lesion becomes a problem, no longer, however, in the form: where is a definite function of symptom localized? but: *how does a definite lesion modify the function of the brain so that a definite symptom comes to the fore?*

The italics are Goldstein's.

The relatively constrained notion of interaction of components which is admitted by the models considered here should not be confounded with interactions which arise as a consequence of the interaction of linguistic capacity (as characterized by these models) and other cognitive systems. The models are all sentence-grammar models, their domains what goes on within a sentence. This abstraction away from such intersentential pheonomena as discourse anaphora and reference is in no way a denial of the assertion that there is some type of structure associated with discourse; rather it is a denial of a more particular claim, to wit, that the well-formedness of a sentence of a language can only be determined in the context of discourse. That is, the string *the man bit the dog* will always be a well-formed sentence independent of whether or not in any instance of its being uttered it is uttered in a discourse in a pragmatically appropriate fashion; by the same token, the string *is the man who wearing green shorts is here*? will invariably be ungrammatical independent of whether or not it is uttered in the context of a discussion of whether or not some man who is wearing green shorts is about. Similarly, the knowledge of the real world which is clearly brought to bear on our everyday language plays no role in these models. To take a long discussed example, the sentence *colorless green ideas sleep furiously* is certainly semantically anomolous in terms of what we know or expect of the world--sleeping is an activity we typically restrict to animates, and even for those possessed of vivid imaginations we would be unlikely to ascribe animacy to their ideas; however, the apparent anomoly of that string can be made to disappear in poetic contexts. Surely no account of the structure of what it is to know, e.g., English can be held responsible to such phenomena.

The appropriate everyday use of a language is to be seen then as involving recruitment not only of our linguistic resources but also of a myriad of other resources ranging from etiquette to general conceptual knowledge. It is well to keep in mind the logical dissociation that while it may be true that much of the knowledge of the world which we use was acquired through the vehicle of language use, from that it in no way follows that our language faculty therefore incorporates what we know of the world. To take one last example on this point: I know where my mother lives, and I can use that knowledge coupled with a use of my knowledge of English to tell people where she lives; but, my knowledge of English would in no way be altered if I didn't know where she lived and therefore couldn't use my knowledge of English to tell people that.

Keeping such distinctions in mind is not only curcial to the development of models of the structure of normal cognitive structure (including linguistic capacity). In cosidering the effects of brain damage on the manifest realizations of cognitive capacity it is also crucial. Deviant use of language surely does not logically entail an impairment of linguistic capacity <u>per se</u>. Just as the components of linguistic models interact with each other, so too must linguistic capacity interact with other components of human cognitive capacity. In attempting to develop precise functional analyses of cognitive deficits which arise concomitantly with brain damage it is essential that there not be pretheoretic conflation across domains.

The abstractions involved in the development of these models are not simply restricted to those away from general knowledge and the like, the factors which affect one of the uses we put our knowledge of language to. These models are also based on abstractions away from mechanisms which are intimately connected with the exploitation of linguistic knowledge in language processing. All the models involve an abstraction away from human memory capacity. It is surely true that our ability to process sentences is severely constrained by limitations on working memory. To acknolwedge that there is such a limitation on processing does not entail that linguistic models of processing incorporate models of memory directly. If it is true that there are general constraints on memory load independent of particular cognitive systems--e.g., linguistic, visual imaging--then it would surely be in error to incorporate those constraints directly into the theory of human linguistic capacity. Under such circumstance what would be demanded is a theory of memory such that it could operate over the distinct vocabularies of cognitive domains, and, quite independently, theories of those cognitive domains such that they are capable of appropriately interacting with the memory theory to provide an account for the relevant phenomena. By the same token, it is equally true that were one to show that there are qualitative and quantitative peculiarities to the memory systems invoked on-line in the employment of some particular cognitive system then such peculiarities would have to be modeled into the account of that cognitive system. At this point there is no reason to believe that there is some peculiarly linguistic memory system for on-line processing; thus, in approaching modeling from the most methodologically and theoretically open-minded position memory factors are not to be directly incorporated in the models.[2]

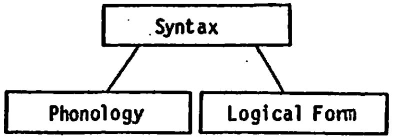
To take a second relevant case, consider the fact that language processing occurs from "left-to-right;" that is, we do not wait until a complete sentence has been presented and then process it wholistically in some fasion. To the extent that this left-to-right'ness is a shared property of multiple cognitive sytems it is not a component of the language faculty <u>per se</u>, and the responsibility of linguistic models to that fact resides only in being able to properly interface with left-to-right implementation. Similarly, if parallel processing is the only processing available for our mental exercises, then it is not directly a component of the language faculty. The exploration of these issues is still in its relative infancy, and we may yet be led to incorporate more and more of such apparently independent systems into our models of the language faculty, but at this point, for the cases noted here the evidence does not seem to warrant such a radical step.[3] It might be noted in this regard that in much of the work in AI where modeling does not involve abstraction away from knowledge of the world, memory, etc., the people carrying out that work deny that they are trying to characterize the language faculty at all. What is desired of models of cognitive systems is that they be as impoverished as an appropriate analysis of the data will allow. In abstracting away from many independent but interacting cognitive systems, the models to be considered here represent attempts to determine the limits the impoverishment, of the human capacity to know and use natural human languages in the normal course of experience. It is, of course, a constraint on such models that they (ultimately) be realizable, i.e., executable, when implemented in a system which takes into account the structure of on-line memory, temporal resolving power, left-to-right'ness, parallel implementation, etc.

<u>2.1</u> It is a regretable and all to frequent fact of the modeling of some specified behavioral domain under different conceptualizations that

there be terminological vagueries and contradictions which obscure any attempt to consider the analyses of more than one model at a time. Each of the models considered here divides the language system up into what are essentially the same sets of component parts, a fact which has in the literature been somewhat obscured by slightly different uses of the same terminology. So, before progressing further to a consideration of each of these models, it is necessary to fix some systematic terminology so that potential empirical (in)consistency not be clouded.

A theory of human linguistic capacity is responsible for accounting for the mapping between the sound realization of a string and its meaning. At the most general level of description the linguistic system can be seen as consisting of three parts: a syntax, a phonology, and a logical form component, plus a lexicon (including a derivational morphology). The component(s) of the model which capture those aspects of sentence structure which contribute both to the sound interpretation (above the level of isolated uninflected words) and the logical interpretation of a sentence are components of the syntax; the phonological component provide an account of those aspects of sentence realization which effect the final phonetic form of a sentence but have no impact on its logical form; the logical form component of the grammar is concerned with the structure of the realization of logical syntax of, e.g., quantification in sentence grammar. Roughly speaking then, the kinds of systems we are concerned with have the structure given in (3).

3.



Given this broad framework of analysis, let us consider with respect to which of these parts the analysis of various aspects of the structure of English sentences is to be assigned. The order of constituent elements in a sentence affects its ultimate phonetic shape so from this it follows that all rules effecting the phonetically realized order of constituents must be incorporated in the syntax or the phonology. Any constituent structure ordering of elements which effects the logical form of a sentence as well as its phonological realization must be accounted for in the syntax, and any rule effecting the structural relations among constituents which has no impact on the logical interpretation of a sentence must be a rule of phonology. Cases of the former sort will be considered first.

Within a sentence a pronoun may sometimes co-refer with some other nominal element within that sentence, while in other instances such co-reference is precluded. This set of co-reference phenomena, known as anaphora, must be accounted for in the logical interpretation of a sentence if that sentence is to be appropriately and fully interpreted. The sentences in (4) contrast in that in (4a) <u>he</u> and <u>Reuben</u> are necessarily disjoint in reference, whereas in (4b) <u>he</u> and <u>Reuben</u> may well refer to the same person (though they need not).

4. a. He thinks Reuben is good looking

b. Reuben thinks he is good looking

These two sentences superficially have the same order of syntactic constituents, yet under interpretation they are quite different. Consideration of such examples demonstrates that (a) to some extent the order of constituents must be fixed in the syntax, and (b) that lexical items (e.g., <u>he</u>, <u>Reuben</u>, etc.) must be inserted into syntactic structures prior to the logical form component. This latter observation follows from the fact that it makes a difference whether the pronoun is inserted in the leftmost nominal position or not.

As the sentences in (5) illustrate, adverbial phrases may occur in one of two positions in many sentences, initially or finally.

5. a. John went to the liquor store before going to the party

b. Before going to the party John went to the liquor store

If we want to capture the fact that such pairs of sentences are systematically related then it is necessary to posit a rule of grammar which will "derive" one from the other, either a rule which moves adverb phrases to the front of sentences or a rule which moves such phrases to the end of sentences. Based solely on the examples in (5) we might be led to conclude that this rule is not a syntactic rule since as far as we can tell on the basis of those sentences no variation in the logical interpretation of the sentences arises as a consequence of whether the adverb phrase occurs initially or finally. If, however, we expand our corpus to sentences with negation, we can readily see that the order of occurrence of an adverb phrase with respect to the main clause does have an impact on the logical interpretation of the string.

6. a. John doesn't beat his wife because he loves her

b. Because he loves her John doesn't beat his wife

In the case of (6a), the sentence is ambiguous. Giving the negative "narrow" scope, the sentence means roughly 'John doesn't beat his wife and the reason he doesn't is because he loves her'; alternatively, if we assign "wide" scope reading to the negative the sentence means something like 'it is not because he loves her that John beats his wife, rather he beats her for some other reason.' Only the former, narrow interpretation is available for (6b). Thus, the rule moving adverb phrases must be a syntactic rule since its application has (for at least some sentences) an impact on the logical interpretation of the sentence.

Also included among the rules of the syntax are the rules of relative clause formation, question formation, and the rules which capture the active-passive relation. Under the grammatical model being considered here, the syntax can be outlined as in (7).

7. a. Phrase Structure Rules

e.g. S→(NP) Aux VP

NP→(Det) (Adj) N (PP) (S)

VP→(Modal) (have) (be) V (NP) (PP)

Aux → [± Tns]

* Lexical Rules——D-Structure

b. Transformations

S-Structure

It is far beyond the scope of this paper to explore this model in any great detail, so discussion will be limited here to a brief description.

The phrase structure rules generate the constituent elements of sentences; in the grammar of particular languages the rules capture the basic order of those constituents. Thus, in English, the rule which rewrites S(entence) assigns an order to the elements NP (Noun Phrase), Aux(ilary), VP (Verb Phrase). It will be noted in the example rules that there is a ( ) notation; an element enclosed in parentheses is optional. In the case of the rule for S, by using this notation the fact is captured that not every sentence must have a subject. As is illustrated by the Aux rule, a category may be rewritten as a specified feature, in this case [± Tns] (Tense); in English [-Tns] sentences include infinitive constructions, while [+Tns] sentences include simple declarative sentences. It should be noted that there is recursion in the phrase structure rules; for example, the expansion of an NP may include an S(as is the case with relative clause constructions, e.g., the man who eats mangoes). Through the application of the phrase structure rules a hierarchical constituent structure is generated (these structures, called phrase-markers traditionally, are often represented as trees or as labelled bracketings). [The rules given under (7a) are meant to be illustrative and should not be taken as being the "official" linguist's rules for English!]

There is necessarily a component of the grammar which relates lexical items to grammatical structures, rules which will appropriately insert lexical items into syntactic structures. These rules, for example, must insure that an intransitive verb such as sleep is not inserted into a phrase marker with a transitive VP (i.e., ... V NP ...) since strings such as George Washington slept Mount Vernon are ill-formed. Similarly, the rules must block the insertion of put into intransitive VP's, and into VP's which only allow for a object NP or only allow for a prepositional phrase since the strings Alice put, Alice put the book, and Alice put in the stacks are invariably ungrammatical; put demands both a direct object and an indirect object prepositional phrase, e.g., Alice put the book in the stacks. The rules which relate lexical items to syntactic structures are called lexical rules; these rules are not part of the syntax per se, but they interact directly with the syntax. The lexical rules apply to insert lexical items into the phrase markers generated by the phrase structure rules. The level of representation of a string which is derived as a consequence of the application of the phrase structure rules and the lexical rules is called the D-Structure.

Considering the relation of the lexicon to the phrase structure system, the organization of linguistic capacity in the brain. We can inquire for example into what extent the different grammatical components are differentially subserved functionally in the brain. Behaviorally this amounts to raising questions such as whether or not it is possible to have a deficit which solely and radically impairs the function captured in this model by lexical rules? It shoudl also be evident that the model makes calims about what aspects of linguistic capacity cannot be differentially subserved. For example, in this framework there is no way it is possible that there could be a deficit which impaired the specific categorial identification of a lexical item but did not impair its restrictions on the syntactic environments into which it may be inserted.

D-Structures serve as the input to the transformational component. Under the theory of grammar adopted here all transformations conform to the schema Move-α, where α is a category, i.e., S, NP, etc. Allowed free reign, such a rule would totally undermine the constituent structure generated by the phrase structure; unconstrained, such a rule would allow any item to move to any place in a sentence. Much of the work in syntax done over the last ten years has been devoted to constraints on the application of transformations of the form Move-α.

These constraints are proposed universals, true of the application of such rules in the grammars of all languages. Consider the sentence the dog is in the yard and it's question counterpart is the dog in the yard. Based solely on the consideration of such pairs we might propose the following rule of yes/no question formation: move the first occurrence of a form of be to the "front" of the sentence. Such a rule would be structure independent, that is not conditioned by the constituent structure of the sentence, but rather conditioned solely by the linear order of items in a sentence. Now consider the sentence the dog who is barking is in the yard. Applying the structure independent rule formulated above we would derive: is the dog who barking is in the yard, a hopelessly ungrammatical sentence. The appropriate question form is, of course, is the dog who is barking in the yard. In order to account for this as well as the original sentence, is the dog in the yard, we need a rule which will select not just the first occurrence of be but rather a rule which will select the first occurrence of be after the full initial NP, and move that element to the left. Clearly such a rule will have to make reference in its application to the structure of the sentence, that is its application must be structure dependent. All rules of grammar are structure dependent; they do not apply to move elements with a blind eye to the structural configuration in which those elements occur. Structure dependence is a crucial feature of the transformational component, and can be formulated as a constraint on the function of transformations. It is in virtue of such universal constraints that the very general transformational schema Move-α is motivated. Note, if such a constraint is true of all rules for all grammars, then a generalization about the structure of grammars would be missed if one were to build the properties of the constraint into each individual rule to insure its appropriate application.

Constraints such as structure dependence are extremely powerful factors in the organization of our linguistic knowledge and in consequence have a significant impact on linguistic performance. While children do make errors in the course of language acquisition, seemingly none of those errors ever involves a violation of structure dependence. Thus, while the child's knowledge of his language may be imperfect, his tacit knowledge of what is possible is essentially perfect--the only possibility that he ever tacitly entertains is that of structure dependence. Normal adults make spontaneous speech errors, but those errors even when they involve a deformation of the order of elements in a sentence never involve a violation of the constraint on structure dependence. Thus, when linguists formulate such constraints they are making claims about the (normal) structure of human beings--when it comes

to language we are inexcerably bound to adhering to structure dependence and other such constraints.

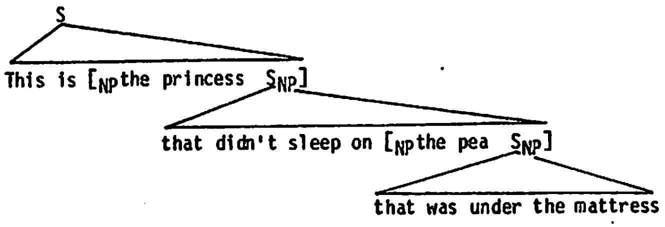
The rules of the transformational component play a central role in the derivation of passive sentences, the formation of relative clauses, and the formation of questions, to cite a few examples. Many potential ill-formed structures are blocked in the course of transformational derication in virtue of the constraints on the application of Move-α. The output of the transformational component is a level of representation designated <u>S-Structure</u>. S-Structures serve as the input to two distinct components of the grammar, the phonology and the logical form.

It should be evident given the richness of the syntax of a anatural language that any analysis of a deficit which comes down to "aphasia of type T is a syntactic deficit" is too vague to be of interest. Given an abstract categorial component for phrase structure rules and a generalized schema for transformations, grammatical representational analyses of deficits in terms of impairments to proper subsets of the rule systems or with respect to the realization of particular constructions do not make any sense. Nor would we ecpect to encounter a deficit which reflected compromise of some proper subset of the constraints. Independent of the particular grammatical model, we would want to rule such analyses out anyway; they are too complex and hardly amenable to sensorimotor translations. Thus, the grammatical model precludes a class of analyses that on the basis of general considerations of gross functional neuroanatomy we would want to be precluded. The model is, furthermore, incompatible with a variety of logically conceivable dissociations, e.g. a dissociation of transofrmational schema and the contraints on the grammatical implementation of the schema. Thus, for the syntax the model both restricts the set of possible deficit analyses while enriching the possibilities for explicit analyses. As such it constitutes an empirical hypothesis as to the gross organization of human linguistic capacity.

Not all rules which effect the order of constituents in a sentence are syntactic rules. Any rule which effects order but which does not have an impact on the logical interpretation of a sentence is a phonological rule. That this is so follows from the preliminary division of linguistic labors into phonology, syntax, and logical form as defined above. Among the phonological rules which effect the order of constituents are the "scrambling" rules of many languages which give rise to relatively free word orders.

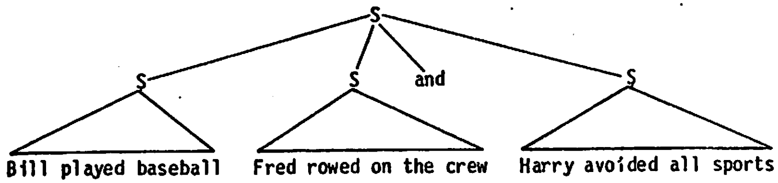
Some rules of the phonological component effect the architectonic configuration of a string without effecting word order. These rules are known as readjustment rules. In the sentence (8) we have a case of a sentence with two embedded relative clauses.

8. This is the princess that didn't sleep on the pea that was under the mattress



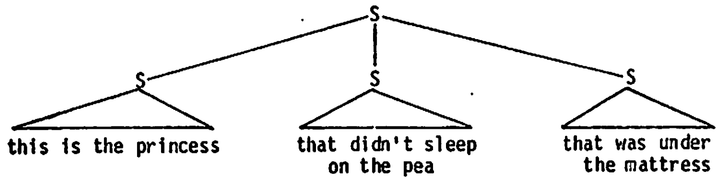
The phrase <u>that didn't sleep on the pea</u> is an embedded modifier of <u>the princess</u>, and <u>that was under the mattress</u> is an embedded modifier of <u>the pea</u>. The architectonic structure of the sentence as is (grossly) illustrated in (8) captures these relations, relations which must be taken into account for assigning the appropriate logical relations to the constituent elements of the string. When such a complex sentence is uttered all the typical phonetic traces of embedding (relatively weaker stress, lowered intonation) are lost; such sentences are realized as if they were coordinate structures structurally analogous to (9).

9. Bill played baseball, Fred rowed on the crew, and Harry avoided all sports



Thus, a rule is needed to readjust the structure of sentences such as that in (8) to structures like that in (10).

10. This is the princess--that didn't sleep on the pea--that was under the mattress



A third class of rules found in the phonology are deletion rules. In many contexts we find the phenomenon of "deletion under identity."

11. a. John is in the kitchen and Harry is in the kitches too

b. John is in the kitchen and Harry is - ϕ - too

Given that the deleted sequence receives an interpretation in the logical form component, it must be the case that that sequence is retained throughout the syntax. Such deletion should be viewed as the marking a string of elements as opaque to phonological interpretation.

Deletion interacts with yet another kind of phonological rule, cliticization--e.g., the phenomena of contraction in English and liason in French.

12. a. John's in the kitchen and Harry's in the kitchen too

b. *John's in the kitchen and Harry's too

c. John's in the kitchen and Harry is too

As is illustrated by the paradigm (11, 12), contraction of be is blocked when the element to be contracted, e.g., "is", immediately precedes a deletion site. Contraction, like the other rules we have considered so far, is then a rule which is sensitive to the structure of a sentence.

It is the structures which are derived through the application of the phonological movement rules, readjustment rules, deletion rules and contraction rules which serve as the inputs to the segmental and suprasegmental phonology, the rules which have traditionally been conceived of as the phonology. Thus, given our original definitions where the output of the syntax must be structures adequate for both logical and phonological interpretation, and, furthermore, syntactic processes are restricted to those rules which have an impact on both the sound and logical interpretation of a sentence, we must postulate a

fairly elaborated phonology. The phonology we are forced to adopt has two major subcomponents, the segmental and prosodic rules, and the structure changing rules discussed just above. Taking as it's input the representations generated by the syntax, S-Structures, the phonological component of the grammar, the box in (3), can be elaborated now as in (13).

13. a. Deletion

b. Stylistic Movement (e.g., scrambling)

c. Readjustment

R-Structure

d. Segmental and Prosodic

Phonology

Phonetic Form

R-Structure is the set of representations generated by the syntax and (13 a-c); it is with respect to these representations that the segmental and prosodic rules of the phonology apply. The segmental rules account for such data as the change in the quality of certain stressed vowels when -ity is suffixed to a word--divine/devinity, profane/profanity, sane/sanity, profound/profundity. The systematic alternations in the quality of sound units are captured in the segmental phonology. The prosodic phonology is concerned with the assignment of stress and intonation to words, phrases, and sentences.

R-Structure is, then, the interface between that part of the grammar which is concerned with constituent structure and that part which is concerned with the phonetic interpretation of individual sound units in a string. The segmental and prosodic phonological rules are sensitive to constituent structure. In English, for example, when two nouns occur adjacently as a compound, as in *kítchen towel*, it is the left one which receives the dominant stress; however, when two nouns occur adjacently in a sentence, not as a compound, as in *Fred gave students bóoks*, it is the rightmost one which receives the dominant stress. Since phonological rules attend to constituent structure, one of the properties of R-Structures must be that they include the constituent structure characterization of a string. If we consider another observation about stress in English, yet another property of R-Structures emerges. In nonemphatic declarative sentences in English, not every orthographic word receives a "word-stress"; thus, in *the boys placed in the sandbox* the words *boys*, *played* and *sandbox* all carry stress, while *the*, *in*, and *the* do not. Nor due these latter three words contribute to the overall sentential stress pattern; both *students read books* and *the boys played in the sandbox* have the same sentential prosody. If we consider the stress patterns of words in English we note that some affixes will cause a change in stress on words, while others never do. In English inflectional morphemes, e.g., the plural, tense markers, the comparative, and the genetive, never effect the stress on a word even when they are realized as full syllables; other affixes, some of the derivational affixes of the lexicon, do effect the stress pattern of a word. Thus, we can contrast the gerundive and progressive suffix -*ing*, a nonstress effecting inflection, with the derivational affix -*ation* as in *divíne*, *divíning*, *divinátion*. What is required is some means for the grammar to

distinguish, in the case of English, the stress sensitive elements from the stress insensitive elements. The sequences which participate in stress assignment in English are the major lexical categories and the phrases dominating these categories. In the structure (14) the units which contribute potentially to the stress and intonational pattern are given in upper case; no other units can participate in neutral nonemphatic stress assignment.

14. the BOYs WALKed to SCHOOL on SUNNY DAYs

There is a simple algorithm for distinguishing these sets of units which will make available a principled notation for distinguishing the stress contributing words from everything else. The algorithm proposed by Chomsky and Halle (1968) linguistics is: insert a word-boundary (*#*) to the right and left of every major lexical category and category dominating a major lexical category. [Details are omitted in (15)!]

15. $[_S\#[_{NP}\#\ \text{the}\ [_N\#[_N\#\ \text{boy}\ \#_N]\text{s}\ \#_N]\#_{NP}][_{VP}\#[_V\#[_V\#\ \text{walk}\ \#_V]\text{ed}\ \#_V][_{PP}\#\ \text{to}$
$[_{NP}\#\ \text{school}\ \#_{NP}]\#_{PP}][_{PP}\#\ \text{on}\ [_{NP}\#[_A\#\ \text{sunny}\ \#_A][_N\#[_N\#\ \text{day}\ \#_N]\ \text{s}\ \#_N]\#_{NP}]\#_{PP}]\#_{VP}]\#_S]$

Applying this algorithm, we can now distinguish those items which are properly bracketed strings of the form [#___#], where ___ contains no *#*'s, from everything else. [For convenience and ease of reading in what follows rather than using the *#* notation, just those items which are properly bracketed string of the form #___#, where ___ contains no *#*'s, will be given in upper case, as in (14); such items will be termed *phonological words* (P-words). Everything else, *phonological clitics* (P-clitics), will be given in lower case.]

If the division of the elements in a string by this algorithm into two systematic phonological classes is an idiosyncratic property of English then the distinction would have to be built into the grammar of English per se. Given that the algorithm is totally structural it can be applied to the representations of strings in the grammar of any language to yield two distinct classes. Having established those two classes within other languages we can then consider whether the grammars of those languages treats the classes in any systematically different ways. If we find that the distinction which shows up in English with respect to stress shows up in other languages with respect to either segmental or prosodic parameters, then that would motivate incorporating the distinction (the algorithm, as it were) into the theory of grammar and not just into the grammar of English. In fact, the P-word/P-clitic distinction is extremely phonologically potent cross-linguistically. It figures in liason phenomena in French, a final consonant devoicing rule in Russian, and a vowel deletion rule in Klamath, to cite a few examples. Since this distinction pervades the segmental and prosodic domains of phonology, it must be representationally captured prior to the application of such rules. It is important to note that there are no segmental or prosodic rules which must apply prior to the introduction of this distinction into the representation of a string, and, by the same token, there is no rule operating in any domain prior to the segmental and prosodic phonology which must take this distinction into account. On the basis of such considerations it must be concluded that the distinction between phonological words and everything else must be given in R-Structure.

From the R-Structure, through the application of the rules of the segmental and prosodic phonology, the phonetic interpretation of a string is derived. The phonetic interpretation of a string, its phonetic form, is a broad phonetic transcription of a string. It is a transcription which does not take into account, for example, such factors as rate of speech; while how fast one talks has an impact on the actual phonetic realization of a sentence such factors are not incorporated into the grammar anymore than is account made in the grammar for the fact that there are certain types of phonetic distortions that can be assocaited with talking when drunk.

As should be evident from this cursory overview, the grammatical analysis of an aphasia as simply "a phonological deficit" would be unrevealing; phonological structure and organization are of such richness that any such description would be without explanatory force. A phonological deficit might involve an impairment with respect to the utilization of the segmental distinctive features or with respect to (some aspect of) the realizations of constituent structure in phonological component. There is, of course, work in aphasia in which both these very distinct aspects of phonology have played distinct and critical roles. In Blumstein's (1973) analysis of segmental (phonemic) paraphasias it is the feature system which is central. In contrast, in Kean (1979) it is constituent structure, in particular what is termed R-Structure here, and explicitly not features which is the pivotal issue in a phonological analysis of agrammatism. Just as distinctive feature theory is an empirical hypothesis as to the parameters of segmental confusion, so too the R-Structure distinction between P-words and P-clitics constitutes an empirical hypothesis as to the parameters of possible categorial dissociability.

As yet little has been said about the logical form component of the grammar. We have noted already some of the considerations at stake in assigning a logical form to a sentence. Among the central functions of the logical form component are the accounts of anaphora and quantification. We will briefly consider an example of each of these functions.

The reciprocal each other must be bound by a plural antecedent in the sentence; in example (16a) "Ford and Carter" bind the reciprocal and the sentence is well-formed. Now consider (16b) which is ungrammatical. In this sentence we note that there is an intervening grammatical subject, "me," between "Ford and Carter" and the reciprocal; rules of binding are universally blocked when a subject intervenes between the antecedent and the variable. Hence the ungrammaticality of (16b).

16\. a. Ford and Carter do not want to vote for each other
    b. *Ford and Carter do not want me to vote for each other

Turning to the case of quantification, consider, for example, the sentences in (17).

17\. a. each man danced with only one woman
    b. only one woman was danced with by each man

In the case of (17a) each man danced with no more than one woman, might have danced with a different woman. In the passive sentence (17b) we understand that of the women there was only one such that she was danced with by each of the men. The output of the logical form component is a representation called a logical form (LF).

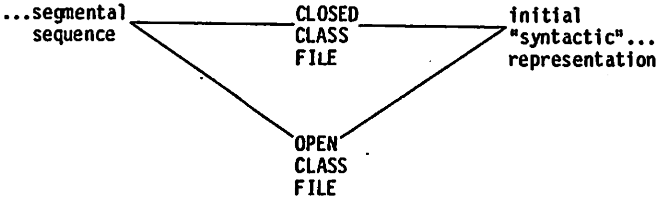
2.2. Throughout the rest of this discussion, the terms syntax, phonology, and logical form will be used in the senses outlined above. That the linguistic data as characterized in each of these models can be analyzed with respect to this terminology is certainly in itself not sufficient to justify the claim that the three models are potentially components of the same performance theory. Justification of such a claim must rest on what one takes the relation between the components of such a theory to be--how the components must be related to each other.

First of all, it should be clear that the same components will not be found in each of the models even when they are fully developed. For example, an essential component of processing language is the mechanism(s) of lexical access, the retrieval of lexical items in the course of production and comprehension. The grammar, being a characterization of what it is to know a language, independent of the mechanisms of use of that knowledge, will contain no access component. Therefore, whatever systematic relations we posit as necessarily obtaining between the grammar and the processing models it cannot be the case that full isomorphism of component systems is demanded.

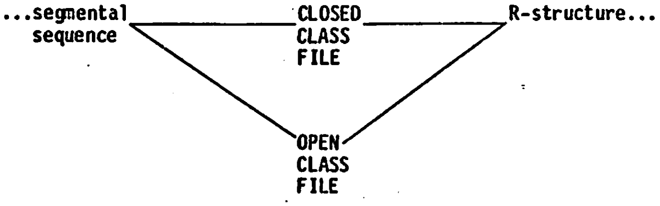
As was noted at the outset, each of the models characterizes linguistic capacity as a partially ordered set of components, each component generating a representation, the characteristics of the function of the components and their output representations being universally specified. It is postulated here that it is a constraint on performance theories that the levels of representation generated by the grammar are all systematically realized by the processors. It should require no discussion that this constraint is quite distinct from a constraint which would demand isomorphism of the components themselves. Not only is it postulated here that the representations generated by the grammar are realized in processing, it is also claimed that these are the only linguistic representations which are systematically realized in processing.

In the grammatical model postulated above, there are two systematic levels of representation within the phonology, the phonetic form of a string and its R-Structure. The R-structure is not only a level of realized representation in processing, ex hypothesi, it also makes available to the processor a systematic distinction of vocabulary into two classes, P-words and P-clitics. While such a distinction of vocabulary types is utilized in the grammar for the purposes of providing a correct account of the segmental and prosodic phonology, the distinction, once available to the processors, need not be utilized solely or transparently for this end (see Kean, 1980b). Consider, for example, the fragment of the comprehension model given in Figure (18a).

18. a.



b.



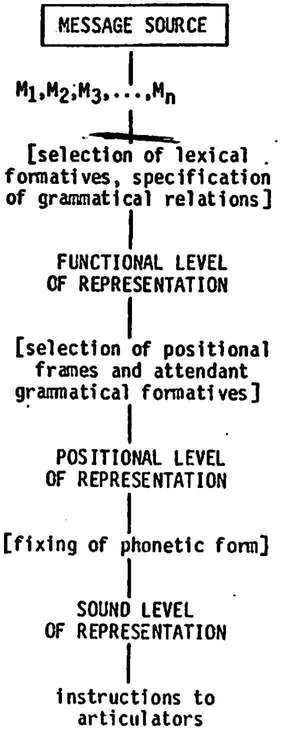
This model is based on the work of Bradley and her colleagues. In a series of lexical access experiments, Bradley and Garrett (in preparation) found that members of the "closed class" (i.e., "function words") were accessed in a fashion distinct from the access pattern found for members of the "open class" (i.e., the major lexical categories). In considering the implications of this finding for modeling comprehension processes, Bradley and Garrett suggest that there is a rapid exhaustive lexical search of the "closed class" file, and that items so accessed provide the crucial cues to initial "syntactic" hypotheses as to the constituent structure of a sentence. On the basis of lexical access, a preliminary constituent structure representation is realized.

The distinction between the "closed class" and the "open class" noted in this work is, for the range jof currently available data, the same as the P-word/P-clitic distinction of R-Structure. It has long been hypothesized by linguists that R-Structure is the preliminary constituent structure representation of a sentence which is realized in processing.[4] The Bradley and Garrett analysis is consistent with this position, and Bradley, Garrett, and Zurif (1980) and Kean (1980b) both suggest that it is in fact R-Structure (also called "phonological representation") which is the initial "syntactic" hypothesis which is the realized output of the lexical access system. Following that interpretation of the results of the lexical access studies, (18a) can be translated as (18b). It should be evident from this example, that many specious arguments can, potentially, arise due solely to non-empirical terminological confusions. In terms of (18a), any analysis made with respect to the representation realized in consequence of lexical access will be called "syntactic" whereas in terms of (3), Figure 1, and (18b)an analysis with respect to R-Structure would be termed "phonological." A failure to use systematic terminology across the models within theories of grammar and processing can only serve to obscure the substantive issues which are at stake in analyses put forward.

If we turn now to the production model in question, we find it is also necessary to carry out some "translation" in order to see whether or not it can be incorporated into the performance theory which includes the grammar and comprehension models already discussed. The model in (19) is taken from Garrett (1976); this model was developed on the basis of an extensive study of the structure of normal spontaneous speech errors. The logic of the approach to speech error analysis in the development of production models is quite straightforward: There must be processing mechanisms for sentence production, speech errors are not random deformations of well-formed sentences, therefore, speech errors must be constrained by the mechanisms of sentence production. Under this line of reasoning, a systematic taxonomy of speech errors can be taken as indicating (some of) the component systems of the production processor

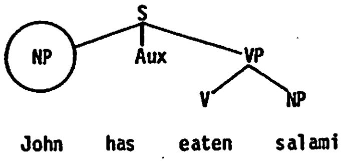
(see Garrett, this volume). The model in (19) characterizes the minimal number of levels of representation which must be incorporated into any model of production which attempts to account not only for well-formed productions but also the range of potential deviances from well-formedness. Under such a model, the notion "impossible speech error" has essentially the same status as the notion "ungrammatical sentence" has for the theory of grammar.

19.



At first glance it would seem that this model has little in common with the grammatical and comprehension models discussed so far. There is no obvious equivalent to the functional level in the grammatical model, or, to be more precise, it is not clear what the appropriate grammatical equivalent might be. We can think of grammatical relations in two ways: First, in terms of the structure of a sentence, we can characterize grammatical relations on purely structural grounds. Thus, for example, the subject of a sentence can be characterized as that NP immediately dominated by S.

20.



If we appeal to underlying grammatical relations, then *John* is the subject of both the active sentence, (20), and of its passive counterpart, *salami was eaten by John*. At the level of S-Structure, however, under the structural definition of subject, *John* is the subject of the active sentence while *salami* is the subject of the passive. Yet another type of grammatical relation one might think of is the thematic relations of the different elements in a sentence, relations such as *agent*, *patient*, etc. Thematic relations are determined in the logical form component of the grammar. At this time the functional level is not sufficiently specified to allow for a specific hypothesis as to its possible appropriate grammatical analogue.

The positional level is more readily localizable in terms of the models we have been considering. The characteristic of the positional level is that there is a systematic distinction between "function words" and inflections, on the one hand, and everything else on the other. This is then, once again, an R-Structure representation, R-Structures being the one level of grammatical representation where that distinction between these classes, P-words and P-clitics, is made. Further evidence that it is R-Structure which is at issue here comes from the fact that the order of elements as represented in the positional level is the same as the order of elements as they are phonetically realized. Again, R-Structure has just this property. Garrett (1975) described the positional level as a surface "syntactic" level. Thus, here we find another case where there is a potential confusion arising solely out of terminological variation. In terms of the models under consideration, each proposes a level of representation with a P-word/P-clitic bifurcation of items in which the elements of a sentence have their realized order. There is no substantive distinction between the models with respect to postulating such a level. For terminological uniformity, this level is being called R-Structure here, and that level is, by definition, a phonological representation.

The claim that the models in question can be taken as components of the same linguistic performance theory may still seem like so much wishful thinking. There surely are differences among the models, the most notable being the considerable disparity in the degree of specificity of each. In the case of comprehension, we only have a small fragment of a model, and, while the production model is more fully outlined, it still lacks the detail of the grammatical model. The production model covers a broader range of stages in processing than does the comprehension fragment, but in its account of the details of those stages it lacks the specificity of the comprehension fragment. Neither of the processing models as currently developed posits as many levels of representation as does the grammatical model, however, from the failure of, e.g., the production model to postulate and LF/D-Structure distinction nothing follows at this time. It is not the case that the proponents of these models deny the LF/D-Structure distinction; it is rather the case that at this time they have no data which bears on that distinction with respect to the models of processing. There can be no substantive dispute over a domain which consists of an open question. To the extent that these models are developed they are mutually consistent. Whether they will remain consistent when they are all more fully elaborated is something only a clairvoyant might know. Given the current form of these models it can be claimed that they can be taken as components of the same linguistic performance theory because they are consistent with each other.

3. An Analysis of a Systematic Deficit

3.0. Agrammatism in Broca's aphasia is typically behaviorally characterized (for English) in terms of a selective inability to exploit "function words" and inflectional elements in spontaneous speech. In a wide variety of studies, this selective failure of the normal capacity to exploit "function words" and inflections has been shown to be a deficit which pervades virtually every modality of language use, including metalinguistic abilities. Simply on the basis of this general description, it is an obvious preliminary hypothesis to correlate this deficit, agrammatism, with phonological R-Structures.

3.1. The grammatical analysis of agrammatism with respect to R-Structures is quite straightforward; as it has been discussed extensively (Kean 1977, 1979, 1980a, 1980b), it will only be reviewed in outline here. A systematic grammatical analysis of agrammatism must, minimally, provide a bifurcation of the elements in a string into two classes, "function words" and inflections _vs_. other items; this is, of course, the P-word/P-clitic distinction in so far as can be determined on the basis of available data. No other level of grammatical analysis would seem to provide anything systematically approximating the distinction in question save for the R-Structures of the phonological component. The claim of the so-called phonological analysis of agrammatism is then simply this:~~—at all~~ levels of representation and with respect to all components of the grammar, save for R-Structures of the phonological component, any grammatical analysis of agrammatism will be purely stipulative.

There are several significant empirical consequences to this analysis. First consider its predictions with respect to English. Because the analysis is based on a general structural property of the language it is in fact greatly underdetermined by the data which motivates it. This is, of course, going to be a property of any analysis which attempts to reach a level of descriptive adequacy. In virtue of being so underdetermined it makes a variety of predictions about classes of items previously not considered in analyses of agrammatism, thereby raising new and specific research questions. For example, as the suffix *-ness* is a P-clitic in English the prediction is that it should pattern with the "function words," while the suffix *-ity* which is not a P-clitic should not, *ceteris paribus*. Several objections have been raised to this sort of claim. Curiously, it has been suggested that since there is no data from agrammatism which directly supports that claim, that can be taken as constituting an argument against the phonological analysis (Kolk, 1978). Surely the relative truth or falsity of a prediction cannot be evaluated if the relevant data are not available. Yet another objection has been that Broca's aphasics do not typically use long multimorphemic words, e.g., words in *-ness* or *-ity*, and therefore the analysis must be wrong because it predicts that they will use such words, in particular, words with *-ity*. As the analysis makes no claims about actual production capacities, and no purely grammatical analysis could, such putative arguments are totally beside the point. To accept any such line of argument would lead one also to accept arguments that there is a grammatical difference between *dogs don't eat plankton* and *orangutans don't eat plankton* due to the fact that there are processing distinctions between the two becase *orangutan* has more syllables and is of lower frequency than *dog*. Finally, it has even been suggested that the analysis must be wrong because even if Broca's aphasics did use words of the relevant complexity to easily test the hypothesis that it is highly doubtful that *-ness* would pattern exactly with, e.g., the plural, and

-ity would be fully retained. It would be in no way surprising if this conjecture were true; it would also be totally irrelevant to the grammatical hypothesis. It is not the case that the deficit of agrammatism compromises all "function words" and inflections in equal degree; there is a hierarchy of to the compromise, *-ing*, an inflection, being relatively well retained, and *-s*, the verbal inflection, being just about completely "lost." Thus, given that there is such a hierarchy one would anticipate that items like *-ness* and *-ity* would fall somewhere on it. Where they would fall is another question. It has been suggested (Kean, 1977) that this hierarchy is found in normal production and that it reflects the diversity of the class of P-clitics; Kean and Garrett (1980) also note the necessity for mechanisms of construal in the lexicon which would give rise to a production hierarchy. Aphasia studies should figure prominently in research addressed to developing a refined and principles analysis of this hierarchy. Whatever the appropriate analysis of that hierarchy, that is totally independent of the claim that R-Structures provide the proper grammatical domain for analyzing agrammatism.

A second empirical consequences of the analysis is that it makes explicit cross-linguistic predictions. The P-word/P-clitic distinction is a structural distinction to be found in all languages. Thus, *modulo* the role this distinction plays in processing, the analysis gives a principled description of agrammatism for all languages. There are two virtues in this. In describing agrammatism cross-linguistically there is a chronic confusion of translation. In a language like English, the oblique cases (i.e., dative, locative, ablative, etc.), save the genitive, are basically captured in the language through special uses of prepositions; in other languages these cases are captured for the language by special case affixes on nouns (e.g., Latin). If in characterizing agrammatism one were restricted to making appeal to descritpions which arise under translation then it would surely be the case that the deficit would be totally unsystematic cross-linguistically. The structural relation of a case marker to a noun is not necessarily the same as that of a preposition to a noun; case inflectons need not be P-clitics. Since one knows from consideration of one language, English, that the deficit is systematic then it must be the case that some systematic nontranslation analysis should be available. Whether or not the phonological R-Structure analysis is correct, it does have the clearly necessary property of any potentially adequate analysis in being language independent. Secondly, as with the consideration of English alone, the analysis goes far beyond the available corpus of data on agrammatism, and in doing so suggests new areas of inquiry and at the same time opens itself to rather direct falsification.

While grammarians have long noted that any analysis of the phonology of a language involves more than just description of how a linear sequence of unit segments (systematic or taxonomic phonemes) interact with one another, this has typically gone ignored by people working in domains outside the study of the structure of human languages. In consequence, this has led some people on occasion to reject the phonological analysis of agrammatism since it involves something other

than linear strings of segments. Such a rejection amounts to little more than a pun. Clearly an analysis cannot be rejected on the basis of a terminological disparity. If one wanted to maintain that phonology is solely concerned with the way segments interact in linear sequences in various languages then the R-Structure phonological analysis could not be properly termed "phonological" (and such phenomena as stress, intonation, and contraction in English would also be nonphonological). As the domain of phonology in grammar has never been so arbitrarily restricted in the past (for good reason it would seem), it would hardly seem warranted to change the definitions of phonology and syntax simply in the service and furtherance of ignorance.

Simply because R-Structure is a phonological level of representation, and because it stands at the interface of rules which attend to the order of constituents and rules which attend to prosodic and segmental realization it does not follow that the phonological analysis of agrammatism makes the prediction that there will be a segmental or prosodic deficit in agrammatism. In fact, the phonological analysis does not make such a claim. First, that claim would only make sense if one took the grammatical model also to be the processing model completely. One might plausibly anticipate that because there is a deviance with respect to the input to the processes of segmental and prosodic realization in production that there would, as a byproduct, be some segmental and prosodic disruption in production and identification, but this is all. By the same token, since R-Structure does not contribute to the segmental analysis of a string in comprehension but is, rather, a product of segmental analysis, lexical access, etc., one might anticipate no significant comprehension deficit at the segmental level. But these are only speculations, of rather limited force in the absence of serious analyses of how R-Structures actually interact with the segmental processes of production and comprehension.

3.2. As was discussed above, Bradley and Garrett (in preparation) argued on the basis of experimental data that in sentence comprehension there were distinct lexical access systems for "function words" ("closed class" items) and other words ("open class" items), a distinction of elements which parallels the P-word/P-clitic distinction over the range of experimental data. They hypothesized that this distinction in access systems played a crucial role in the construction of initial "syntactic" hypotheses in parsing in comprehension. Members of the "closed class" provided, it was suggested, crucial cues to the constituent structure of a sentence; items in the "open class" they noted are, with great frequency, ambiguous as to syntactic category, an ambiguity which can in large measure be resolved by attending to the particular "closed class" item(s) in their local domain. In virtue of this two track lexical access system an initial constituent structure representation is realized.

Bradley, Garrett, and Zurif (1980) noted that it had been hypothesized by linguists that R-structures (called "phonological representations," as in earlier linguistic work) were the initial level of constituent structure realized in comprehension. Given the characteristics of R-Structures, that they distinguish "open" from "closed" class items, and provide a gross constituent structure analysis of a string, it would then be natural to take it that R-Structures are the initial constituent structures realized comprehension. Following the traditional nongrammatical description of these structures, they termed them "syntactic" and not "phonological." Again we have here simply a difference in terminology. To the extent there is relevant data available, those data rather directly implicate R-Structures.

Given the evidence that there is a comprehension deficit which parallels the production deficit of agrammatism, in the light of the Bradley and Garrett (in preparation) results it was obvious to raise the question of whether or not one would find evidence of the same sort of two track access system with agrammatic subjects. Bradley et al. (in preparation) carried out a study with Broca's aphasics which directly addressed this question. The results of that study were that the aphasic subjects did not show the capacity to do a rapid search of a segregated file of "closed class" items, rather, they appeared to treat those items as if they were members of the "open class." Under the hypothesis that an independent access system for members of the "closed class" plays a crucial role in the realization of initial hypotheses as to the constituent structure of a string, the agrammatic access data can only be interpretted as indicating that due to their loss of the ability to exploit the "closed class" file agrammatic aphasics are not able to make the appropriate initial constituent structure parsings of sentences, that is, they have an impairment to a crucial component of the capacity to realize R-Structures.

The lexical access studies discussed here all involved visual presentation of stimulus items. Obviously such studies are to be supplemented by auditory studies of the general comprehension argument, analysis is to be maintained. In a recent auditory study using a word monitor technique, Swinney, Zurif, and Cutler, (forthcoming) obtained results which are consistent with those of the visual studies. They too describe the deficit in terms of an inability to realize an initial "syntactic" representation; this simply reflects the tradition of psycholinguistics to refer to all matters of constituent structure as "syntactic." There is in these data and their analysis no basis for claiming a substantive empirical difference in this analysis of agrammatism from that which is termed "phonological" with respect to R-structure.

While the comprehension model of (18) makes claims about stages in processing, it makes little claim as to the nature of the mechanisms available for these stages. To be sure the model demands that the mechanisms attend to a distinction between "open" and "closed" class items, but it makes no claim as to how these two classes of items are integrated in the realization of R-Structure. In a recent comprehension experiment, Schwartz, Saffran, and Marin (forthcoming) provide some experimental evidence which suggests that agrammatic aphasics have difficulty with the proper realization of word order in comprehension. Those data are both suggestive of the scope of the deficit and deserving of considerable attention in any attempt to develop hypotheses as to the nature of the mechanisms in question. Surely, whatever the mechanisms involved, they must be capable of the maintainence of word order. How that is effected is the issue. If, for instance, the positional constituent structure frames made available through the "closed class" access and parsing system are in some sense crucial keys to maintaining appropriate word order relations in comprehension, then the limitation in the capacity to utilize the "closed class" access system would provide at least a partial explanation for any failures in maintaining word order relations in comprehension in agrammatism. This is, to be sure, speculation, but it is suggestive of the type of avenue to inquiry which one must pursue if attempts to provide principled analyses of deficits are to be pushed with any degree of seriousness. At this point in time, the relevant data for making justifiable proposals along these lines are wanting.

<u>3.3.</u> While production has traditionally received the greatest attention in describing agrammatism, particularly clinically, it has received the least attention in the development of systematic and principled analyses. As the most salient characteristic of agrammatism is the selective failure to exploit "function words" and inflections in spontaneous speech, in terms of the production model it is the positional level which is implicated. The positional level involves an R-Structure representation of sentence frames consisting of the superficial constituent structure bracketing of a sentence and its grammatical formatives, i.e., "function words" and inflections, or "closed class" items, or P-clitics. Into these frames the lexical formatives, i.e., "major class" items, or "open class" items, or P-words, are inserted under Garrett's (1975, 1976, 1980) model.

What is unclear at this point is how exactly the positional level is implicated. As with the previous two analyses, the production analysis suggests specific avenues for research. Extrapolating from the comprehension analysis, it would be reasonable to attempt to further refine the production analysis in terms of an inability to exploit the "closed class" lexical file to the end of establishing appropriate positional frames in production. As Garrett (this volume) points out this is one area where there is good evidence of an overlap of the production and comprehension systems. If the deficit is ascribed to a failure in establishing positional frames, and if, following Garrett's model, these frames are the vehicle for establishing the appropriate surface word order in normal production, then it would be predicted that agrammatic aphasics would have as a byproduct of the deficit a problem with word order in sentence production. Recent research by Saffran, Schwartz, and Marin (forthcoming) provides evidence which suggests that there is in fact a word order problem in production. It is essential to keep in mind that under the analysis being suggested here there is no deficit with respect to establishing the word order of major lexical formatives per se, but rather that there is a deficit which has the consequence of impeding the normal appropriate execution of the insertion of major lexical formatives into appropriate positional frames. At this point there is insufficient production data to allow for the further refinement of this hypothesis. What is required are both studies in which the P-clitic/P-word distinction for production can be systematically contrasted for normal and agrammatic subjects and studies of both populations which would allow for closer scrutiny of the mechanisms for relating major lexical items to positional frames in production and comprehension.

It has been suggested by Schwartz, Saffran, and Marin (forthcoming) that their word order studies provide evidence against the grammatical phonological analysis. Evidence in support of that suggestion can, by the logic of the situation, only come from showing either (a) that the word order production data are inconsistent with the predictions of the phonological analysis under the appropriate production model, or (b) that a better explanation is available under some other model. As has just been outlined, there is reason under the production analysis coordinate of the grammatical phonological analysis to anticipate some problem with word order, though, to be sure the precise character of that problem is currently left open. Saffran et al. suggest an analysis in terms of Case Grammar (Fillmore, 1968). It is implausible to think that that analysis will in the end prove to provide a better explanation of the data since Case Grammar was long ago abandoned by linguists due to the fact that it was fundamentally inadequate as an approach to accounting for the basic structure of human languages. At this point the production model is not sufficiently specified and the relevant data are wanting to be able to closely consider whether the production analysis with respect to the phonological R-Structures of the positional level will provide an adequate analysis of agrammatism or not. What is currently provided by the production analysis is the outline of a framework for systematic research in the future.

Another recent study of the agrammatic production was carried out by Kolk (1979). Kolk used a story completion paradigm to elicit contrasting pairs of sentences like *the lion is able to kill* vs. *the lion is easy to kill*. While the surface linear word order of such pairs is identical, they differ significantly in that *the lion* is the subject of *kill* in the former sentence while it is the object of *kill* in the latter one, a distinction which is grammatically captured by the fact that there is a significant structural syntactic difference between the sentences. Kolk trained subjects on sentences of the *able* type in story completion, and in his training would explicitly tell his subjects what the appropriate target was and encourage them to produce it. Having been trained on the *able* type sentences, the subjects were then given stories which demanded an *easy* type sentence for completion. Kolk found no transference of training to the *easy* sentences. He argued that this provided strong evidence against the phonological analysis and in favor of a syntactic analysis. However, given that the distinction between *able* and *easy* type sentences is syntactic and not phonological, the failure of transference provides evidence that the subjects were tacitly aware of at least that aspect of English syntax. Thus, if the experiment points to anything it points to an intact syntactic capacity.

Kolk notes that the majority of the errors which his subjects produced were "syntactic" errors, where "syntactic" errors involved all those errors which were (a) not segmental paraphasias, and (b) not omissions of "function words" or inflections from *the appropriate target sentence*. Thus, if a subject produced a sentence which was not of the target structure, but rather some alternative structure, and that production was ill-formed in virtue of the omission of a "function word" such an error was called "syntactic," whereas an error involving the omission of a "function word" from a target sentence was not called a syntactic error. Using this rather curious differentiation, Kolk claims that the phonological analysis is not supported since it fails to predict that the majority of errors will be "syntactic." Such reasoning hardly warrants discussion. To be sure, Kolk's error data deserve close scrutiny. For example, his subjects showed definite preferences for producing some of the possible alternatives to the target constructions. Little is known of nature of structural preferences in processing, and as Kolk's corpus is a controlled sample over a restricted set of structures it is a valuable resource for beginning to develop some understanding of preference phenomena.

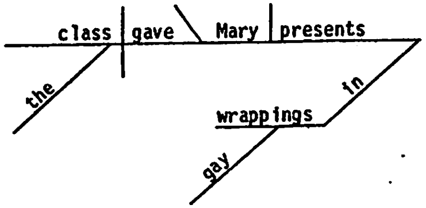
Kolk has attempted to interpret the production deficit of agrammatism in terms of Garrett's model. Initially, Kolk (1978) argued that the deficit should be associated with the positional level; as Garrett (197) had characterized the positional level as "syntactic," Kolk argued that if the analysis in terms of the positional level were correct then the phonological grammatical analysis was wrong. This is yet another instance of the confusion of terminological differences with empirical differences (Kean and Garrett, forthcoming). More recently, Kolk (1979) has offered a second analysis in terms of Garrett's model with one modification: he postulates two positional levels of representation, one "syntactic" and one "phonological." Agrammatism, he claims, involves a production deficit in the realization of the "syntactic" positional level. No data are presented to motivate these two distinct levels. Furthermore, it is unclear in what way they are distinct. As the characteristic of the positional level is, according to Garrett, a distinction between sentence frames with grammatical formatives (P-clitics) and the set of major lexical items, presumably this is a characteristic of both of Kolk's positional levels. Now, if we accept both Kolk's elaboration of the model and his breakdown of error types into those involving segments, those involving P-clitic omissions from target sentences, and "syntactic" errors, given that the majority of errors are not of the P-clitic class, under Kolk's elaboration of the model it could be neither the "syntactic" nor "phonological" positional level which is implicated. Thus, if we accept Kolk's model, his analysis in terms of that model does not hold up, and, independently, there is no evidence in support of his model as an alternative to Garrett's.

3.4 Looking at agrammatism from the perspectives of grammar, production, and comprehension where the models of the conceptualizations of language are mutually consistent, we begin, I think, to have a framework for the close scrutiny of functional deficits. Without such models analysis of deficits will remain capricious; an anarchic approach to functional characterization has had no theoretical utility. While this work is still in its early infancy, it does allow for the liberation of research from pretheoretic conceptions of the structure of language and human linguistic capacity. Agrammatism and at least some of its concomitants are no longer a random collection of symptoms; one begins to see some functional coherence to the symptom-complex. Experimental studies have enriched our conception of agrammatism; no longer can one think of the agrammatic Broca's aphasic as a person with just a production deficit. The systematic analyses of agrammatism which are beginning to emerge direct our attention to new avenues of inquiry where we can, I think quite safely, anticipate future experimental research to further enrich our appreciation of the scope of the agrammatic deficit. As such research proceeds it is also quite clear that our understanding of the structure of normal function will in turn be enhanced.

Footnotes

1. Reed and Kellog sentence diagrams were the backbone of elementary school grammar lessons until relatively recently. Below an example is given of such a diagram.

The class gave Mary presents in gay wrappings



2. Frazier and Fodor (1978) have proposed a parser which explicitly interfaces with memory constraints; where short term memory limitations come to bear on syntactic parsing and the vocabulary of linguistic elements over which such constraints operate is specified in their proposal. See also Frazier (this volume) for further discussion.

3. For the sake of argument, let us assume that there exists some constraint C which operates across all domains of human cognitive capacity. In such circumstances we would not be motivated in directly incorporating C into our models of those systems. This would remain

the case even were we to discover (a) that for the cognitive systems in question each was discretely neuroanatomically localized and (b) that the neuroanatomical substrate of C were equipotentially realized across each relevant area. In this it is not the case that the functional models are not responsible to a physical interpretation; rather, one could not justifiably propose an equipotential physical sybstrate for C were it incorporated independently into the models of each cognitive system.

4. Chomsky and Halle (1968), for example, state:

> It appears that the syntactic component of the grammar generates a surface structure Σ which is converted, by readjustment rules that mark phonological phrases and delete structure, to a still more superficial structure Σ'. ...We might speculate, then, that a first stage in perceptual processing involves recovery of Σ' from the signal using only the restricted short term memory, and that a second stage provides the analysis into Σ and the deep structure which underlies it. (p. 10)

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Edgar Zurif: Comments on Levine and Kean Papers

I am interested in providing a functional decomposition of language that is experimentally defensible and in which distinctions among constituent processors correspond to neurologically natural separations of function. Kean seems to share this interest; I want only to enter some cautionary notes concerning her paper (with which Kean is likely to be in agreement), and to use her analysis as a means of raising some issues for future research. However, Levine's data on the neuropathological correlates of language dysfunction need to be recast if they are to bear upon distinctions among linguistic information types, and at the risk of applying undue force to his data, I will attempt to fashion a fit -- but in the broadest of terms only.

Among the opinions expressed by Levine in his talk and in a printed effort (Levine and Mohr, 1979), are (1) that Broca's aphasia does not result from a lesion to the third frontal convolution (Broca's area) but rather follows from more extensive fronto-parietal lesions of the dominant hemisphere, critically involving damage to the motor cortex itself; and (2), that such recovery that does take place following a lesion in Broca's area is mediated by preserved regions of the dominant hemisphere (superior and posterior to Broca's area) and not by the non-dominant hemisphere. That is, the co-existence of an extensive right-sided lesion is claimed not to adversely affect recovery consequent to a Broca's area lesion. These are interesting and contentious claims. It is not always clear from Levine's descriptions that the left-sdied lesions involving motor cortex produce Broca's aphasia: rather, in some

instances, they seem to produce a global aphasia -- an aphasia in which the output is not so much agrammatic as nonexistant and in which there is a severe comprehension disturbance. To note the obvious: more detailed observations are required.

My greater concern, however, is with the form in which Levine has charted the language impairments -- both for those patients that seem to be Broca's aphasics and for those patients that have small left-hemispheric lesions coexisting with extensive right-sided lesions and who do not seem to be Broca's aphasics. As it stands, although Levine disputes the anatomical details of the classical model of Broca's aphasia, he nonetheless frames the language deficits squarely within this model. Namely, the deficits are still primarily described in terms of disruptions to one or another of the language faculties of speaking, listening, repeating, and so on. Thus, in its present version, Levine's work pays scant attention to the details of the structures inherent in grammatical descriptions, and correspondingly, to the disabled linguistic structures in the aphasias.

A partial -- and hopefully not too fanciful -- reconstruction of Levine's clinical descriptions is possible, however. He has not treated aphasic speech and comprehension as totally unanalyzable wholes. And in this respect, it appears that whereas the left-brain damaged patients he has characterized as Broca's aphasics (Levine and Mohr, 1979) do not all show impaired vocabulary skills, they uniformly present with an inability to process features of sentence form. Extensive damage to the right hemisphere (with or without a small lesion to the third frontal convolution) likewise appears to variably impair vocabulary skills, but, in contrast to the previous clinical picture, seems always to spare a "syntactic" ability.

Admittedly, given Mohr's (1976, see also Levine and Mohr, 1979) neuropathological analyses, the exact location of the focal point of the lesion producing Broca's aphasia is less certain than it used to be. Even so, there is no question that the responsible lesion is anteriorly placed in the left hemisphere. Accordingly, the ability to analyze sentence structure can still be viewed as depending upon the integrity of a delimited left-anterior region, whereas no such well-defined location seems to subserve the ability to name objects and to understand the meanings of individual words (see also Whitaker's paper, this conference).

This outcome is not too surprising. Granting that the conceptual structure in which word meaning is embedded is the repository of at least all practical knowledge, it seems reasonable to expect that the matrix of properties and relations comprising such structure will not receive the delimited neuroanatomical specification accorded language-specific (syntactic) mechanisms. Quite apart, then, from unique input and output factors, the organization of the brain seems, broadly, to honor the distinction between lexical semantic factors, on the one hand, and features of sentence form, on the other.

Kean, too, draws distinctions of this general sort. But to say the least, hers are more responsive to the structural details of linguistic analysis, and they are less abvious. Moreover, her linguistic distinctions accomodate a processing device which, though only sketchily worked out, appears to be geared specifically to the assignment of structural analyses.

Thus, in alignment with her grammatical -- specifically, phonological -- distinction between open and closed class vocabulary items, there appear to exist separate routes for the lexical access of these two classes. By hypothesis, the closed class route may be considered to serve as input to a parser, permitting the on-line construction of a structural representation -- or as Kean states it, a sound-syntax interface (see also Bradley, Garrett, and Zurif, in press; Bradley, Garrett, Kean, Kolk, and Zurif, forthcoming).

The fact that patients who do not control this specialized closed class retrieval -- who treat open and closed class items alike at the point of lexical access -- are also agrammatic, strengthens this hypothesis. Further, it must be emphasized that the failure to distinguish open and closed class elements is not a consequence of brain damage in general; rather, it seems to be tied to the agrammatism of left-anterior brain damage. A number of posteriorly damaged patients presenting primarily with a word-finding difficulty in the context of grammatically well-formed utterances have already been tested and have shown the normal dissociation of the two vocabulary classes (Bradley et al, in press).

With this by way of summary and added detail, it should now be apparent that even though the distinction between open and closed classes is worked out by reference only to the phonological level, the processing ramification of this distinction appears at the point of lexical access, and not as some have claimed (e.g., Kellar, 1978; Brown, this conference) at the point of assigning a phonological representation to the acoustic input. To be sure, Kean's grammatical characterization is elaborated for English speaking aphasics in terms of stress and particularly, in terms of the fact that closed class items neither receive stress (except fo emphatic purposes) nor contribute to sentential stress patterns. But these are grammatical claims, and they are not to be taken as literally indicating that the brain damage underlying Broca's aphasia forecloses grammatical analysis simply by blocking off the analysis of unstressed (closed class) items on a purely acoustic basis. In fact, as Kean (1979), herself, has pointed out, closed class items carry no special physical signal which distinguishes them from open class items: stressless syllables abound in open class items, yet these are recovered by Broca's aphasics.

There is also experimental evidence to suggest that the Broca's comprehension problem is other than one of dealing with the acoustic structure of closed class items (Blumstein, Cooper, Caramazza, and Zurif, 1977; Caramazza, Gardner, and Zurif, 1979; Swinney, Zurif, and Cutler, in press). In the Swinney et al (in press) experiment, for example, though both Broca's aphasics and neurologically intact subjects responded faster to stressed than to unstressed words in a monitoring task, only the Broca's patients showed an effect of vocabulary class by responding to open faster than to closed class items, regardless of stress. Again, the fact that closed class items place an extra burden on the Broca's processing capacities presumably reflects the Broca's inability to preferentially access the special closed class file.

The fact remains, however, that Broca's aphasics do recognize closed class items as belonging to their language. Accordingly, it seems likely that the closed class is "doubly registered" -- once in its specifically accessed bin, which by hypothesis supports syntactic analysis, and again, in the bin that includes also the open class items. If so, normal operation would presumably require that the operation of the special closed class route block the operation of the frequency sensitive (open and closed class) access route. But there is as yet no independent evidence concerning the relative ordering, or for that matter, the relative speeds of operation of these two systems. And this remains an issue for future research.

Another issue is whether -- in complementary fashion to the syntactic function hypothesized for the closed class system -- the frequency sensitive system serves a predominantly semantic function. One straightforward manifestation of this possibility would be that Broca's patients can appreciate the semantic value of closed class items even though unable to use them as structural markers. This issue too ought to be addressed.

A final note to be entered here has to do with the already much discussed relation between comprehension and production processes. Kean rightly introduces the grammar as a component of the performance model, suggesting that it serves at least to specify the representations implemented or targetted by the processors. But although the information inherent in linguistic descriptions must be represented whether we speak or listen, the question remains concerning the extent to which the processes underlying speech and comprehension share components.

The data from Broca's aphasia bear intriguingly on this question: Given the convergence of agrammatic output, agrammatic comprehension, and the results of the lexical decision experiments, it seems reasonable to suggest at least some form of sharing -- the locus of the connection possibly residing in the lexicon (see Garrett, Frazier).

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Shared Aspects of Speech Perception and Production

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Both perception and production of speech have to relate utterances with structural descriptions. It would be surprising if two processes whose tasks are so related have nothing in common. I shall present psycholinguistic evidence to support the claim that many aspects of the representations used in both processes have many features in common, but this will not show that there are actual components used by both. To see the distinction, consider the motor theory of speech. This has been instantiated in many ways. The strongest form argues that speech perception is based on analysis-by-synthesis, with a production system serving as an integral part of perception by generating templates to be matched against the acoustic signal. A weaker form simply takes the data to indicate the need for a production taxonomy in classifying acoustic signals in a commonality of representation without an overlap of processors.

In what follows, I shall cite evidence from studies of exploitation of the lexicon to suggest the commonality of representation along three dimensions: the open class (content word)/closed class (function word) contrast; the meaning/form contrast; and morphological analysis. The comprehension studies ask the subject given an orthographic or acoustic stimulus to determine whether it embodies a word. The production studies use data on hesitancy, reaction time, and speech errors, such as spoonerisms.

The Open Class/Closed Class Contrast

Speech errors seem to reflect the centrality of the open vs. closed distinction in production. In exchanges, segments, words or phrases may get swapped. In word exchanges, such as

He thought that bones didn't have fish

the words exchanged are of the same class (usually open; I've only seen a few examples of closed class exchange), never do we see an open/closed exchange. In sound exchanges, such as

What I have is a case of lag grode flu

the exchanges seem restricted to open class words.

Work with Dianne Bradley, motivated by a view of parsing which places a heavy role on function words, showed an open/closed class distinction in the perception of words. The items were presented in isolation, with a visual exposure of 100 msec. For open class words, we found that reaction time varied with word frequency, with lower frequency words taking longer to recognize. However, closed class words exhibited a significant lack of frequency dependence in their frequency range (which is a subset of that for open class words). If this reflects the structure of lexical retrieval, then the retrieval mechanism must be different for the two classes.

Taft and Forster studied the dimension of left-to-right processing in reaction time to classify an item as a nonword. They found an item with initial segment a closed class item took longer to react to than a similar item which did not have such a prefix (e.g. toastle vs. poastle), but that there was no such effect for a suffix (as in pletoast vs. plepoast). Bradley and I carried out a similar experiment with closed class prefixes, and found that they did not affect reaction time -- 'thinage' had a greater reaction time than 'thonage', but 'thanage' and 'thonage' had similar reaction times.

Meaning/Form Contrast

David Fay and Ann Cutler have noted the dissociation of meaning and form in speech errors. In malapropisms like sympathy/symphony, no semantic relation seems involved; but one also sees semantic errors like tall/short, question/answer, fast/slow, finger/thumb in which there is no form relation. These dissociable errors in production seem to offer valuable cues about the structure of access to the lexicon.

The dissociation of meaning and form is also seen in comprehension, suggesting the independence of form search and meaning search of the lexicon. David Sweeney's studies show that even within a disambiguating context, we may still pick up effects of ambiguity of form of a word -- as when the word 'bug' in 'He used DDT to kill the bugs' can facilitate recognition of the probe word 'spy' as well as the probe word 'ant'.

Morphological Analysis

Taft and Forster show that stripping off prefixes plays a role in lexical retrieval, thus demonstrating morphological analysis in perception. There are also production errors which involve the separation of inflectional elements from their stems. For example, Fay notes that the error recounting/decanting becomes more explicable if one regards the choice of prefix as conditioned by the error in the stem. Other examples of morphological analysis occur in stranding exchanges like

That's a fantastic toy's kid.

and segment shifts like

Mary Baker's Eddy_.
They get weird_ everier day.

which "leave a hole behind".

The study of phrasal structure provides further evidence for commonalities of representation in both perception and production. In summary, then, it seems plausible to argue that both systems break down information the same way, with much similarity in the representation of lexical items. But whether or not there is any overlap in the use of subsystems by perception and production remains a totally open question.

Shared Components of Production and Perception*
Lyn Frazier: Comments on Garrett's Paper

In principle there are a variety of aspects of language processing that might be shared by the language production and comprehension systems. Information about the well-formedness constraints of the language might be mentally represented in some form which is neutral between production and comprehension and thus a common body of grammatical knowledge might be accessed and utilized in both tasks. Likewise, a common set of procedures might be employed to retrieve grammatical information, to schedule the flow of information and decisions and/or to guide the processor's decisions at choice points.

Garrett has suggested that the actual routines used in lexical retrieval may be common to the production and comprehension systems. However, his data only show that the same set of distinctions are evidenced in both tasks and thus are consistent with the weaker claim that it is only a common body of lexical information, not a common set of lexical retrieval procedures, which is shared.

Garrett's arguments address only the question of shared lexical information or lexical retrieval processes. The present comments will be addressed to the question of what syntactic information or processes may be shared. First we will take up the question of shared syntactic information. We will then turn to the question of whether there are shared processing units in production and comprehension. Finally, we will explore the implications of production-comprehension correspondences for the development of linguistic theory.

1. Shared syntactic knowledge

I am not aware of any arguments or direct evidence that a common body of syntactic knowledge is - or is not - shared by the production and comprehension systems. In the absence of such evidence, perhaps the only way to proceed is by examining what is known about the representation and use of syntactic information in production and perception to determine whether it is at least consistent with the claim that the production and comprehension systems exploit the same body of syntactic information.

Naturally there are several logical possibilities with respect to the mental representation of syntactic information. The syntactic information used in comprehension might be stored together with, and inextricably intertwined with, the decision principles and action plans used during sentence comprehension. Thus, the very form of the representation of this information might render it inappropriate or useless for purposes of production. For example, there is considerable psycholinguistic evidence that perceivers make certain systematic errors during the comprehension of sentences (cf. Bever, 1970; Frazier, 1978). In a sentence fragment like (1), perceivers tend to initially analyze the phrase <u>the solution to the problem</u> as a simple direct object of the verb <u>know</u>, as in (1a), rather than taking it to be the subject of a complement clause, as is necessary for the correct analysis of (1b).

(1) Nobody knew the solution to the problem....
 a. Nobody knew the solution to the problem by heart.
 b. Nobody knew the solution to the problem was easy.

The principles which guide the parser's decisions in such cases of (temporary) ambiguity have been called "parsing strategies." And, it is at least a logical possibility that these strategies are inextricably bound up with, and inseparable from, the representation of the syntactic information which is used during comprehension. For instance, the fact that a verb like <u>know</u> may legitimately take a sentential complement might be represented by a rule like (2).

(2) If a noun phrase which has been assigned as the direct object of a verb like <u>know</u> is followed by a finite verb, reassign that noun phrase as the subject of the following verb.

Rules like (2) may be used to specify the set of possible syntactic structures in a language indirectly, by specifying perceivers' first and preferred analysis of a sentence, together with permissible changes in this analysis (see, for example, Lakoff and Thompson (1975), where rules of this form were proposed).

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From the perspective of sentence comprehension, rules like (2) which directly encode parsing strategies into the representation of syntactic information do not seem too implausible. However, if speakers were to rely on such rules when they were formulating utterances, speakers would have to construct the same incorrect intermediate structures when they produced sentences that perceivers construct when they comprehend sentences. In producing a sentence like (1b), speakers utilizing a rule like (2) would be obliged to construct an intermediate structure in which the ambiguous noun phrase was assigned as the simple direct object of the verb know.

The claim that speakers construct the same intermediate hypotheses when they are planning a sentence that perceivers construct when they are garden-pathed by a sentence is extremely implausible and, to my knowledge, there is absolutely no evidence which supports it. Yet, if perceivers' strategies are directly encoded into the representation of the syntactic information used in comprehension, this is precisely what is predicted by the assumption that there is a common body of syntactic information shared by the production and comprehension systems.

Alternatively, the syntactic information used in comprehension might be stored together with, but not inextricable from, perceivers' parsing strategies. In the ATN framework, for example, syntactic information is represented as a network of "states" and "arcs" and perceivers' strategies may be represented in the network itself by ordering the alternative arcs leaving a state (cf. Kaplan, 1972) If we take this sort of representation seriously (rather than thinking of it as merely a convenient notation for indicating the ordering which is imposed on arcs by a separate body of strategies or scheduling principles), then in one sense syntactic information would be stored together with perceivers' strategies. However, the syntactic information contained in the network would not be inseparable from these strategies (as it was in the previous example). Speakers might use the syntactic information contained in this network when they were producing sentences but simply employ different principles (i.e. some principle other than the ordering of arcs encoded in the network) to govern the order in which they attempted different arcs. Hence, this alternative

would permit speakers to utilize the same syntactic information as perceivers, but it would not necessarily entail that speakers also relied on the same decision principles as perceivers (and thus it would not lead to the implausible prediction that speakers are garden-pathed by the same set of sentences that garden-path perceivers).

Finally, syntactic information might be stored by itself, completely independent from the processor's decision principles. In this case, syntactic information would be removed entirely from perceivers' parsing strategies and thus isolated from those aspects of processing where there is reason to expect differences between production and comprehension. Hence, this alternative would be entirely compatible with the claim that production and comprehension share a common body of syntactic information.

Fodor and Frazier (1980) argue at length for this third alternative. One of their arguments is that a particular parsing strategy, Minimal Attachment (below), can be explained very naturally if it is assumed that syntactic phrase structure information is stored in a separate rule library which must be accessed during sentence comprehension.

> Minimal Attachment: Incorporate incoming lexical items into the phrase marker being constructed using the fewest nodes consistent with the well-formedness rules of the language.

Given the assumption of a special rule library, Minimal Attachment will follow as an automatic consequence of minimal rule accessing. (in terms of time) Assuming that there is some cost/associated with accessing a rule, then accessing more rules will of course take more time. And thus if the parser merely accepts the first analysis available to it, this will automatically result in a preference for minimal attachments. In short, given the assumption of separate rule storage, the parser's preference for minimal attachments may simply be attributed to the general time pressures involved in sentence processing.

This explanation of Minimal Attachment enjoys several advantages over conceivable alternative explanations. Since the parser will accept the first analysis available to it regardless of the particular construction under consideration, this explanation accounts for the generality of the preference for minimal attachment across the wide variety of different constructions in English. And, since minimal rule accessing will be operative regardless of the particular details of the rules being accessed, this explanation permits minimal attachment to be generalized not only within a single language, but across different languages as well. Further, this explanation eliminates the need to postulate a special node-counting device in order to account for the parser's preference for minimal attachments. This latter point is especially important. Surely it is more in line with what is known about the human brain to assume that it simply performs operations in the quickest way it can (this is suggested, for example, by the prevalence of "horse-race" models in contemporary psychology) than to assume that the brain has evolved special counting devices whose only purpose is to evaluate the outcome of other operations solely on the basis of the number of steps taken to perform those operations.

Another argument which Fodor and Frazier present in support of syntactic information being stored separately from perceivers' strategies concerns the interaction of different parsing strategies. Specifically, when the parser's preference for low attachment and its preference for minimal attachment are in conflict, Minimal Attachment will prevail in some circumstances (when the minimal attachment site is visible within the restricted viewing window of the first stage processor) and the preference for low attachment will prevail in other circumstances (namely, when the low attachment is visible to the first stage processor, but the minimal attachment is not). If parsing strategies were stored together with the representation of syntactic information, then the interaction of these strategies is extremely difficult to explain. (In terms of an ATN representation in which arc ordering is encoded in the network, capturing this interaction requires placing completely _ad hoc_ conditions on arcs to insure that in cases of conflict the length and structure of preceding constituents, not the relative order of arcs in the network, will determine which strategy will prevail and thus which arc will be attempted first. See Fodor and Frazier, where this argument is laid out in detail).

If we accept the conclusion that the syntactic well-formedness constraints used in comprehension are stored separately from perceivers' decision principles, then we have at least established that the production system and the comprehension system _could_ share a common body of syntactic knowledge. In sum, the claim of shared syntactic knowledge is at least coherent and consistent with available evidence concerning the mental representation of syntactic knowledge.

Clearly the claim that the same body of syntactic information is exploited by both the production and comprehension systems is a stronger claim than one which allows the representation of the syntactic information used in production to differ in any way at all from the representation of the syntactic information used in comprehension. Hence, at present, surely the best working assumption is that the production and comprehension systems do access and utilize the same body of syntactic information.

2. Shared processing units

Another place where we might expect to find a correspondence between production and comprehension is in the size and nature of the processing units which are important in each of these tasks. Most of the structural units of linguistic theory have at one time or another been proposed as the important processing unit in terms of which sentences are comprehended (e.g. the entire sentence, the surface clause, the deep clause, every syntactic phrase, etc.). More recently, Frazier and Fodor (1978) have argued that the important units in sentence comprehension do not correspond to units of any one particular structural type but rather correspond to "phrasal packages" whose size depends largely on the length of the constituents in a sentence. Due to the restrictions on human short-term memory, these phrasal packages will typically contain roughly six or seven words. Thus, a short clause consisting of five or six words might be structured together into a single phrasal package, whereas a long clause might be divided up into a number of different phrasal packages (which would be integrated only at a later stage in the processing of the sentence).

Though the memory and computational capacity of speakers is not very well understood, it is probably safe to assume that there do exist some restrictions on the immediate memory and computational capacity available to speakers when they are formulating utterances. Hence, we might wonder whether these restrictions lead speakers to plan and execute utterances in 'chunks' which are roughly the same size as the phrasal packages which are constructed by perceivers during comprehension. A correspondence of this type would be of considerable interest, especially since it would suggest that listeners do not have to construct phrasal packages from scratch when they process a sentence, but rather they might reconstruct - or recover - phrasal packages which have been encoded into the acoustic signal by speakers during the production of the sentence.

Though this hypothesis has not been directly tested to date, there are a number of highly suggestive findings which support it. First, Fromkin (1971) reports that speech errors rarely involve elements separated by more than five or six words, which indicates that the units of sentence production correspond very nicely in size to the phrasal packages constructed by perceivers. Secondly, Grosjean, Grosjean and Lane (1979) measured the pauses produced by speakers during the reading of familiar material. This study indicated that the best predictor of pause location was a metric which took into account not only the syntactic structure of the sentence, but also the distance of each possible pause location from the midpoint of the sentence. All of the example sentences which are presented in this study are twelve words in length and thus the midpoint of these sentences was always between the sixth and seventh word of the sentence. Thus, assuming that speakers are most likely to pause between planning units rather than within a planning unit, this study also suggests that speakers are organizing sentences into chunks which are roughly six or seven words long (though, as expected, the exact length of these chunks also depends on the constituent structure of the sentence).

Suci (1967) demonstrated that subjects find it easier to learn a list of sentences if the presentation of the sentences respects the pausal segmentation of the sentence (i.e., if the words which would be included between two pauses when a speaker produces the sentences are presented together) than if the presentation respects only the syntactic structure (major constituent

segmentation) of a sentence. This finding implies that the units of sentence production are also ideal units for purposes of perception and thus it lends further support to the hypothesis that phrasal packages are involved in both the production and perception of speech.

In a study of pauses in spontaneous speech, Boomer (1965, p.151) notes that "In order to exceed six or seven words a clause must usually include one or more extended anacolutha ...", or syntactically mixed constructions. Boomer's interest was in the study of pauses *per se* rather than in the presence or distribution of anacoluthic expressions, and thus he does not present the data on which this observation was based. Nevertheless, the observation is intriguing since it again supports the notion that speakers must restrict the length of their planning units or else risk exceeding their capacity and thus forgetting or losing access to the syntactic commitments which they have taken on in earlier portions of the sentence.

In short, a variety of psycholinguistic findings suggest that speakers plan sentences in units which consist of a constituent or series of constituents which are roughly six or seven words in length. And thus there is converging evidence from different types of production studies which support the hypothesis that phrasal packages are the basic processing units in production as well as in comprehension.

3. Shared complexity rankings

From the perspective of linguistic theory, one of the most interesting correspondences between the production and comprehension systems would be a correspondence in the relative complexity rankings they assigned to different sentence types. That is, we might expect the grammar of a language to be most heavily influenced by performance considerations, not in cases where the exigencies of sentence production and the exigencies of sentence comprehension are at odds, but rather in cases where the two systems coincide and thus both exert a pressure on the language to change in the same direction.

If some construction or sentence type is particularly easy both to produce and to comprehend, we might expect that construction to be unmarked and frequently occurring both within a single language and across different languages. Similarly, if a construction places an especially heavy burden on both the production system and the comprehension system, we would expect the language to either develop a simpler alternative construction or incorporate some constraint which excluded the complex construction from the language. Of course, at present very little is known about the complexity ranking which the sentence production system assigns to different sentence types. However, if we pursue the reasoning of the previous section (where it was argued that the limitations on speakers' immediate memory and computational capacity lead them to plan and produce utterances in terms of phrasal packages), then there are a variety of examples which may reasonably be argued to be cases where the complexity ranking of the production system and the comprehension system coincide.

Assuming that speakers and hearers process sentences in terms of phrasal packages, it seems likely that both speakers and hearers would prefer to have items which form a coherent semantic unit occur adjacent to each other in the lexical string (where they may be structured together into the same phrasal package), rather than having these items separated by some long intervening constituent (in which case these items might very well end up in separate phrasal packages). If this "adjacency preference" is in fact shared by both the production and comprehension systems, then this is a case where the grammar could accommodate itself to the needs of both systems simultaneously and thus we would expect the"adjacency preference" to have a quite strong impact on the grammars of different languages. In Frazier (1979) it is argued that this preference explains many of the implicational universals proposed by Greenberg (1965). For example, it accounts for the tendency for postpositional languages to place relative clauses before their heads, genitives before their governing noun, etc. (see discussion of the "In-positional Universal" and the "Head Adjacency Principle" in Frazier, 1979). This of course supports the notion that constructions which are particularly easy both to produce and to comprehend are "unmarked" and thus correspond to the expected case across a wide variety of different languages. And, in terms of developing a theory of markedness (such as "Core Grammar", cf. Chomsky and Lasnik, 1977) it may be important to distinguish unmarked constructions which might be widespread simply because they have been favored by both the production and comprehension systems from constructions which are unmarked but may only be attributed to language-learners' initial hypotheses about the structure of the language. In short, the theory of markedness will be more revealing of the basic structure of the language acquisition device if we are able to abstract away from (or separate out) the influence of the adult production and comprehension systems.

Turning to constructions which are particularly difficult to process, we may begin by considering a construction which is relatively difficult to comprehend, but not to produce. As mentioned earlier, a sentence like (3) is more difficult to comprehend than a sentence like (4), where the presence of the complementizer _that_ prevents the parser from incorrectly interpreting the phrase _the solution to the problem_ as a simple direct object of the verb _know_. However, from the perspective of sentence production, there is no reason to expect (3) to be any more difficult than (4).

(3) Nobody knew the solution to the problem was easy.
(4) Nobody knew that the solution to the problem was easy.
(5) Nobody knew the air was polluted.
(6)*Nobody knew the air.

Given the perceptual complexity of (3) relative to (4), one might have thought that the grammar of English would simply prohibit complementizer deletion in sentences like (3) where the temporarily ambiguous noun phrase (_the solution..._) may coherently be analyzed as a simple direct object of the preceding verb (as opposed to (5), where this analysis is semantically incoherent). However, given the devices standardly available within a restrictive theory of syntax, a constraint on complementizer deletion could not be formulated in such a way that it would exclude all and only those instances of complementizer deletion which result in perceptually complex sentences (i.e. the constraint would be unable to discriminate between sentences (3) and (5)). In this situation there are only three choices available to the grammar: (a) it could prohibit complementizer deletion across-the-board, thereby excluding the perceptually complex sentences, together with a large range of sentences like (5) which do not pose any particular problem for the sentence comprehension mechanism; (b) the grammar might incorporate some entirely new type of device which would permit it to exclude all and only the perceptually complex sentences; or, (c) the grammar might do nothing at all (i.e. the language might simply tolerate the perceptually complex construction). It appears that in general the grammars of natural languages do not resort to extreme options like (a) or (b) in response to constructions which are only difficult to comprehend, but not to produce (see discussion of the Minimal Exclusion principle in Frazier, 1979).

But suppose (counterfactually) that exactly the same instances of complementizer deletion which cause difficulties for the sentence comprehension mechanism also caused problems for the sentence production system. We might speculate that under these circumstances the grammar of English might develop a constraint excluding all and only the complex constructions even though this would entail incorporating some new device into the grammar or relaxing the usual restrictions on the form of syntactic rules. In other words, when the production and comprehension systems are in collusion, speakers and hearers might be willing to "bend" the rules of the grammar or, perhaps, ignore them altogether(see below).

Though speculative, this line of reasoning leads to the hypothesis that whenever we find a "wrinkle" in the grammar (i.e. exceptional behavior whose grammatical treatment requires either expanding the vocabulary of linguistic theory or incorporating some new and otherwise unwarranted type of mechanism into the grammar) the 'offensive' construction may be attributed to the collusion of the production and comprehension systems. (The implications of this "Collusion Hypothesis" will be spelled out in a moment.) To take a specific example, in general syntactic rules are completely insensitive to the length of constituents; typically one does not find rules which, say, specify that a verb phrase may consist of a verb followed by a long noun phrase, but not of a verb followed by a short noun phrase. However, there are a few well-known exceptions to this generalization: the rule of Particle Shift (which is responsible for the atrocity in (7b)) and

the rule of Heavy Noun Phrase Shift (which is responsible for the sentence in (8b)).

(7)a. Henry sent out some reports about the numerous accidents that occurred at Three Mile Island last year.
b. ?Henry sent some reports about the numerous accidents that occurred at Three Mile Island last year out.

(8)a. John gave a copy of his latest article about the role of the media in the militarization of American society to Susan.
b. John gave to Susan a copy of his latest article about the role of the media in the militarization of American society.

(9)*John gave to Susan a book.

In the case of Particle Shift, accounting for the apparent sensitivity of the rule to the length of constituents is not problematic, since there are independent reasons for supposing that sentences like (7b) in which the particle has been moved over a long intervening constituent will be unacceptable (cf. Frazier and Fodor, 1978). However, the sensitivity of Heavy Noun Phrase Shift to the length of the moved constituent is problematic if we wish to maintain the generalization that syntactic rules can not refer to the length of constituents; regardless of whether we formulate this rule as an extraposition rule (in which case we must explain why a short noun phrase like the phrase *a book* in (9) can not be extraposed) or as an intraposition rule (in which case we must explain why the rule is obligatory just in case the relevant noun phrase is short) it appears that the rule must make reference to the length of the moved constituent. (Notice that this same argument goes through even if we assume that it is the prepositional phrase, rather than the noun phrase, which is moved.) And, by contrast with the Particle Shift example, we can not explain these facts by appealing to the notion of an unacceptable but grammatical sentence since there is no reason to believe that the sentence comprehension system would find a sequence of *short* phrases (such as the prepositional phrase and noun phrase in (9)) particularly difficult to process. In other words, the problem in the case of Heavy Noun Phrase Shift is that we must explain why a construction *is* permissible just in case certain constituents are long, rather than why a legitimate construction is *not* permissible just in case certain constituents are long.

In the model of sentence comprehension proposed by Frazier and Fodor (1978) there are clear reasons for expecting that a sentence like (8a), which contains a "heavy noun phrase" which has not been shifted to the end of the sentence, should be difficult to parse. If the first stage processor receives a long noun phrase, such as the direct object in (8a), then by the time it receives subsequent material (e.g. the prepositional phrase to Susan) the lexical material preceding the long noun phrase will no longer be available within the restricted viewing window of the first stage processor. Thus, in a sentence like (8a), the first stage processor will not have access to the correct attachment site for the phrase to Susan (i.e. to the VP-node dominating the verb give) at the time when it encounters this prepositional phrase. However, if the heavy noun phrase is shifted to the end of the sentence (as in (8b)), this problem will not arise since the first stage processor may incorporate the phrase to Susan into the same phrasal package that contains the verb give. (And, of course, there will be no problem parsing the long direct object since all of the items contained in it may correctly be structured together with other nearby items and the resulting phrasal packages may be integrated with preceding material by the second stage processor. See Frazier and Fodor for details.)

A similar argument holds in the case of sentence production. In sentences like (8a), where the long noun phrase has not been postposed, speakers must remember the predicted prepositional phrase while they are elaborating the long intervening noun phrase. By contrast, in planning and producing sentences like (8b), speakers may relieve themselves of this commitment before they begin elaborating the longer and more complex noun phrase. Hence, the rule of Heavy Noun Phrase Shift appears to facilitate the task of the sentence production system, as well as the sentence comprehension system. In other words, though the rule of Heavy Noun Phrase Shift must refer to the length of constituents, there are independent reasons for believing that, as predicted by the Collusion Hypothesis, 'unshifted' heavy noun phrases create complexities for both the production and comprehension systems -- complexities which are not engendered by the alternative (shifted) constructions created by this exceptional rule.

If the Collusion Hypothesis can be maintained, it will provide a principled means for imposing stringent constraints on the form of syntactic rules in general, by permitting those constraints to be relaxed under very restricted circumstances (i.e. in cases of collusion). Alternatively, if a sufficiently detailed theory of acceptable ungrammaticality can be articulated (so that it will not incorrectly predict that any intelligible ungrammatical string should be judged to be well-formed by speaker-hearers of the language, cf. Langendoen and Bever, 1976), then perhaps the rule of Heavy Noun Phrase Shift could be banished from the grammar entirely. If so, then the Collusion Hypothesis may be viewed as simply an initial step toward developing a more constrained theory of acceptable ungrammaticality.[1] In either case, it appears that identifying production-comprehension correspondences will permit us to construct more restrictive theories of natural languages without having to dismiss certain troublesome data in a relatively unprincipled fashion.

[1] This latter approach is especially tantalizing in so far as it might provide some insight into the genesis of "free word order" languages. That is, if in cases of collusion speakers and hearers are willing to simply violate certain rules of the language and then proceed to systematically do so in circumstances which are governed by 'performance' factors and thus are not describable in terms of a set of innately-specified grammatical categories or in terms of a natural class of structural conditions, language-learners might well conclude that there are only (or predominantly) stylistic constraints on the word order of the language they are acquiring.

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Sheila E. Blumstein: Comments on Garrett's Paper

Models of linguistic competence assume a final common pathway between perception and production mechanisms of both speech and language. Such a view is intuitively satisfying as it provides an economic characterization and conception of speech-language processing. Nevertheless, as parsimonious as such a theory might be, it may represent a vast oversimplification of the processes involved in language use.

To address this question, I will confine my remarks to the relation between perceptual and production abilities in the speech of aphasic patients. There is a large amount of data in the literature which would seem to support the view that perception and production processes go hand in hand. Analyses of production errors (Blumstein, 1973; Lecours and Lhermitte, 1969) and perceptual confusions (Blumstein, Baker, and Goodglass, 1977) show that aphasics are more likely to show deficits in single destinctive feature contrasts as voice or place than double feature contrasts. Moreover, a similar hierarchy of feature breakdown can also be shown (Blumstein, 1973; Blumstein et al., 1977). Nevertheless, although these findings would seem to support similar organizational principles for the two systems, they do not necessarily indicate that they are subserved by a common level of processing.

One means of investigating the possible commonality between such processes involves exploring the relation between production and perception abilities. That is, are production deficits followed by concomitant perception problems and vice versa, or can there be a dissociation between the two? Obviously one can always find dissociations between production and perception, e.g. an adventitiously deafened individual clearly represents an example of such a dissociation. What I am referring to here is a dissociation at a more central level of processing.

A recent investigation on the perception and production of voice-onset time speaks to this question (Blumstein, Cooper, Zurif and Caramazza, 1977). Voice-onset time (VOT) represents the timing relation between release of a consonant and onset of glottal pulsing and corresponds linguistically to the phonetic dimension of voicing, e.g. [p] - [b]. Perception abilities were measured by discrimination and labelling of a voice-onset time continuum, and production abilities were measured by acoustic analysis of the VOT of spoken utterances. Results can be summarized as follows. Posterior (Wernicke) aphasics could discriminate VOT, but could not differentially label the stimuli thus discriminated. Their inability then to use this dimension in a linguistically relevant way suggests a more central (i.e. linguistic) rather than peripheral (i.e. low-level auditory, non-linguistic) deficit. Their VOT productions on the other hand were similar to normals. In contrast, Brocas's aphasics generally exhibited good perception of VOT, as measured by both labelling and discrimination. However, they showed a marked deficit in VOT production, particularly in articulatory implementation manifested by phonetic errors, but also in phonological planning, manifested by phonemic errors (Blumstein, Cooper, Goodglass, Statlender and Gottlieb, 1980). These results suggest then that speech perception and production abilities can be dissociated by different lesion sites, and more importantly suggest that despite sharing common underlying linguistic principles, the production and perception mechanisms may be organized independently as two separate systems.

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DISCUSSION OF KEAN AND GARRETT PAPERS

Marshall: We must distinguish commonality of knowledge from commonality of mechanism. A similar partitioning of classification in comprehension and production does not imply a commonality of knowledge or process.

Levine: The fact that we can understand what another speaks, in particular that we can appreciate and laugh at slips of the tongue, must mean that there are overt similarities between the two systems, whatever their internal mechanisms.

Arbib: You learn to produce the language that you comprehend, and vice versa. But the fact that two systems are tuned by some external process to be "inverse" to one another in no way implies commonality of subsystems. As I suggest in my paper, syntax in production could enter as a side-effect of a planning and translation process, quite different from its explicit role in parsing.

Hudson: My Ph.D. thesis offered a new vocabulary based upon a psycholinguistic analysis and developed a process model for the language process; the point then was that a diagram such as a brain diagram which is a classic copy of standard anatomical approaches, can only be fruitful, and extend beyond mere location of symptom, if the functions that are located are expressed in some functional/process vocabulary. Only such models would enable us to partition what is and what is not localised/

localiseable in terms of language functions. Only such models will enable us to finally tie the Galaburda-cytoarchitectonics / Arbib-nerve nets / Language processes / Kean-Linguistics mess together. Without such there is no reason for the different approaches not to pass each other completely by. One of the problems in the linguistic approach is that the physical parameters just cannot appear in any current model. This is to say that a Linguistic approach wouldn't distinguish between the slow speech rate effects of the Broca and the highly fluent (if unintelligible) productions of the Wernicke's aphasic. Nor do they include the critical dimension of the patient's awareness of his situation. On the other hand the cytoarchitectonics can't tell us whether a bit of brain will work faster or slower or not at all when damage occurs. To tie the whole nerve cell-syntax and semantics continuum together we certainly need more highly articulated models.

Halwes: Experiments suggest that some particular syllables, particular acoustic tokens, could be described as intrinsically ambiguous. For example, one such token might be evaluated as, say, 30% 'ba', 20% 'ma' and 50% 'va' on the basis of averaged response distributions. But thismultiplicity does not seem to be a part of our conscious perceptual experience. On a given occasion, we hear only one of the alternatives, and are not aware that we "could have heard" some other syllable. (See especially Spencer and Wollman, 1980.)

To us, this suggests that conscious perceptual experience

results from a generative process (rather than a merely interpretive process). Accoustic phonetic information is used, along with other evidence, to constrain the construction of a determinate perceptual form. That process of construction may have much in common with processes involved in speaking.

Marcus: I have the beginning of a production model (not the one in my thesis) that might explain Bradley's result on open vs. closed class items. Imagine a parsing grammar based on "demons", i.e. pattern-action rules. To distinguish "A hundred pounds" vs. "A hundred pound block", we need grammatical categories for "a", for numbers and for measure terms. We thus need specific lexical items and lots of housekeeping rules in the parsing grammar. Imagine that the parsing is done by a large parallel machine, so that there are lots of rules looking for their preconditions in parallel. When looking for an adjective, we can let it bubble up through standard lexical access. But 'all', 'both' and 'a' are function words occurring in many patterns, and so we can run tests for all these words in parallel (thus the standard response time) to find the appropriate rules. Since the mechanisms for open and closed class items are different, the retrieval times should differ, but there is no reason one should be faster than the other.

Woods noted that function words are hard to detect by bottom-up phonemic analysis. This could be compensated for by the mechanism I have posited, instantiating a rule to look for each function word on the basis of limited use of context. This would certainly distinguish function words from unstressed content words.

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Language as a Translation Process

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Introduction

In arguing against reductionism, Jerry Fodor has recently written:

> If psychology is reducible to neurology, then for every psychological kind predicate there is a coextensive neurological kind predicate, and the generalization which states this coextension is a law. (Fodor 1975, p.17

He goes on to say:

> Yet, as has been frequently remarked in recent discussions of materialism, there are good grounds for hedging these bets. There are no firm data for any but the grossest correspondence between types of psychological states and types of neurological states, and it is entirely possible that the nervous system of higher organisms characteristically achieves a given psychological end by a wide variety of neurological means. (Fodor 1975, p. 17)

and further:

> What I have been doubting is that there are neurological kinds coextensive with psychological kinds. What seems increasingly clear is that, even if there are such coextensions, they cannot be lawful. For it seems increasingly likely that there are nomologically possible systems other than organisms (viz., automata) which satisfy the kind predicates of psychology but which satisfy no neurological predicates at all. (Fodor 1975, pp. 17-18)

According to Fodor, what is needed is a medium of representation for the computation required for language processing. But the hardware involved might be employed in any number of ways. The position is evidently quite pervasive. Geschwind (1974), one of the most outspoken localizationists has said:

> I would have to accept the view that what one might call the realization in hardware of an axiomatic system would not necessarily be, so to speak, isomorphic with that system. One would hardly expect that damage to an ideal computer, capable of deriving theorems of Euclidean geometry, would produce loss of individual axioms (p. 505)

I find two objections to this line of reasoning. The first one is theoretical. If all the hardware provides is a medium in which the (presumably mentalistic) computation takes place, one is left with the problem of who or what is doing the computation. Any theory of language which leaves one with little men doing the job we are trying to account for is no theory at all, since one is left having to explain how these homunculi work.

My second objection is on empirical grounds. If the hardware does nothing more than provide a medium for computation, how is one to explain the large number of data relating site of lesion with behavioral deficit in uniform ways across speakers and across languages? Kean (1978) makes a similar point regarding the characteristic symptomatology of Broca's aphasia across speakers of different languages.

Computers can perform grossly differing logical operations with constant hardware because they have been built in such a way that the flow of logic depends largely on software which is interchangeable. And this point pertains only to general purpose computers. Special purpose computers do not share this property. There seems to be little reason to compare the human nervous system to a general purpose computer.

Since people did not build human neurological systems they do not know how they work a priori. Therefore, it seems recommendable to follow the admonition of Jason Brown (1977) when he says:

> Symptoms should, in fact, be the mortar of the psychology, not just chosen to illustrate this or that theoretical formulation, since the diversity of clinical symptoms is such as to support by the manner of selection almost any a priori assumption. (p. 2)

I am not trying to argue that the route to discovering how language is represented in the brain is by localization studies. On the contrary; localization is no substitute for explanation: with more refined localization, the homunculi just get smaller and smaller, doing smaller and smaller jobs. Arbib and Caplan (1979) make a similar point in their position position paper in their critique of "faculty models".

Lamendella (1979) in his review of neurolinguistics for the Annual Reviews of Anthropology puts it as follows:

> Theoretical linguists could not be content to merely identify a "phonological" level of language structure and not go on to derive a theory of the organization of phonological structures. Theoretical neurolinguists should not be content to identify a "motor speech region" (Broca's area) or a "sensory speech region" (Wernicke's area) without having as a high priority the description of the organization of speech functions and subfunctions which accounts for the capacity of these areas to process speech.

Thus the question of where language faculties and speech functions are located seems premature. 'What' and 'how' are what we need to know before the question of 'where' becomes intelligible. So we have to the problem of having to know what it is we are seeing linguistically when we "see something" neurologically.

There are some who would argue as Von Eckhardt Klein (1978) that

> It is the job of linguistic theory to provide us with a description of a person's knowledge of his language: it is the job of psycholinguistics to provide us with a description of the processing mechanisms by which that knowledge is put to use. Although neurological evidence may lead to insights as to the most appropriate functional theory and certainly must be consistent with such theories, in general, research must proceed from psychology to neurology. The reason is simple. We will not be in a position to discover how LRCS is realized neurologically until we know what is so realized. In other words, evidence of the neurological realization of LRCS cannot be properly evaluated except in the context of linguistic and psycholinguistic models of LRCS. (p. 5) [LRCS = language responsible cognitive structure

But I think that this misses the crucial point that defining human language is not the same as defining an electric motor, or even a general-purpose computer. Language can be defined in many different ways depending on perspective (e.g. social, psychological, cultural, political, formal, etc.) and even within a single perspective. As Bresnan (1978), speaking from a more or less cognitive perspective puts it:

> The difficulty in linguistics is that we can characterize our knowledge of language in too many ways. What has been called the grammatical characterization problem — the problem of representing the language user's knowledge of language — is not very well defined. Therefore, it does not seem reasonable to argue that the grammatical characterization problem should be solved in advance of what has been called the grammatical realization problem: it is not clear that we will ever come to a solution to the grammatical characterization problem as it has been defined in the past.
>
> But the grammatical realization problem can clarify and delimit the grammatical characterization problem. We can narrow the class of possible theoretical solutions by subjecting them to experimental psychological investigation as well as to linguistic investigation.
>
> (p.59)

The point can be applied to neurolinguistics as well. The way language appears to be represented neurologically can give us information as to how language is to be characterized. So I would see the proper direction for investigation to be opposite to that proposed by Von Eckardt Klein: symptoms should be the mortar of the theory.

A theory of language must be relatable to and must conform to what we know about the nervous system, based on evidence from pathology as well as from other investigatory paradigms. There can be little doubt of the hierarchical nature of the nervous system, in which higher levels exert control over lower levels and in which lower levels operate more or less autonomously in the absence of control exercised from above, and constantly

provide input and feedback to higher levels. It can hardly be disputed that language functions to relate cognitions, thoughts, ideas, feelings to a form of representation such as sound or writing for transmission to another organism; and to receive the transmitted form and relate it to cognitions, feelings, etc. of the receiver. As a higher cognitive function, language is part of the larger hierarchy which is the human mind-body, and as such, displays hierarchical structure as well. Thus, one can think of encoding as a process by which thoughts are first put into predicational form (in which arguments are distinguished from predicates, topics from comments, etc.) and along the line, major lexical items are chosen (but not always). From this point, the message is filtered through language "housekeeping" rules, traditionally called (surface) syntactic, morphological, and morphophonemic rules or constraints, before being passed on to be mesotically encoded.[1] The mesotic will usually but not always involve phonology. Since each level will have input from the next lower level, lower levels will infact influence higher ones. Thus the level of cognition could indeed be influenced by the linguistic structure of the language of the speaker. So one can thus conceive of a linguistic hierarchy in which the highest level is cognitive and is really not linguistic <u>per se</u>. The next level down is semantic, in which thought is "linguisticized" into predications, and in which, usually, major-class lexical items are drawn from the lexicon. The next level subjects these predications to individual constraints. Here we find superficial syntax and inflectional morphology, and perhaps some derivational morphology, although I suspect that the larger part of the latter is provided ready-made by the lexicon. At this point the message can be encoded phonologically, graphemically, or otherwise, and hooked up to the appropriate peripheral motor command systems.

Note that the foregoing discussion entails that a human language is not a set of sentences, but rather a transducer between thought and mesotic (or means of transmission). In <u>de</u>coding it transforms mesotically encoded messages into thought, but I shall postpone discussion of decoding until I have given evidence for the conception I am presenting.

<u>Evidence from Language Pathology</u>

I wish now to present evidence for considering Wernicke's Area to be the site of what I have been calling the semantic or linguistic predication function and for considering Broca's area to be the site of what I have been calling the "housekeeping" function.

Broca's Aphasia--

In a Broca's aphasic, Wernike's area is intact. In the speech of a Broca's aphasic, contentives are present and we find basic predicational relationships intact. Broca's aphasics speak as though they were trying to communicate in a language of which they do not know the grammar. When a normal adult learns a foreign language, what he in fact has to learn is "housekeeping rules" (as well, of course, as vocabulary, phonology, phonetics and orthography). He does not need to learn basic predicational relationships. This kind of speech uttered by the Broca's aphasic is understandable most of the time. We can usually understand telegrams as well, The language used in telegrams differs from normal language by virtue of its elimination of many of the "housekeeping" elements of ordinary language. Pidgins originate in an attempt to facilitate communication with non-speakers of a language by reducing the "housekeeping" rules.

The receptive ability of Broca's aphasics is generally good. This makes sense. One usually does not need "housekeeping" rules to understand what someone means. One can understand a foreign language , having only imperfect

command of the grammar. But in specific receptive tasks requiring attention to morphology or to superficial syntax, Broca's aphasics fail.

Luria (1975) discusses two kinds of tasks presented to aphasics.

> One set involved testing patients' assessment of constructions expressing logical relationships (such as "the father's brother" vs "the brother's father," or "a triangle over a circle" vs "a circle over a triangle") by having them explain the difference in meaning or point to a diagram picturing the relationship.

This task would involve the semantic ~~or~~ predicational level in the hierarchy, which I attribute to Wernicke's area.

> The other set of tasks involved asking the patient to judge the grammaticality of sentences among which were included sentences containing morphosyntactic errors of a relatively superficial nature (e.g., "Parokhod idet povod*oj*" (The steamship sailed through by the water) or "Zimoj ludi kataiutsja na sania*my*" (In winter people travel about on with sleighs)), and asking the patient to correct the errors if possible.

This task, on the other hand, would involve what I have called "housekeeping rules". Luria finds that patients with anterior lesions have little trouble in performing the first type of task, but they find difficulty in evaluating the grammaticality of and correcting the morpho-syntactic (i.e., "housekeeping") errors in the sentences in the second set of tasks. This result follows from what I have been claiming.

If this interpretation of Broca's aphasia is essentially correct, how does one account for the presence of phonemic paraphasias so frequently found in the speech of Broca's aphasics? I would claim that since the level of phonological realization gets input from Broca's area, an impaired Broca's area would send down an imperfect signal which could not be completely specified phonologically. The paraphasias could then arise from indeterminacy in the signal. If this is correct, Broca's aphasics should be able to recognize phonemic paraphasias in receptive tasks, even though they do not recognize agrammatism, as Luria notes. The reason for this will be come clear later.

Eric Keller (1979) has recently been arguing rather persuasively for a distinction between planning and execution stages in speech production.

In so doing, he argues against a strict separation of phonemic and phonetic processing in speech. If he is correct, then phonemics may belong strictly to the realm of perception. In production, Broca's area sends signals consisting of lexical and grammatical formatives encoded for motor commands. If Keller's position is correct, that is, if there is no separation between phonemic and phonetic encoding, one wonders why phonemic paraphasias found in Broca's aphasics characteristically do not violate the phonotactic constraints of the language. I would suggest that this may be because the motor-command packages signaled are highly overlearned, and when imperfect signals are received from Broca's area, the context sensitive motor commands continue to operate normally. The imperfect signal from Broca's area does not distort the motor-command package (which is the product of a lower level); all it does is signal the wrong one.

Wernicke's Aphasia--

In Wernicke's aphasia, Broca's area is intact. I have claimed that Wernicke's area handles semantics--basic predicational functions. In the speech of Wernicke's aphasics, we find a lack of intelligibility of message, due to the absence of logical structure. We find superficial syntax, morphology, morphophonemics, etc. preserved. The speech patterns of Wernicke's aphasics arise from a functioning Broca's area taking care of houskeeping, but receiving a distorted input from Wernicke's area. Naturally, comprehension is significantly impaired in Wernicke's aphasia since what comprehension basically is, is the interpreting of the encoded message into its basic predicational functions. The housekeeping rules play only a minor role in this.

Luria notes that in the studies cited, patients with posterior lesions have little difficulty with the grammaticality judgments' task, but have a great deal of trouble assessing the logical relationships. The grammaticality judgments are handled by the intact Broca's area.

In terms of the approach I have been discussing, one could account for other syndromes in a straight-forward manner as well. Dementia could be looked at as a nonlinguistic deficit affecting the cognitive level and providing impaired input to the semantic predicational level (Wernicke's area). Isolation syndrome could be looked at as a disconnection between the linguistic and cognitive levels or, in the case of a severely damanged cognitive level, a linguistic system operating in the absence of cognitive input. At least one type of anomia may be due to disconnection in the lexical access path between the cognitive level and the semantic predicational level. Thus such anomics know what they want to say and do not demonstrate agrammatism, but cannot access the lexical item they wish to use. This could be because the cognitive matter on being put into logical predicational form in Wernicke's area arrives without lexical encoding, due to some kind of disconnexion between the lexicon and Wernicke's area. I really do not wich to speculate on this any further in view of the sizable number of different anomic-type syndromes for which Benson (1979) has adduced evidence.

It would seem that the agnosias, the dysarthrias, and perhaps conduction aphasia are not language deficits *per se*.

Linguistic Theory

In *Aspects of the Theory of Syntax* (Chomsky 1965) in the context of warning against transformational grammars being misconstrued as model of speech production, Chomsky said:

> a reasonable model of language use will incorporate, as a basic component, the generative grammar that expresses the speaker-hearer's knowledge of the language; but this generative grammar does not, in itself, prescribe the character or functioning of a perceptual model or a model of speech production. (p.9)

A good deal of research was devoted to trying to discover how a generative grammar might be incorporated in a model of language use. In spite of this, no one was able, finally, to construct a model which incorporated competence grammars of the Chomskyan type which offered any evidence of psychological validity. The results led Bever to conclude that " the relation between linguistic grammar based on intuition add that based on the description of other kinds of explicit language performance may not just be 'abstract' (as maintained by Fodor and Garrett) but may be <u>nonexistent</u> in some cases" (Bever 1970, p.34, Bever's italics).

The failure to find such a relationship led to a reduced interest in transformational grammar among psycholinguists. Some researchers, however, have decided that it may be the linguistic theory which is at fault, and that a criterion for grammar selection should be psychological plausibility (Bresnan 1978). Probably the property of transformational grammars which has shown the least likelihood of having psychological reality has been the transformations themselves. Recently, however, a number of linguists (for example, Brame, Wasow, Bresnan, Jackendoff) have been arguing for drastic reduction of the number of transformations and for a large number of tasks previously assigned to the transformational subcomponent to be relegated to the lexicon.

Chomsky himself, on "internal linguistic grounds", has been gradually reducing the number of transformations up to the point of proposing that the transformational subcomponent may be reducible to one rule, "Move Category" (Chomsky 1977). Furthermore, the development of "trace theory", first introduced by Chomsky in 1973, has allowed for semantic interpretation to be done entirely at the level of surface structure. Since one of the principal arguments for a level of syntactic deep structure had been that this was the only point at which (certain significant aspects of) semantic interpretation could take place, the introduction of trace theory significantly weakens the case for deep structure. With transformations and deep structure significantly reduced in importance to linguistic description, a number of linguists have been moved to do away with them altogether and to develop alternative theories of grammar.

In the past few years a number of theories ofgrammar have been proposed which can be grouped, I think, into three main types.

The first type is what I call <u>functional</u>. Here I mean 'functional' in the sense of accounting directly for communicative function by means of the model. In this group I place Dik's "functional grammar" (Dik, 1978), Foley and VanValin's "role and reference grammar" (in preparation), augmented transition networks, being developed by a number of investigators (q.v., e.g. Woods 1970, Kaplan 1972, Wanner and Maratsos 1978) and Lakoff and Thompsons "cognitive grammar", which makes use of augmented transition networks (1975a,b). McCawley's recent work also seems to be 'functional" in this sense, although he still retains transformational derivations. Clearly the "HEARSAY" system espoused by Arbib and Caplan (1979) would belong to this group. Although these approaches differ markedly in their formal properties, they share the conception of language as a communicative tool and regard the goal of linguistics to be the explication of how the tool works to achieve its end.

A second type of theory may be charactierized by its emphasis on an enriched surface structure. Such models include only one level of morpho-syntactic structure. This is achieved by including a lot more in a representation than merely trees with syntactic category labels: semantic and syntactic functions and relations are all included at the single level as well. In this group I would classify Richard Hudson's "daughter-dependency grammar" (Hudson 1976;

and Michael Kac's "Corepresentational Grammar (Kac 1978).

I label the third group "axiomatic definitional" systems, for want of a better label. In these, a variety of techniques are used to formally specify the language. The most obvious example of this is Montague grammar, of which a clear exposition for the non-logician can be found in Partee (1975). A Montague grammar consists of recursive definitions of sets of sentences. I also classify Gerald Sanders' "equational grammar" (1972) in this group. Sanders' approach involves statements of which linguistic representations are equivalent or nonequivalent to which other linguistic representations. These statements can theoretically be used to prove equivalence or nonequivalence between semantic rppresentations and phonetic representations.. I also think that Brame's (1979) "realistic grammar" (somewhat different from Bresnan's realistic grammar in that the latter still uses transformations) belongs to this group. Relying heavily on lexical specifications, the grammar consists essentially of compositional and interpretive rules.

I have not intended this list to be exhaustive, but merely illustrative of the various trends in linguistic theory at the present time. And it should be borne in mind that the approaches referred to differ greatly in the extent to which they have been worked out in detail, many of them being only at the stage of offering a promissory note.

Stratificational grammar, although belonging to the functional group in terms of stated aims, belongs to the "axiomatic definitional" class in terms of its formal properties. I have recently argued (Schnitzer 1978) infavor of stratificational grammar as a possible framework for neurolinguistic research since it seems well equiped to model the relationship between static knowledge and dynamic performance in representing hierarchical linguistic structure. Since stratificational grammar has relations only--no items, no processes--one can conceive of knowledge as the static network as always represented, and of performance as the framework put into use with impulses running

through it. But as such, stratificational grammar provides only a framework. As is true of any theory of language, research needs to be done with respect to the analysis of specific aspects of specific languages. Perhaps ideas gleaned from some of the approaches just mentioned could be incorporated directly into a relational network format of stratificational grammar.

My arguments in Schnitzer 1978 in favor of the adoption of a stratificational model were made in the context of a structural realist approach to the mind-body problem. If it turns out that the competence-performance distinction,as I have therein defined it in neuropsychological terms, is viable, then we must concern ourselves not only with processing, not only with computation as it were, but also with <u>knowledge</u>. The appearance in a given aphasic of the same linguistic errors across radically different linguistic tasks in all modalities would indicate a deficit not in computation, but in knowledge. Some evidence that such phenomena occur is reviewed in Schnitzer (1978).

In any case, in doing language description, I think it important to take the cue from Jason Brown in the passage that I cited at the outset. "Symptoms should . . . be the mortar of the psychology", that is to say, of the <u>linguistic</u> theory. Various versions of stratificational grammar have been proposed which consist of various numbers of strata. Typically, a stratificational grammar has the following strata:

1) <u>Hypersememic</u>- (or <u>Cognitive</u>)-- This is, strictly speaking, an extralinguistic stratum which is intended to represent cognitive structure with which the linguistic system interacts.
2) <u>Sememic</u>-- This would include basic predicational relations, logical form, deep cases, focus, topic, etc.
3) <u>Lexemic</u>-- This stratum deals with basic word order in different types of clausal structure.

4) Morphemic-- This level handles inflectional morphology, productive derivational morphology, and part of morphophonemics.

5) Phonemic-- This would probably include syllable, cluster, and segment structure, contrast, certain alternations, as well as phonological feature composition.

6) Phonetic-- This level would handle nondistinctive phonetic specifications. Like the hypersememic, this stratum may possibly be extralinguistic.

¶ Each stratum in a stratificational grammar contains a realizational system connecting it with the stratum above and the stratum below; and a tactic pattern--that is to say, a specification of the combinatorial possibilities of the units of that stratum. This is one of the key features of the stratificational framework. On the other hand, the particular list of strata which I have presented is meant only to be illustrative. There is considerable diversity in the number and composition of strata among practitioners of SG. See Makkai and Lockwood (1973) for representative papers.

Following Brown's admonition, let us now consider a hypothetical example of two typical Broca's aphasics--one an English speaker and one a Spanish speaker. One would expect most of the agrammatism produced by the English speaker to involve syntactic deviation, whereas most of that produced by the Spanish speaker would involve morphological deviation. For example, if the English speaker said "go" or "you go" or "would go" when a normal speaker would have said "you would go" this would appear to be agrammatism of a syntactic type. But if the Spanish speaker said, for example "yendo" instead of "irías", this would be considered to be agrammatism of a morphological type. But this is only because in 'you would go', the four morphemes are represented by three lexemes, whereas in 'irías', the three morphemes ir ('go'), ía ('conditional'), and s ('second person') are represented by a single lexeme.

If symptomatology is to be the mortar of our theorizing, then this phenomenon provides motivation for collapsing the lexemic with the morphemic stratum. Not to do so would lead to the absurd conclusion that since lesions in Broca's area affect mainly syntax among English speakers and mainly morphology among Spanish speakers, there must be a physical difference between anglophones and hispanophones.[2]

Yet quite clearly, English syntactic relations and corresponding Spanish morphological patterns are performing the same kind of "housekeeping" functions of which I sopke earlier. Looked at functionally, one could say that the loss of housekeeping rules would have to involve principally syntactic problems for English speakers and principally morphological problems for Spanish speakers. This indicates that the lexemic and morphemic should be combined. I shall henceforth refer to it as the "morpho-lexemic level" and to "housekeeping" operations as "morpho-lexo-tactics".

In his remarks to the Milwaukee Conference on Current Approaches to Syntax, Bill Sullivan (1979) mentioned that Henry Gleason had in fact already suggested that lexotactics might simply be the upper portion of the morphotactics, rather than part of a separate stratum. When I asked for clarification, Sullivan gave me ~~a response~~ which included the following:

> he didn't say that LT and MT could be collapsed. He just said that he hadn't seen sufficient convincing evidence for the establishment of two strata. (Personally, I see both tactic and realizational evidence. The former involves idioms like kick the bucket, which are lexemic, and understand, which are morphemic. The latter involves the time-tense system of English. Other languages I know well also have both kinds of evidence. As I see it, the question is what constitutes necessary and sufficient evidence. That is, there is some, but is it enough?... .) (Sullivan, persnal communication, 1979)

I think that using pathology as the mortar of the linguistics can help deal with questions of this kind.

Mary-Louise Kean (1978) has recently come to a similar conclusion regarding the importance of a multilingual approach to evaluating aphasic symptomatology. However, she claims that what Broca's aphasia affects is not morphosyntax, but rather phonology. She says:

> A Broca's aphasic tends to reduce the structure of a sentence to the minimal string of elements which can be lexically construed as phonological words in his language. (p. 88)

But she does not say why or how. And in the model she implicitly adopts, "phonology" contains much that other approaches have included in morphology, morphophonemics, superficial syntax, and the lexicon.

If, on the other hand, we adopt a hierarchical relational-network approach to neurolinguistic modeling, such as I have been arguing for, we can claim that Broca's area contains the morpho-lexemic stratum and that Broca's aphasia damages the interconnections of the tactic pattern at that level. Thus, in speaking, well-formed semantic units (perhaps coming from Wernicke's area) are passed through Broca's area without being subjected to a properly functioning tactic pattern as they are passed along for speech or other mesotic encoding. This, I am claiming is the source of agrammatism in Broca's aphasia. I am claiming that Broca's area contains the morpho-lexemic stratum and that Wernicke's area contains something like what has been called the sememic stratum.

Competence and Performance

I have been speaking about both competence and performance. The Hayekian (cf. Hayek 1952) I have implicitly adopted in connection with a stratificational network framework allows one to look at input processing, output processing, and linguistic knowledge as all related by a constant structure which can be looked at in both a physical and a mental representation. One can look at potential connections in a system, or imagine the system in operation with impulses traveling through it. This applies to the human mind-body in general and to the linguistic system in particular. We can call the static aspect 'competence' andthe dynamic one'performance', I think, without changing the traditional sense of the words. The main difference is that by adopting structural realism, we can look at a given structure under two representations--one mental and one physical-- with the same static-dynamic distinction under both (cf. Hayek, 1952; Schnitzer 1978).

I can now say a word or two about decoding. I have argued that Wernicke's area deals with basic semantic functions: logical form, deep case structure, etc. When speech is heard, impulses, undoubtedly already somewhat analyzed subcortically, arrive at Heschl's gyrus, adjacent to Wernicke's area. Wernicke's area, with direct connections to the lexicon (which may be diffusely localized) interprets the message in general terms and allows a basic understanding. Usually no more linguistic processing is necessary. If it is necessary, that is, if interpretation depends crucially on morpho-lexemic analysis, then the partially decoded form can be sent to Broca's area for morpho-lexotactic analysis.

This proposal thus explains in a straight-forward way why Broca's aphasics have good comprehension and why Wernicke's aphasics have poor comprehension. It also explains why, notwithstanding, Broca's aphasics make mistakes on receptive tasks which are analogous to the errors made in their speech.

The fact that Wernicke's aphasics also have problems with reading comprehension may be due to the fact that if what is perceived is linguistic, the impulse winds up in Heschl's gyrus anyway. This needs to be investigated by electronic monitoring techniques.

I can also now address the question of why the approach I am advocating would entail that although a Broca's aphasic will not recognize agrammatisms when they are presented to him, he should, ceteris paribus, recognize phonemic paraphasias. Since Broca's area handles morpholexemics and not phonology, one would expect someone with a damaged Broca's area to have difficulty evaluating agrammatisms but little difficulty evaluating phonetic deviations, even though he may not be able to correct them. My claim needs to be tested, of course, by presenting Broca's aphasics with phonemic paraphasias and asking them to identify them. Such a task ought to distinguish between Kean's approach to Broca's aphasia and mine, since she claims that Broca's aphasia is a phonological deficit. I assume she would predict that they would not be able to recognize phonemic paraphasias, but it is difficult to say since her discussion does not deal with how the system might operate.

Lateralization

There is some interesting non-lesion evidence which lends support to the position for which I have been arguing. Lassen (1979), using radioisotope xenon-133 injected into the internal carotid artery, found a thirty percent local increase in blood flow in the following regions under the following conditions:

1) In listening to speech
    (a) the superior-posterior temporal lobe,
and (b) intermittently, the inferior frontal region.
2) In automatic speech (i.e., counting from 1 to 20 repeatedly)
    (a) the superior-posterior temporal lobe,
    (b) the primary mouth area in the central region,
and (c) the supplementary motor area in the superior frontal lobe (Penfield's superior speech area).
3) In "fluent normal speech"
    all of the areas in which the increased blood flow was found in automatic speech, plus the lower frontal area.

I have suggested that in decoding, Broca's area would not normally be used, since generally the message is understood without recourse to precise morpho-lexotactic analysis, but that when it is necessary, Broca's area is used for such analysis. This is consistent with with Lassen's observation that in listening, the inferior frontal region shows only "inconstant" hyperactivity.[3]

In speaking, if Broca's area's contribution is morpholexotactic, then it is no surprise that no hyperactivity is found in this region when counting to twenty repeatedly. On the other hand, this area was found to demonstrate hyperactivty during "fluent normal speech", as would be predicted by my position.

Also note that the posterior-superior temporal region is an area of hyperactivity in all of these language tasks. This is also consistent with my approach.

There is another interesting datum reported by Lassen, namely that all of these changes in regional blood flow are observed bilaterally. Recently, Albert and Obler (1978) have claimed that there is a significant right-hemisphere linguistic participation among bilinguals. They base their claim primarily on the abnormally high percentage of aphasia cases due to right hemisphere lesions in dextral bilinguals, and on the absence of the predicted right visual field effect in tachistoscopic tests of verbal material in several studies involving bilinguals.

They claim that the right hemisphere is always used in language acquisition at any age, and that as knowledge of a language is perfected, its representation becomes more and more left lateralized. This lateralization occurs with much greater facility prior to puberty. Hence, the only examples of complete left lateralization of more than one language may perhaps be balanced bilinguals who acquired both languages in childhood.

In a personal communication, Lassen informed me that all of his subjects were Danes who knew another language besides Danish. Except for one monolingual. Unfortunately, Lassen did not gather any right hemisphere data from that subject.

Before closing, I would like to cite two other studies in support of Albert and Obler's thesis. One is Curtiss' 1977 study of first language acquisition in a post pubescent individual, in which dichotic listening, tachistoscopic, and cortical evoked potential studies indicate the use of the right hemisphere for language. The question of course remains as to whether Genie's language dominance will ever begin to shift to the left. The other study, Pettit and Noll (1979) involves some dichotic tests which were given to some aphasics over a period of two months. The authors found that over this period the aphasics' performance on general language tests improved, and that their performance on the dichotic tests showed a _left_ ear advantage that increased over the the two-month interval. Right ear scores (inferior to begin with) did not improve during the the two months. The authors take these data as evidence for a dominance shift hypothesis: dominance shifts to the right hemisphere in aphasia. But if Albert and Obler are correct, these data may indicate instead that the aphasic is re-_learning_ a language and hence, the right hemisphere participation. In view of the data from Curtiss and from Lassen, as well as the studies noted by Albert and Obler, this alternative certainly cannot be ruled out.

Conclusion

In closing, I should like to re-emphasize that the approach I have been advocating is not reductionist. I am not saying that linguistic structures can be reduced to neurophysiological ones. What I am saying is that the

neurophysiological system that represents what we mentalistically and phenomenally call knowledge and behavior, competence and performance, must in some sense have the same structure as the system of language which it represents. I have stated elsewhere that this is the only way which seems sensible to me of dealing with the mind-body problem in so far as language and the brain are concerned.

Nontheless, the specific proposal I have made here today does not require the adoption of structural realism. It is undoubtedly compatible with a dualistic approach to the question of language and the brain, and in view of the growing amount of serious research in the field of parapsychology yielding positive results, it no longer seems possible to automatically rule out a two-substance theory. See Randall (1977) for a cogent review of the literature.

But there is one major problem for dualism: if the mind and the body are of two distinct substances which interact in some way as yet unknown, then why is it that characteristic brain lesions produce characteristic cognitive deficits?

Notes

[1] See Schnitzer (1976) for a discussion of the notion "mesotic".

[2] Sixteen years ago, Vildomec (1963) noted that one would expect to find different manifestations of agrammatism in analytic and synthetic languages.

[3] Lassen does point out, however, that since this region overlays the basal ganglia, one cannot decide if the area of increased blood flow is cortical or subcortical.

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ALFONSO CARAMAZZA: COMMENTS ON SCHNITZER PAPER

The goal of neurolinguistics is to explain psycholinguistic observations in terms of neurological mechanisms. We still lack an adequate vocabulary for this task. Moreover, we have problems extrapolating from the aphasic brain to the normal brain. But let us see what we can learn from the data we already have about aphasia.

The pattern of language dissolution in adult aphasics is not random. We want to explain this pattern in terms of damage to localized processing components. But to what extent can damage dissociate components? And to wat extent can such dissociation let us localize components in brain regions?

Schnitzer localizes morpholexotactics in Broca's area and a semantic/lexical component in Wernicke's area. But is such a linguistic description in any sense an explanation? There are two aspects to Schnitzer's claim about Wernicke's area.

1. Sememic processes are absent in Wernicke's aphasics.
2. Sememic processes are conducted in Wernicke's area.

But (1) does not imply (2), the Strict Localization Hypothesis.

Schnitzer assumes that Broca's and Wernicke's aphasia are well-defined syndromes. But, in fact, there is a huge range of symptoms, all too seldom defined in precise psycholinguistic terms. The localization associated with these syndromes in general can thus be quite vague, but this does not yield a precise description of the individual patient. We have to add precise descriptions of many symptoms beyond those used by Schnitzer (cf. Zurif and Caramazza). Broca's aphasics show poor articulation, dysprosody and agrammatic reading. It is only by paying attention to these symptoms that we can begin to truly model the processes underlying performance, rather than conflating performance with competence as Schnitzer does in his stratificational model.

Further progress in neurolinguistics will need new data of the right kind, culled from experiments based on improved modules combining linguistic and neurological insights.

DISCUSSION OF SCHNITZER PAPER

Kean: I believe that stratificational grammar is incomparable with the framework I use, and so do not see the use of any type of comparative test.

Caramazza: You seem to suggest your model is not testable!

Kean: No, no! I believe that the choice of linguistic theory will affect how one addresses the aphasic data, and thus determine what data are relevant and one's approach to neurolinguistics.

Zurif: I have data . . .

Lecours: My impression is that phonological paraphasia is phonologically governed, so that deficits in Kean's "phonological

component" would lead to phonological paraphasia.

Arbib: It strikes me that a stratificational grammar is like the system Bill Woods describes, but without the control structure. Wood stressed the need for control to avoid getting swamped by alternative interpretations of the speech signal. If Schnitzer would address these control issues, we might see the emergence of a very nice synthesis of two different approaches.

Schnitzer: My approach is based on a concern with closing the gap expressed by the mind-body problem. I do not suggest that my approach to Broca's and Wernicke's aphasias is as far as we can go, but it does provide an initial bridge which can help the process of solving the mind-body problem.

Kean: Agrammatism looks different in different languages. The direct translation of agrammatic output in one language does not yield agrammatic structure in another, generally. The search for a structural description that lays bare what is common to agrammatism across languages provides an important new form of data on language universals.

Brown: Agrammatism in Russian is said to occur with posterior lesions, perhaps because of dependence on noun declensions.

Lavorel: I have studied 10 cases of French agrammatism. It seems to appear 6 weeks to 2 years after the onset of the phonological disruption in Broca's aphasia in Francophones. French agrammatics say "sa auto" rather than "son auto", i.e., they use the correct gender rather than the supplanting phonological rule. They say "son ami" [#s~ #ami#] rather than the elision "sonami" [#sonami#]; and they cannot use "savant anglais" (English scientist) in distinction from the elided form "savantanglais" (learned Englishman) to distinguish meaning. Thus agrammatism can often be secondary to phonological problems.

Lecours: The French view agrammatism as an evolving symptom, and only consider it present when the patient has many words in his speech repertoire.

Brown: Brain lesions tease apart psychological processes along natural lines. This is true for the frontal speech disorders we have been discussing. These disorders are a mixed bag of qualitative states -- it's dangerous to lump them together and make composite maps of lesion areas.

Wood: It seems dangerous to assume that lesions necessarily break behavior down along psychologically meaningful lines. Recall Richard Gregory's "The brain as an engineering problem" -- just because a radio howls when a resistor is removed does not mean that the resistor is a "howl inhibition center"!

Brown: Well, we couldn't be further apart. Personally, I have little question but that the pattern of symptom change is orderly and predictable, with close links to normal function. Perhaps this only goes to show that the brain is not a problem that will be solved by engineers!

Marc Schnitzer: A Response

In this paper I will respond to some comments made on the papr I presented at the November meeting, as well as extend that material along the lines of some of the issues raised. But first I would like to discuss an issue which I consider to be of fundamental importance for neurolinguistics, the relevance of which I assumed (I think mistakenly) everyone who was at the meeting would have realized.

1. The Mind-Body Problem.

Much to their credit, the organizers of these conferences invited participants from several diverse fileds. Nonetheless, they neglected to invite a participant from from one extremely relevant field---that of philosophy. There are today philosophers of mind who are acutely aware of developments in neuropsychology (such as Woodfield and Demmett) and are knowledgeable in neuroanatomy and neurophysiology as well. The reason that a philosophical perspective is needed is because the basic question of neurolinguistics---'How is language represented in the human brain?'--- is not merely a "reverse engineering problem".[1] If we could treat the human body as a machine and take it apart and test the various parts and subsystems in any way we wished, and then put them back together, repeatedly, without damaging the organism, this alone would not suffice to answer this question. If the system of human language were a physical system (only), theoretically, a team of well-trained bioengineers, working under the ideal conditions stated, would eventually be able to solve the problem. But this is even theoretically impossible. Language is inherently a mental phenomenon. Phonemes, morphemes, noun phrases, clauses, linguistic meanings, in themselves could not be found anywhere in the brain. So even if we solved the reverse engineering problem, we would still not understand how language was represented in the brain.

To deal with this problem, we must deal with the mind-body problem in some way. In my November paper and in Schnitzer (1973), I propose the approach of "structural realism", suggested by Hayek (1952), in which he claims that the gap between the mental and physical orders be bridged by assuming that they have the same structure. A structure is an abstraction which can be instantiated in various media. The complex of lines and dots written by Beethoven on a piece of paper when he composed the Eroica Symphony has the same structure as the piece when performed by the Cleveland Symphony. Similarly, human language can be represented in speech, in writing, in manual signs, in Morse code, etc. Grammatical (including phonological and semantic) structure is involved in these representations of language. This structure can be represented in a written grammar of a linguist (perhaps someday). It is represented in the human mind: we know the structure of our language, as is evidenced in the multitude of tasks in which we make use of it. Is is also represented in the brain"; to deny this would be to deny the possibility of doing neurolinguistics at all.

What I have been claiming is that at the appropriate level of analysis, the structure of language as represented in the brain must be the same structure as our (mental) knowledge of the language. The problem remains as to what the appropriate level of analysis is. David Caplan, in his concluding remarks, said that he thought it dangerous to assume that if the brain is a certain way, then language must also be that way (or vice versa). David Levine expreseed similar reservations over lunch, also noting that, at the cortical level, there was no reason to believe the brain to be hierarchical; hence neuroanatomical hierarchy could not be used as evidence for stratificational grammar.

These comments miss the point, I think, that the level of analysis at which the mental grammar and the grammar in the brain must have the same structure is certainly not the level of neuroanatomy, nor of box-diagram neurophysiology. The hierarchical structure of language in no way needs to reflect the evolutionary

hierarchy of the development of the brain. But at the appropriate level of analysis of brain function, I claim that the two hierarchies are the same.

During one of the breaks, I mentioned to David Caplan that I could not conceive of a reasonable alternative to my approach. He mentioned that at one time it was believed that pain came about via mediation of special "pain cells". I asked him what made the cells cause pain. He replied "their very nature". But this does not deal with the problem. To say that we feel pain because of special pain cells ignores the issue of what makes the physical happenings represent what we mean when say we feel pain. That is, what about what is going on makes it be the painful sensation?

Daniel Dennett (1978) deals quite elegantly with this problem in a paper entitled "Why You Can't Make a Computer that Feels Pain". The issues are extremely complex, and I refer the reader to that paper. Suffice it to say that the mind-body problem is involved here, and ignoring it will not get us any closer to understanding pain.

Hayek (1952) discusses color perception. He calims that our phenomenal perception of color exactly parrallels the physical mechanism of selective sensitivity to different wavelengths of light. His structural-realist solution is to say that the distinctions we make among different hues ('hue' being a phenomenal term) are just those distinctions made among wave lengths by the physical sensory and interpretive apparatus. The difference between the two orders (phenomenal and physical, i.e. neurological) is essentially between two vocabularies. I wish to argue the same point for language: the difference between the linguistic description of language and the neurophysiological one will turn out to be a difference in vocabulary used to describe two orders which have the same structure.

This is not a logical point: the relation between the two orders might be otherwise. I cannot think of another plausible solution. But, to paraphrase George Lakoff, lack of imagination does not consitute evidence. Hence, I present this as a challenge to you all to suggest a plausible alternative.

I do nonetheless think that there is evidence for the position I hold. My position depends on there being such a thing as (linguistic) knowledge. If what we think of as knowledge is a mere artifact of various tasks that we perform, then there is no reason to assume that the linguistic grammar per se is encoded neurologically. But there is evidence that we do have linguistic knowledge. Those studies which have found error-type constancy across widely diverse types of linguistic-performance tasks argue for an underlying linguistic competence or knowledge which is brought to bear in performing the various tasks. Several of these are cited in my November paper and in Schnitzer (1978). The stratificational approach which I have been advocating can be viewed statically as representing the inherent knowledge, or dynamically as operating in performing linguistic tasks of various kinds.

Michael Arbib, in his reply to my commentary on "Neurolinguistics must be Computational" said that he thought that I was wrong in viewing knowledge as necessarily task independent. He says "to say that some KSs may be envolved in a variety of tasks is not to deny that each task may involve a specific set of KSs" (Arbib 1979, 166). Aside from the philosophical point that, as I understand it, what we mean by 'knowledge' (in the sense of knowing that, as opposed to knowing how) does entail task independency, since Arbib's KSs are computational modules, Arbib's approach would entail that we have knowledge only when we are computing something, that knowledge exists only when put to use. One of the advantages of stratificational representations is that they can be viewed statically as knowledge structures or dynamically as computational mechanisms.

It is possible that knowledge in the sense I am discussing does not exist. But the preliminary evidence from pathology supports my position. Much more evidence is needed since, if my view of knowledge is correct, characteristic errors should appear in patients with linguistic pathology which affects the knowledge system under any linguistic task, not just under certain "varieties of tasks".

Before I leave this point, I wish to reply to some comments made privately in which it was supposed that stratificational relational networks were intended to represent neuronal relationships directly. This is certainly not the case. The logical relations represented in a network diagram could be realized by any number of possible neuronal-level models, including one such as presented by Chris Wood. Stratificational grammar uses as primes only the notions of conjunction, disjunction, precedence and "upward and downwardness". Certainly these notions are ones used outside of language, and any microlevel neuro-psychological theory would have to provide a way of dealing with these notions anyway.

2. The Problem of Control.

At the November meeting, Michael Arbib stated that he thought that the stratificational network diagram that I presented was similar to the system presented by William Woods, but without control. I would now like to argue that control is built into the stratificational network.

Woods' system contains independent components of feature extraction, lexical retrieval, matching, bookkeeping, syntax, and semantics, all interrelated only by another independent component called 'control'. One of the goals of this system is to deal with the indeterminacy of the signal in speech perception.

I believe, however, that the relational-network approach (stratificational grammar) handles this problem by virtue of the inherent <u>interrelatedness</u> (i.e. non-independence) of the components. Let us consider some examples from English:

i. If a particular segment of running speech were indeterminate as to voicing, as for example /s/ vs /z/ in the nonsense sequence [$^{s}_{z}$pow], the phonotactics would immediately assign the sequence to /spow/ rather than to /zpow/, since /#zp/ violates English phonotactics.

ii. If a similar situation occurred with respect to /k/ and /g/ in the sequence [hiywəzlUkIŋæt⁻də$\left\{\begin{smallmatrix}k\\g\end{smallmatrix}\right\}$ayt⁻], the relational network would assign the sequence to /kayt/ since no lexeme would be found with a phonological realization /gayt/.

iiia. If in the course of a conversation the following sequence occurred (in which the question mark indicates an indeterminate segment):

[dəsæ:ndwIčəzondəbiyč ə ? dra:y],

the morpholexemic stratum would determine that the segment in question must be either /r/ or /z/, depending on whether the intended message had been "The sandwiches on the beach are dry" or "The sand which is on the beach is dry".

iii.b. It would be [illegible] the sememic level, which deals with topics, which would determine whether the segment in question was /r/ or /z/, depending on whether the topic of conversation had been sand or sandwiches.

iv. The cognitive stratum would be required to determine between /r/ and /z/, if there indeed existed phonetic indeterminacy between these two, in the sequence [fla:yIŋple:ynz ə ? de:ynǰərəs], since, from a sememic standpoint, presumably in a given conversation, either <u>flying</u> or <u>planes</u> would be a plausible topic. Discourse block considerations would be needed to determine whether the intended sentence was "Flying planes are dangerous" or "Flying planes is dangerous".

Granted these examples are not the most plausible possible, but I think

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they serve to illustrate the way in which the interrelatedness of strata in relational networks can obviate the need for a special "control" component.

3. Relational Networks and Aphasia.

In his commentary, Alfonso Caramazza took me to task, correctly, for not dealing explicitly with the details of Broca's aphasia, and accused me, I think unjustly, of providing no more than another "competence" grammar. Privately he suggested that I provide more [illegible] examples of how specific cases of aphasia would be analyzed in terms of stratificational networks.

Zurif and Caramazza (1976) review some research they did involving relatedness judgments analyzed by hierarchical clustering, among (inter alii) Broca's aphasics and "mixed anterior aphasics". Among their findings are that aphasic patients' control of "functors" shows parallels in their speech and in metalinguistic judgments.. This is the kind of result which I take to provide evidence for the existence of a task-independent linguistic "competence", such as I discussed in my November paper and in Schnitzer (1978).

More importantly, they find that for Broca's aphasics "such control as is shown, seems determined more by the semantic force of a functor (if it has such force) than by a fully computed syntactic representation". This result is in accord with my claim that Broca's area is in charge of "housekeeping", of morpholexotactics. Those functors which are not truly functional, that is, which are merely required by the morpholexotactics of the language would be predicted to be more adversely affected than those which have realizational connexions to the sememic stratum. Consider thefollowing Spanish sentences:

1. Aquí tenemos a la señora Sánchez. (Here we have Mrs. Sanchez.)
2. Le dijo Pedro que Juan no iría. (Pedro said (to someone) that Juan would not go.)
3. Le dijo a Pedro que Juan no iría. ((Someone) said to Pedro that Juan would not go.)

In the first sentence the word <u>a</u> is required by Spanish morpholexemics because the direct object (la Sra. Sánchez) is human. It has no semantic function and would have no representation in the sememic stratum. On the other hand, the *a* in sentence #3 serves to mark *Pedro* as the indirect object and to distinguish sentence 3 from sentence 2 in which *Pedro* is the subject. My stratificational approach (and the results of Zurif and Caramazza's research) would thus predict that Broca's aphasics would have better control (both in speaking and in metalinguistic tasks) over the *a* in sentence 3 than over the *a* in sentence 1. The *a* of sentence 3 would have realizational connextions to the sememic stratum (which handles deep cases) and would hence be a *functional* functor (from a communicative standpoint (It functions to identify deep cases.)), whereas the *a* of sentence 1 would not.

Zurif and Caramazza find that Broca's aphasics have less difficulty with prepositions than with articles. This stands to reason, since in English, prepositions frequently mark deep cases, but articles have relatively little (and sometimes no) sememic value. The word *to* was found to be under better control when used in a prepositional phrase than when used to introduce a complement clause. In the latter case the *to* is required by English morpholexotactics, but has no sememic representation.

Goodglass (1968) found similar results with Broca's aphasics, noting the tendency to omit "the small words of grammar" and inflectional endings. To deal with these forms he creates the notion of "saliency", about which he says, "The intuitive definition that was suggested is that saliency be considered the psychological resultant of the stress, of the informational significance, of the phonological pronimence, and of the affective value of a word." Thus Broca's aphacs perform better (i.e. less agrammatically) when the functors have "salience". As far as informational significance and affective value

are concerned, the relational-network approach would predict better performance (in all sorts of tasks) with informationally significant functors (since these have sememic realizations) aw well as those with affective value (since these would presumably have cognitive realizations).

With respect to Goodglass' other criteria for salience, "stress" and "phonological prominence", it is important to note that when these were the only criteria for salience involved, performance was found to be enhanced on a *repetition* task. It is quite conceiveable that the phonological salience allowed the aphasic(s) to repeat correctly by using a strategy for repetition involving processing no higher than the phonemic stratum, bypassing morpholexemic and sememic processing. More research needs to be done to find out whether phonological salience alone is sufficient to improve the performance of Broca's aphasics with respect to non-sememically realized "functors' in tasks other than repetition.

4. Some Illustrations.

I would now like to illustrate how the notion of the stratificational network can be used in the analysis of aphasic errors. First I will briefly review an analysis of some dyslexia data of Whitaker and Keith reported in Schnitzer (1978). Essentially, what found was that when the patients were asked to read aloud sentences of the form '$\left\{\begin{matrix}\text{That}\\ \text{This}\end{matrix}\right\}$ is a [ADJ] [N]' (e.g. 'This is a general solution) that there was greater average latency between the article and noun-derived or verb-derived adjectives (such as *accidental* (noun-derived) or *reliable* (verb-derived)) than there was between the article and pure adjectives (such as *possible*). Figure 1 is a stratificational schematic of this relationship. It illustrates in a highly abbreviated form the relatively greater amount of structure involved in the derived adjectives than in the pure ones. Looking at this structure dynamically (with impulses traveling through it in real time by whatever neural mechanism turns out to be correct) we would expect a greater

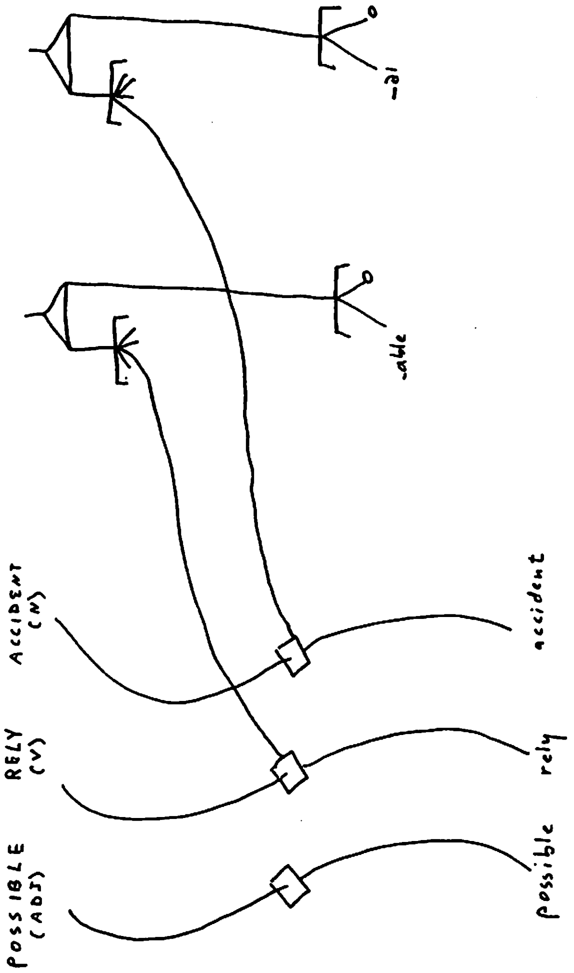

Fig. 1

latency in processing derived adjectives. This illustrates how a relational-network approach can deal with the competence-performance distinction: the relational network represents the always-present linguistic knowledge (competence). The activated network represents processing (performance) and allows for relative latency predictions.

I wish now to turn to some data from a moderately agrammatic Spanish-speaking aphasic. Spanish has a highly inflected verb system involving three stem classes and inflecting for person, number, tense, aspect, and mode. It is schematically represented in Figure 2. I have included only one complete example, in order to insure legibility, viz., the morpholexemic representation of the sentence 'Ellas hablaron' (They (feminine) spoke (preterite).). And the diagram ignores compound tenses completely. The aphasic had a marked tendency not to inflect verbs, but to substitute gerunds and infinitive forms for correctly inflected verb forms. Table 1 is a list of verbal forms which he used in a five-minute discussion of some photographs. I have been arguing that Broca's aphasia is a disruption of the morpholexemic stratum. Let us now look at the errors to see whether they suuport this position.

Of the seven incorrect verbal forms, three are instances of gerunds, and one of an infinitive which replaces an expected inflected form of the verb. In Spanish, gerunds are generally used adjectivally or adverbially and infinitives nominally (as in complement clauses). The gerund and infinitive forms do not participate fully in the verbal morpholexotactics, yet they carry the sememic value of the related verbs.

The other three errors can be viewed as errors at the level of morpholexotactics. The form /empyese/ could be looked at either as an error in mode (subjunctive where indicative is expected) or as an error with respect to

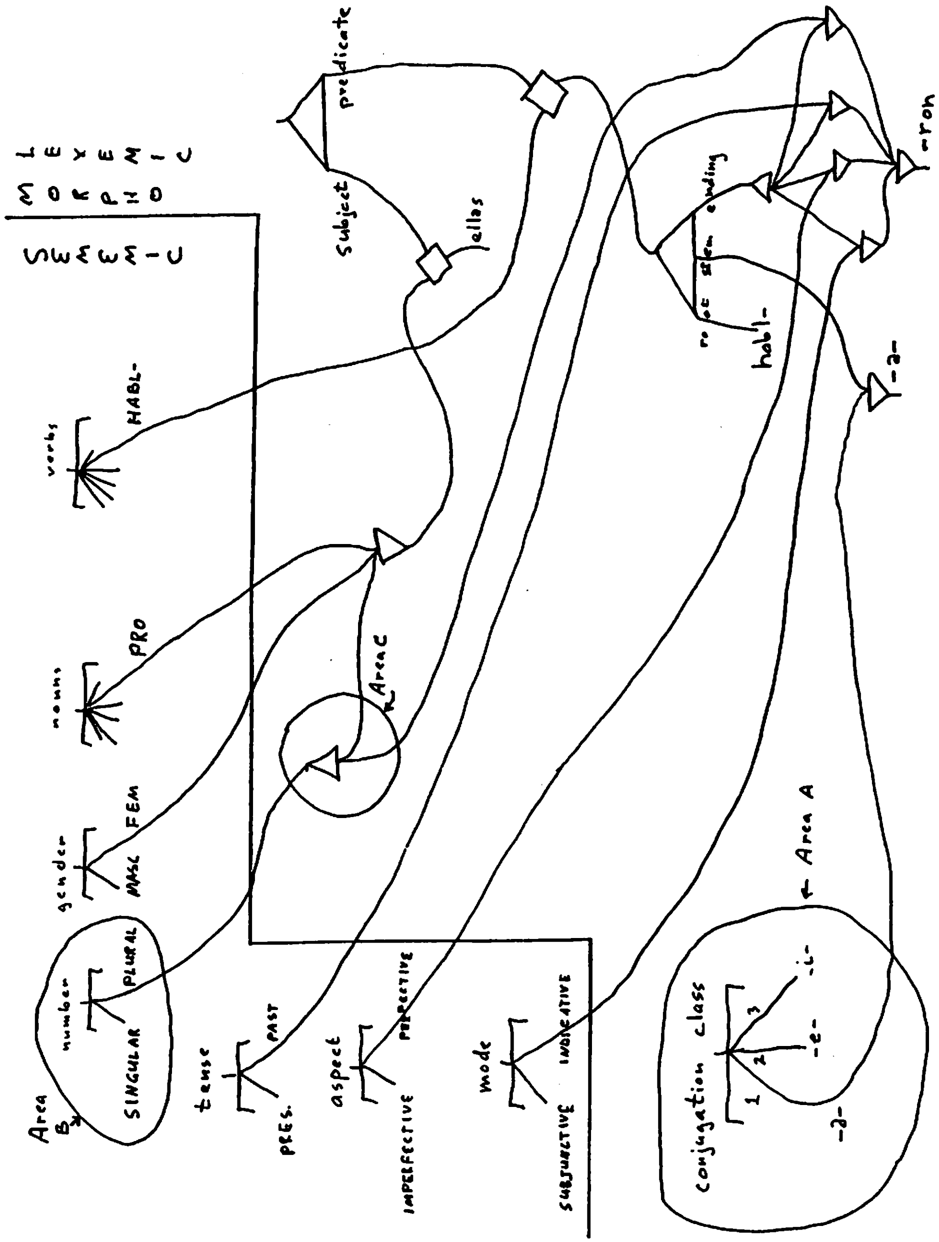

Fig. 2

Table 1

Verb Forms Used in an Agrammatic Discourse

Correct Forms Used	Incorrect Forms Used	
	Form Used	Should Have Been
está	preguntando	pregunta
empieza	/empyese/	empieza
hablar	encontrando	encuentra
escribir	escribiendo	escribe
está	/komprer/	comprar
será	encontrar	encuentra
fué	dice	dicen
pagar		
es		
parece		
pagarle		

conjugation class (second (e-stem) where first (a-stem) is expected). If it were a modal error, it would have ramificatons at the sememic level. But conjugation class (Figure 2 Area A) has no sememic realizations. This type of error would be purely morpholexotactic. The fact that one of the other remaining errors, **comprer* for *comprar*, can be explained in these same terms, but not in terms of modal error, makes the conjugation-class analysis more plausible.

This latter example is of a correctly used (but incorrectly formed) infinitive. Infinitives do not inflect for person, number, tense, mode, or aspect. They do each have a characteristic conjugation class and this example shows this class incorrectly represented. On other occasions, this patients made other errors of this type.

The last error, *dice* for *dicen*, is an error in number. Number obviously has sememic realizations. But the word was uttered in the context "Dos otros dice" (*Two others says*.). Clearly, the patient had the humber of the subject correct and was making an agreement mistake. In a stratificational network, the sememic concept of number would apply to the subject. The number *agreement* for the verb would be a purely morpholexotactic phenomenon. This distinction can be found in Figure 2 with area B representing the sememic singular-plural distinction and area C representing the morpholexemic agreement phenomenon.

It is important to note that all of the verbal errors can be accounted for at the morpholexemic level alone. Sememic patterns seem to be preserved. It is also important to note that the four errors which resulted in gerunds are counterevidence to Kean's theory of Broca's aphasia, since she says that "A Broca's aphasic tends to reduce the structure of a sentence to the minimal string of elements which can be lexically construed as phonological in his language." (Kean, 1978, 88)

and these gerund forms certainly do not qualify as minimal strings of elements which can be lexically construed as phonological in Spanish..

I would like to close with a discussion of some data which relate to the claim I made in my November paper to the effect that my model would predict that Broca's aphasics ought to be able to recognize phonemic paraphasias even if they could not correct them.. At the meeting, Edgar Zurif said that the fact that these patients keep trying to correct themselves in their speech provides some evidence in favor of this claim. I wish now to present some further evidence in the form of phonemically distorted words which were presented to a Broca's aphasic to be judged for correctness and corrected if wrong. Table 2 contains the presented distorted word, the correct form and what the patient said.

In the first example, the patient produced morphologically and semantically related words, but could not produce the correct form. This is true of example 6 as well. In example 5, the patient produced a word phonologically similar to the correct form, but unrelated semantically and morphologically. In example 3, the patient produced two phonemically similar words before getting the correct form. The patient succeeded in the task in examples 7 and 9, and failed entirely for examples 4 and 8.

These comments have been in reaction to numerous discussions, both "official" and informal, at the last meeting. Your comments on this material would be most welcome.

Table 2

Correction of Distorted Words

Item presented	Correct Form	Response
1. telesivor	televisor	T V, televisión
2. ustedas	ustedes	ustedes
3. maripesa	mariposa	mariguana, marimacho, mariposa
4. otupado	ocupado	no
5. sirvilleta	servilleta	cerveza
6. veniendo	viniendo	venir
7. bicicleca	bicicleta	bicicleta
8. /abániko/	/abaníko/	---
9. biccionario	diccionario	diccionario (after 2 minutes)

Note

[1] By an "engineering problem" I mean one in which a task needs to be performed, and one (the engineer) needs to devise a system which can accomplish the task. A "reverse engineering problem" is thus that of examining a system which is functioning to accomplish a certain task, in order to figure out how the system accomplishes that task.

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Neurolinguistics, 'Coding', and the Interpretation of Models

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1.0: These notes arise from my long-standing conviction that, in the sciences at least, metaphors must be taken absolutely literally if they are to be at all interesting and useful (Marshall, 1977; Marshall and Fryer, 1978).

1.1: Somewhere (I have repressed the reference) Karl Pribam tells the following story: Pribam was lecturing about the 'holographic theory' of memory. After the lecture a girl in the class stood up and asked 'But Professor Pribam, why doesn't the laser beam burn a hole through the brain?' Pribam gives the impression of considering the girl a poor student.

COMMENT: In my opinion, the girl was right, Pribam was wrong.

1.2: Another example: Democritus of Abdera (460-360 B.C.E), the laughing psychologist, taught the 'siege-engine theory' of perception. Objects throw off images or replicas of themselves which upon entering or being caught by the sense organs are the (quasi-) proximal causes of our perceptions (Lewes, 1867; Beare, 1906). To which Theophrastus (372-288 B.C.E.), eventually, replied: 'Suppose that you and I are standing facing each other, looking at each other. Why don't our images crash into each other and fall to the floor in a broken heap?' (Stratton, 1917).

COMMENT: Theophrastus was right, Democritus was wrong.

1.3: Jorge Luis Borges (1951) writes: "Perhaps universal history is the history of the diverse intonation of a few metaphors."

COMMENT: Yes, but precisely because we often refuse to take them at face-value.

1.4: John Hughlings Jackson (1888) writes: "I have nothing whatever to say of the nature of the relation between two utterly different and yet concomitant things, cerebration and mentation, to one another. As an evolutionist I am not concerned with this question, and for medical purposes I do not care about it."

COMMENT: Well, alright .... but can we really get away with such agnosticism?

1.5: Patrick Wall (1974) writes:

> " Behaviour, including mental behaviour, must operate in the context of the body. At a minimum, some medium must exist which allows communication between the periphery of the body and the shape of mental processes. What is the nature of this connecting matrix? Textbooks of physiology and psychology answer the question by a confidence trick. They place chapters on peripheral nerves, sensory mechanisms, perception, and cognition next to each other in sequence, leaving the impression that the chapters are connected by something other than the binding of the book. Unfortunately we cannot postpone taking a position on what are the likely connections between body and mind because the design of even the simplest experiments for following nerve impulses within the brain assumes some hypothesis about the working of the brain."

COMMENT: Yes, yes, and yes again.

2.0: As far as I know there is only one mode of theorizing about disorders of higher cognitive function that has ever had the slightest heuristic and predictive value. The mode in question is that devised by the diagram makers:

2.1: When diagrams of this type were made famous by Wernicke (1874), they were accorded a rigourously 'realist' interpretation. The very subtitle of Wernicke's monograph - "A Psychological Study on an Anatomical Basis" - indicates that this is so.

2.2: Wernicke (1874) was quite unsatisfied by the work of Baginsky (1871) in which the symptomatology of the aphasias was explicated by means of a diagram that is not given a strictly anatomical interpretation:

> " There is a significant difference between the invention of various theoretic centers (coordination center, concept center) - with complete neglect of their anatomic substrates, because the unknown functions of the brain up to the present have not warranted anatomically-based conclusions - and an attempt, based on an exhaustive study of brain anatomy and the commonly-recognized laws of experimental psychology, to translate such anatomic findings into psychological data, seeking in this way to formulate a theory by use of the same kind of material."

Lichtheim (1885) is equally committed to a straightforward, literal, anatomic interpretation:

> " Our task is to determine the connections and localization of the paths of innervation subservient to language and its correlated functions. On the supposition of our having reached this end, we should then be able to determine the exact place of any solution for its symptomatic manifestations with the same precision as we do for those of a motor or sensory paralysis depending on a lesion of the peripheral nerves."

2.3: Within such models a 'real' lesion (demonstrable anatomical or physiological damage to the real brain of a real person) is interpreted as a 'lesion' in the hypothetical model. These latter 'lesions' can be of two sorts - destruction of or damage to a 'centre' (⊗), or destruction of or damage to a 'tract' ($\overset{x}{\rightarrow}$) that 'connects' two or more centres, or that connects a centre to the periphery (that is, the body).

3.0: A little historical digression. From the 17th century onwards the hypothetical anatomy and the hypothetical psychology developed together 'hand in glove.' Anatomically, the diagrams were supposed to reflect the 'fact' that in the nervous system,

long fibres conduct between the body and the brain; short fibres compute an internal representation; long fibres within the brain 'link' two internal representations.

Psychologically, the diagrams were supposed to reflect the 'fact' that the mind looks like this:

$S \rightarrow (s) \rightarrow (r) \rightarrow R$

3.1: Wernicke (1874) is quite explicit about the fact that he is taking over Meynert's notion of 'association tracts' which are the cortico-cortical analogues of the 'projection tracts' (efferent and afferent) that connect the brain with its body (Meynert, 1867).

3.2: This whole three centuries of work on 'higher cognitive functions' can be thought of as an attempt to shoot the piano-player of Johannes Muller (Marshall, 1977; Fryer and Marshall, 1979; Fryer, 1978); or, equivalently as an attempt to 'ablate' the pineal gland (compare Descartes, 1662, with La Mettrie, 1748). The assassination was ultimately successful (see Freud, 1891, or anything written by Hughlings Jackson, e.g. 1875).

3.3: The murder of the soul was, however, totally irrelevant to the real scientific issues of how to interpret diagrams. To put the point more generally, what is the ontological status of any model that uses the notational conventions of the diagram-makers? What is the price that one pays for any successes that such models may have in describing or indeed predicting the consequences of cerebral lesions?

3.4: John Hughlings Jackson (1875) writes:

> " For clinical purposes it matters nothing whether we believe (1) that conscious states are parallel with active states of nerve fibres and cells, the nature of the association being unknown, or (2) that mental states and nervous states are the very same thing, or (3) whether we believe that there is a soul acting through a mere mechanism. I wish to insist that to hold any one of these beliefs does not one whit justify us in omitting anatomy. Betwixt our morphology of the nervous system and our psychology there must be an anatomy and a physiology. Morphology has to do with cells and fibres or with masses of them. Anatomy has to do with sensori-motor processes."

3.5: But Jackson was fully prepared to localize symptoms; he was also prepared to use the 'centre and connection' terminology: "So then, to speak of nervous 'centres for ideas,' of 'centres for memory of words,' etc., is to use not anatomical, but a mixture of morphological and psychological terms. Such statements are perfectly true, no one denies that the higher centres are for mental states; the statements are, nowadays, mere truisms" Jackson (1875). Jackson also actually (literally and physically) drew diagrams (Jackson, 1894).

3.6: And Kurt Goldstein was also prepared to use the basic terminology of the diagram-makers, and never seriously disagreed with their localizations of symptoms (see Geschwind, 1964).

3.7: What the hell is all the fuss about then?

3.8: Geschwind (1974) has suggested that we should stop fussing and just quietly get on with describing symptoms and the responsible lesions.

We might even manage to improve our diagrams. In short, "Le concept de disconnexion (est) l'histoire d'une idée banale mais importante" (Geschwind, 1974).

4.0: But this is the place to start, not the place to stop. How should diagrams be interpreted? Specifically: (1) Is it really the case that centres and tracts are anatomically distinct in the way that 'centres' and 'tracts' are? Are they really in different places? (2) Is it really the case that destruction (total or partial) of nerve cells and fibres corresponds with destruction of 'centres' and 'tracts'? And if so, how?

4.1: What if, with Eric Lenneberg, one refused to countenance the claim that the brain is "a collection of more or less independent apparatus connected to one another by cables" (Lenneberg, 1973)?

4.2: What if, with many of the classical diagram-makers, one was willing to jettison the notion of punctate localization? Bastian (1898) defines the idea of a sensory centre as

> " ..... a set of structurally related cell and fibre mechanisms in the cortex whose activity is associated with one or with another of the several kinds of sensory endowment. Such diffuse but functionally unified nervous networks may differ altogether from the common conception of a neatly defined 'centre' and yet for the sake of brevity it is convenient to retain this word."

What consequences for the interpretation of diagrams would follow from this notion of 'diffuse but functionally united nervous networks'?

4.3: Friedrich Eduard Beneke (1833) argues:

> " As to the phrenologists, they really ought to see into what palpable contradictions they fall with the strange mental powers which they invent, such as 'philoprogenitiveness', or 'love of unity','combatativeness','secretiveness', 'acquisitiveness', 'love of praise', etc. Our psychical forms may, without being changed, form part of groups and series of the most diverse kinds; and by so doing they become interwoven and crossed one with another, so as to form a kind of variegated web. From this it unquestionably follows that one and the same form may take place in philogenitiveness, in combatativeness, acquisitiveness, etc.; for everything is capable of becoming a constituent part of that with which it fits, and by which it is required."

I have heard similar remarks made about nerve cells. If they were true, would traditional diagrams be interpretable?

4.4: Von Monakow (1914) writes:

> " The generally accepted theory according to which aphasia, agnosia, apraxia, etc., are due to destruction of narrowly circumscribed appropriate 'praxia, gnosia, and phasia centres' must be finally discarded on the basis of more recent clinical and anatomical studies.

> It is just in the case of these focal symptoms that the concept of complicated dynamic disorders in the whole cortex becomes indispensible. Brutal local injury causes not only associative diaschisis (vicinity symptoms) but also commissural diaschisis, which affects points in both hemispheres depending on the course followed by the disrupted fibres."

In that case, how come traditional diagrams do such a good job? Or do they? Does Wood (1978) provide the beginning of an answer to the first question?

4.5: Kurt Goldstein (1948) writes:

> " The question of the relationship between the symptom complex and a definitely localized lesion again became a problem, no longer, however, in the form: where is a definite function or symptom localized? but: how does a definite lesion modify the function of the brain so that a definite symptom comes to the fore?

5.0: What is a diagram? A diagram is a (systematic and intentional) category-mistake. It contains things that look like physical objects, circles and arrows, ~~and are~~ frequently interpreted as sets of nerve cells and fibres; and it also contains 'things' that are clearly 'mental' - the words written inside the circles. These words include 'acoustic memory image', 'semantic representation' and worse.

5.1: The lines transmit 'information' in 'code'. What information in what code?

5.2: "We have known for some time that the anterior commissure connects visual association cortexes. Pandya has recently shown that there are also areas of auditory association cortex connected by the anterior commissure. It thus seems possible that even in cases of total agenesis of the corpus callosum the large anterior commissure would be capable of transmitting verbal information between the hemispheres" (Geschwind, 1975).

What does 'verbal information' mean in this quotation?

5.3: Berlucchi (1975) reflects similar problems:

> " According to the simplest assumption, all of the above mentioned visual functions of the corpus callosum may be based on one and the same anatomo-physiological mechanism: the transmission of basic visual information between the cerebral hemispheres. In other words, the corpus callosum would ensure interhemispheric communication of visual information at an early stage of information processing, and the information exchanged would then be differentially elaborated within the hemispheres for the attainment of various functional goals: binocular stereopsis, visuomotor control, formation of memory traces and so on. Alternatively, the exchange of visual information between the hemispheres via the corpus callosum may occur at many different stages of

information processing, the information transmitted at any one stage being utilized for one specific function. For example, binocular stereopsis in the midsagittal plane might make use of relatively undigested visual information exchanged by the corpus callosum, whereas the callosal transfer of visual training might require the communication of more elaborate information."

5.4: The legend to Figure 1 of Geschwind (1975) reads: "Af = Arcuate fasciculus transmitting verbal commands from Wernicke's area to premotor region....."

How?

5.5: Geschwind (1975), as usual, is exceptionally clear concerning the value of diagrams:

" I have no disagreement with the statement that everything cannot be explained by means of disconnection. I have in fact made this point repeatedly and I repeated it in the introductory lecture of this conference. Let me list here again some of the features of pure alexia without agraphia that present difficulties in interpretation. These 'difficulties' in fact illustrate the value of a connectionist analysis. Such an analysis forces us to a precise definition of mechanisms. It therefore permits us to know when something is not explained by the postulated mechanisms.

Thus the standard explanation for the reading difficulty of pure alexia without agraphia is that the patient can see only in the right hemisphere but cannot transmit the seen words to the left hemisphere because of the lesion in the splenium. We are, however, all aware of the fact that such patients can typically name objects quite well and in many instances can read numbers very well. We might advance two theories to account for this. We could on the one hand assume that the right hemisphere could name objects and numbers, i.e. that it was not necessary to transmit these categories of visual information to the left hemisphere. We know that in general, however, this explanation is not the correct one since a patient with a complete lesion of the corpus callosum and of the anterior commissure shows poor performance in reading and naming objects and reading numbers in the left visual field.

We are thus forced to assume another hypothesis, i.e., that information concerning objects or numbers seen in the left visual field can be transmitted to the left hemisphere over different pathways from those which must be used for the transmission of written material. We have now learned an important lesson, namely, that the nervous system handles seen numbers and objects different from seen words. Since written numbers and written words consists of arbitrary symbols this means that the differences in the methods of dealing with numbers and words are not innate, but must be based on differences in the way these categories are learned. The disconnection analysis has thus led us to an important psychological conclusion, that we might not have deduced on the basis of a purely psychological analysis. As we discover repeatedly the nervous system has its own classifications that differ from the ones we might deduce on purely logical grounds."

5.6: Gazzaniga (1970) also worries about the 'code':

" The answer to that question would give evidence as to the amount of processing each side of the cortex does on the discrimination task, i.e., whether the callosal transmission is some sort of 'Go, no-go' message or an elaborate read-out of raw visual information."

6.0: Why are diagrams so useful? Because you can always add another box or another arrow.

6.1: Newcombe and Ratcliff (1975) comment: "However, this approach - if carried further - would allow one to postulate multiple disconnections of specific pathways to account for the consequences of any cerebral lesion, making the concept of disconnection so flexible as to deprive it of its explanatory power."

7.0: How can we constrain diagrams so that they really do become theories not just summaries of the data?

7.1: The legend to the block diagram of Figure 1 in Crowder and Morton (1969) reads: : "No reference whatever to locations or pathways in the nervous system is implied."

QUESTION: Implied by the author or implied by the diagram?

7.2: And later, Morton (1979) adopts an explicitly 'fictionalist' interpretation. The status of the model is described as follows:

" Its relationship to actual neural and chemical activity in the brain is obscure, but in a sense irrelevant, inasmuch as if the model accounts for data and generates further understanding, it fulfils its purpose as a psychological model. In any case, the functions described by the model could be equally well implemented by a number of structures and if any of these functions turn out to be necessary constructs it will be someone else's responsibility to find the neural substrate."

COMMENT: Realistic? Disingenuous?

7.3: ".... the three sensory association areas are important in the recognition of familiar configurations experienced through their respective modalities; the motor association areas are important in the organization of purposeful movements. We do not know what the nature of the code is, but, it appears to be such that an entire association zone participates in each process, much as every part of a holographic plate contains information for the reconstruction of an entire picture" (Goodglass and Geschwind, 1976).

QUESTION: What evidence points to that conclusion?

7.4: "Perception corresponds to the activity of a small selection from the very numerous high-level neurons, each of which corresponds to a pattern of external events of the order of complexity of the events symbolized by a word .... it is as if, at high levels, the size of the alphabet available for representing a sensory message was enormously increased. Perhaps it would be better to say that, if the activity of a low-level neuron is like the occurrence of a letter, that of a high-level neuron is like the occurrence of a word - a meaningful combination of letters" (Barlow, 1972).

COMMENT: You pays your money, you takes your choice.

7.5: "Reductionist attempts to ascribe perceptual or cognitive functions to the activity of single cells or localized anatomical regions are both experimentally and logically untenable" (John and Schwartz, 1978).

7.6: What about the monkey's paw (Gross, Rocha-Miranda, and Bender, 1972)?

7.7: "Physico-chemical means of expression are common to all natural phenomena and remain mingled, pell-mell, like the letters of the alphabet in a box, till a force goes to fetch them, to express the most varied thoughts and mechanisms" (Claude Bernard, 1865).

7.8: "For the nervous centres do not represent muscles, but very complex movements in each of which many muscles serve. In each of the two centres discharged the very same muscles are represented in two different orders of movements. In one there are represented movements in which the arm leads and the leg is subordinate; in the other, movements in which the leg leads and the arm is subordinate. The very same notes are made up into two different tunes; in chemical metaphor, the fits are isomeric" (Jackson, 1875).

7.9: "The elementary signs of language are only twenty-six letters, and yet what wonderfully varied meanings can we express and communicate by their combination! Consider, in comparison with this, the enormous number of elementary signs with which the machinery of sight is provided. We may take the number of fibres in the optic nerves as two hundred and fifty thousand. Each of these is capable of innumerable different degrees of sensation of one, two, or three primary colours. It follows that it is possible to construct an immeasurably greater number of combinations here than with the few letters which build up our words. Nor must we forget the extremely rapid changes of which the images of sight are capable. No wonder, then, if our senses speak to us in language which can express far more delicate distinctions and richer varieties than can be conveyed by words" (Helmholtz, 1868).

8.0: "The nervous system stands between consciousness and the assumed external world, as an interpreter who can talk with his fingers stands between a hidden speaker and a man who is stone deaf - and Realism is equivalent to a belief on the part of the deaf man, that the speaker must also be talking with his fingers" (Huxley, 1874).

OSCAR MARIN: COMMENTS ON MARSHALL PAPER

In the course of evolution, a number of systems dealing with relatively simple aspects of the environment had to become integrated and cooperate within each organism. This has some crazy effects -- bones and membranes both depend on calcium, and the perathyroid cannot optimize both at the same time. The brain has strange characteristics, too.

The brain has the task of ensuring survival, even if there are lesions, and it is thus a redundant system. In vision, a locative system (the tectum) served as basis for the evolution of a recognition system (visual cortex), and now we cannot tease the two apart.

The overlap of subsets of the brain imposes a certain commonality of code at the interface. But individual neurons are cells with their own local survival criteria, and know nothing about action in the world or of language. It is only at the transducers, the sensory and motor interfaces, that the neural code correlates directly with the external world. Subsequent transductions involve suprising and complex feature extractions. The basic neural code is homogeneous, but the brain is heterogeneous. Near the periphery, or where we have somatotopy, we may hope to find well-circumscribed processes. From there on, there is a tremendous overlap and convergence.

The "speech machine" has a sensory and a motor end. Many talk of Broca's area as the motor end, but all that is constant

in descriptions of Broca's aphasia is the articulatory deficit. In Wernicke's aphasia, only verbal deafness gives the common ground. It is time to leave Broca and Wernicke, these great men, alone and stop using their names in such inconstant ways.

We need structural diagrams of what elements are present, which peripheral and which central. I believe grammar and the lexicon will not be localized but that the "articulatory machine" might be. Unfortunately, symptoms of one stage me hide the next stage in processing. If area B needs area A for its expression, then seeing symptoms of damage to A makes it impossible to see the further effects of damage to B. A 30% reduction of oxygen uptake in the motor strip yields complete hemiplegen -- so we can never tell what the symptoms of a 100% reduction would be.

Each of us is, to at least some minor extent, anomic, agrammatic and amnestic. [Lecours: We are paragrammatic, not agrammatic!] We all forget a word or jumble a sentence from time to time. How then do we characterize the lesioned patient? By frequency of deficit, complexity of task, or evolution of symptoms? There is no easy correlation between lesion, brain structure, and symptomatclogy.

The only hope I see for neurolinguistics is that each of us -- neurologist, linguist, computer scientist -- learn to respect each other's diagrams! Each specialist must spell out the minimal rules of his game. Then we must try to synthesize these rules to develop a common language.

DISCUSSION OF MARSHALL PAPER

Marslen-Wilson: Names of people enter the language in such expressions as "She *boycotted* the ceremony", "They *bowdlerized* the novels", "He *leninized* the party-line", etc. But what about: "He *marshalled* the data"?

Whitaker: Your discussion of the patient who names a knife as a plate and then acts as if it were a plate may be explained by saying that it is the incorrectly recalled name that directs further responses. This is the standard textbook description of the syndrome of the posterior cerebral artery.

Kertesz: The situation in associative agnosia could be due to semantic confusion, or because the patient learns new strategies when the visual system is damaged based on knowing what objects may be presented, or their semantic caegories, as well as perseveration.

Marin: There can be strange effects between language perception. I had one patient who was not agnostic but was anomic. He could do complex perceptual tests like the elephant-assembly problem so long as we stopped the linguistic machinery from interfering by having him repeat nonsense or count backwards. But when he was asked to describe the task, he called the trunk a leg and moved the trunk piece to the leg position!

Marshall: I had a deep dyslexic patient read out a printed word while doing forced choice pointing to one of a set of eight

pictures. Given the word 'goat', he pointed corectly while saying 'sheep'; given the word 'grave' he pointed to the picture of a coffin while saying 'grave'. So we can't say that the name has simply taken over from the object perception.

Levine: We have seen a patient (Levine, D.N. and Calvanio, R. Visual discrimination after lesion of the posterior corpus callosum. Neurology 30: 21-30, 1980) with isolated damage to the splenium and no damage to the hemispheres. She was alexic in her left visual fields but not in her right. The nature of her deficit in the left visual fields could not be described well with simple terms such as "visual defect" or "verbal defect". When a group of letters was exposed tachistoscopically to her left visual fields she was not only unable to name them but also unable to draw them or to point them out on a choice card. So, even when overt naming was not required, the deficit was apparent. Moreover, she was unable to name one of a group of three letters, tachistoschopically exposed, when the examiner told her in advance which one she should name (e.g. "the left one"). However, if a letter was presented in isolation, without other letters nearby, she could name it. This adverse influence of adjacent letters would stamp the deficit as "visual". Yet other visual tests, such as kinetic perimetry, flicker perimetry, and discrimination of nonsense shapes showed no differences between the right and left visual fields.

The deficit in this patient's left visual field was specific both with regard to the task employed and the stimulus materials used. Such sweeping terms as "perceptual" or "semantic" were too coarse to specify the problem. We need lots more data on performance in a variety of tasks in order to develop a proper vocabulary.

Arbib: It would seem that the word "neurolinguistics" enshrines a category mistake for Marshall, combining as it does the physical term "neuro" with the mental term "linguistics". Yet for most of us it presents a challenge. People speak and understand; damage to their brains impairs this linguistic ability. We seek a framework in which to comprehend these phenomena. To dismiss such an effort as a category mistake is itself a mistake.

In describing the brain, we should use the most precise language available. We maystart with a crude lable of "visual information", and we may always precede such a mentalistic term by "a pulse train representation of". But neurophysiological research does allow us to become more precise. For example, Hubel and Wiesel combined microelectrode technology and questions about edges of moving objects to initiate a body of research which no longer restricts us to a rough overall description of a brain region, but lets us speak of how depth, orientation, movement, etc., are represented by the spatiotemporal pattern of activity played out over a population of neurons. As brain theory develops, I expect many cells to be described not by correlation with activity at the periphery, but rather by their role within some computational theory of (a limited aspect of)

brain function.

A final remark about Marshall's "category mistake". John Szentágothai and I coauthored "Conceptual Models of Neural Organization". He is a neuroanatomist primarily concerned with function. After chapters presenting a functional overview and a structural overview, we provided two examples of structure-function integration. What is noteworthy here is that the first example, stereopsis, was functionally defined; the second, cerebellum, was structurally defined. The integration of structure and function is at the very heart of neuroscience.

Kertesz: Lichtheim's modification of Wernicke's model is the one most easily correlated with anatomy. The sensori-motor dichotomy is at the basis of our understanding of the brain; and Wernicke imposed language on top of it. This is still the best model we have.

We cannot localize function completely, but we can make much more detailed analysis of symptoms and of anatomy. My group has very precise and objective measurement on a scale from 0 to 200.

Caplan: Your scale is fine as a global measure; but many of us feel that the unbundled measures of multiple aspects of comprehension and production will prove more useful than any single global scale.

Marshall: I agree with Kertesz that the Wernicke model is the best we have, but it is only an outline, and needs to be further developed.

AMHERST, NOVEMBER 16, 1979 WORKING MANUSCRIPT

SIMULATION OF SPEECH PRODUCTION WITHOUT A COMPUTER

André Roch Lecours*

In the context of this paper, the word glossolalia should be taken to designate a fluent, euarthric, discourse-like production which is entirely or near entirely neologistic. Glossolalic utterances can thus be viewed, on the one hand, as essentially made of a succession of spoken entities which are word-like, although they are not there to be found in the dictionaries, and which are combined into sentence-like entities bearing no conventional messages that a qualified listener might decode. Glossolalic production can also be viewed and transcribed, on the other hand, as a succession of segments which are assimilable to conventionally pronounced phonemes and syllables.

As a rule, the first impression of one witnessing glossolalic behavior is that one is listening to a *bona fide* language that one does not know: an it-sounds-like reaction is typical. I might add that the contention of many a glossolalic speaker is that he or she is talking, when under the influence of ghosts, gods, devils, deads, computers, martians, russians and what not, one or several *bona fide* languages that he or she has never learned (and usually does not understand, anyway) : hence, the term xenoglossia often used among believers to designate glossolalic behavior. In this respect, my friend Jacques Mehler has told me about a South American fellow who managed to be convincing enough, when fluently speaking archaic Babylonian, to have his psychoanalyst indulge in the publication of an astonished — and astonishing — case report, with a whole set of appropriate freudian interpretations; likewise, Pierre Marie Lavorel might tell you, later on, about an English lady who has become, obviously without apprenticeship, proficient in spoken pharaonic Egyptian.

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To this day, I have studied twenty samples of tape recorded glossolalic discourse. They were produced by one schizophasic speaker, eight pentecostal charismatic believers, two healthy nurses, one poet and, given my definition of glossolalia, by two elderly Wernicke's aphasics. A broad I.P.A. transcription was made in all cases.

Now, provided a set of rules, phonemic and syllabic segmentation of glossolalic discourse is no more a source of difficulty — and sometimes it is appreciably easier — than phonemic and syllabic segmentation of samples of natural tongues one does not know. On the whole, segmentation in sentence-like entities is not a problem either, in view of relatively clear prosodical clues and longer pauses. On the other hand, and but for a proportion of strongly word-like entities, it is more difficult and sometimes impossible — if pertinent — to reach common agreement of several listeners as to word-like level segmentation, a fact you should keep in mind when pondering transcriptions of glossolalic discourse. This proportion of strongly word-like entities varies from one sample to another, depending mostly on pauses and prosodical clues (such as tonic accent), on recurrence of identical or similar strings, on amount of affix-like segments, and so forth.

Given these preliminaries, I will first tell you about eleven of the samples I have studied. They are those of the schizophasic, of five of the charismatics, and of the two nurses. All of these subjects are exclusive unilingual speakers of the French language, Quebec brand. On the other hand, many of them admit to an interest in passive exposure to spoken foreign languages, such as the exposure one gets at night when tuning one's radio along the short waves bands : I can understand this.

When "speaking in tongues", the schizophasic subject considers himself the reluctant but subdued phonatory instrument of malevolent wills from Mars, where he once worked as a crooner. It is thus that he can speak in four different tongues, to which he refers as his "temperaments" : one of these, he calls his "English temperament", another his "funny temperament", and the last two his "French temperaments" (the second apparently being an excrescence of the first). Examples of each are given in INSERT 1.

INSERT 1

SCHIZO-ENGLISH

[zaʀa ʀakuʀu bʀubjɛʀ paʀa bʀazjɛm nɛʀgɛs kaʀakuʀu bʀubjɛʀ mizœpʀiz aʀakaska ʀœkaʀœ bʀazjɛm nɛʀgɛs kɛʀakɔʀu bʀubje ʀamisi azumba bɛʀgɛs koʀo bʀubjɛʀ paʀa bʀazjɛm nɛʀgɛs kɛʀakuʀu bʀubjɛʀ mizœpʀiz aʀakaska ʀœkaʀœ bʀazjɛm nɛʀgɛs kɛʀakoʀo azumba bɛʀgɛs kuʀu bʀobjɛʀ paʀa]

SCHIZO-FUNNY

[apalagmala kakili bidi elak kalamala kakisi lipidi kala kakulu ili elak malakala kala kili agumla bala kakisi lipili kala kăkulu eli ilak manaka lakala paʀa kaza kaʀbisi eli biʀak kolo malakalk kili paʀa kalakolu agumla bala kakisi lipiʀi kala kakili ili elak]

SCHIZO-FRENCH 1

[la ʀœkɜ̃fjɔʀanis ʔtʀɔbeʀɔgad bʀakal dœ ʀœkɜ̃fjɛʀjanis ʔtʀœbeʀɔgad bʀakal dœ ʀœkɜ̃fjɔʀjɑsjɜ̃ mɑ̃bʀal yn bʀibɛʀgasjɜ̃ pineʀo peʀikal bʀabal dœ ʀœkɜ̃fjɛʀjanis ʔtʀøbeʀɔgad bʀabal la ʀœkɔmfjɔʀasjɜ̃ lastʀø bœnalfjʔ pineʀo peʀika bʀabal dœ ʀœkɔmfjœʀjanis ʔtʀøbeʀœgal bʀakal ʀjanik]

SCHIZO-FRENCH 2

[la kɜ̃tʀavizeʀjasjɜ̃ la manœʀapaʀa bʀakal dœ ʀœkɜ̃fjɔʀjanis ʔtʀøbeʀɔgad bʀakal dœ ʀœkɜ̃fjɔʀjasjɜ̃ bineʀomenal bʀakal dœ ʀœkɜ̃fjɔʀizi yn bʀibɛʀgasjɜ̃ pineʀomenal dœ paʀjetegʀal epineʀopeʀika bʀabal dœ ʀœkɜ̃fjɜ̃ʀjanis ʔtʀøbeʀɔgal lɑ̃ lœkoloʀjɔl de paʀjetegʀal epineʀopeʀikal]

The five charismatics, for their part, "speak in tongues" when under the infinitely benevolent influence of the Holy Ghost. They are obviously fond of doing it although, like the schizophasic, they consider themselves to be the mere phonatory instruments of a stronger will. Partial transcriptions of the Ghost's messages through these five are provided in INSERT 2.

Besides the linguistic aspects I will discuss in a moment, the schizophasic and the charismatics have other points in common : (A) all are fully aware of the unconventional nature of their glossolalic utterances; (B) all state that they themselves never decide on the moment of glossolalic behavior, this being the exclusive privilege of the stronger will, but, when recorded, all turned out to be capable of immediate production on demand; and (C), finally, all believe that their glossolalic utterances do belong with real languages, human or not, archaic or contemporary.

INSERT 2

CHARISMATIC #1

[o kiʀia ʀamatuʀeiʃ silia maʀamakoleiʃ sikolea ʀamataiʃ maʀanataiʃ ʃikolia ʀamataiʃ ʃikolia ʀapataiski o jaʃimdea ʀamatuʀaiʃ ʃekilia soli aʀmakulaiʃ o jaʃimdea ʀakoleiʃ silia ʀamataʀaiʃ o kulia ʀamataʀaiʃ o kulia ʀapataiski o jaʀa matoiʃ ʃekilia sulia ʀamakolaiʃ]

CHARISMATIC #2

[monoʃkolo kola monoʃkœli kynɔm monokwɔle kwɔmo kɔlekɔmwe aʃkulu kunœkɔmwa alaʃkɔla kɔʀaja manaʃkolo keʀeʔ manaʃkolo akʀi malaʃkɔlo keʀekɛ monokɛle kɛlokɔlo kɔnœkɛ ʀokœna mwɛlaʃkolo kyʀija akolo ʀamanaʃ koloka ʀakeja manakolo kuʀane kalakɔ mwanaka akulijam]

CHARISMATIC #3

[o kwena kana maʃe kana maʃina ina kwena ʃanana kanana o kwina kama naʃina naʃena ina kwena ʃimine nana o kwena kana maʃina ina swina kanama naʃina o kwina kama naja ina kwina nanaʃa o kwina kana maja ʃana ina kwena ma o kwina mo ina mina ina kwina o na mo]

CHARISMATIC #4

[putʃta jato amadea se atʃtu ʜoʀa o maʀia ʃtuja talasul e maʀja atunda asuja inʃtigoso jɛtʃteni o maʀja tuskundea deseu in dios kuna majʃte o njanatʃe maʀjana idonjaʃte koskena ɛ no njɔnɛskena o niʃtɛne maʀja tosɛ no no swoʃtɛnei ɛ no ɛʃeʀo swɔndinu udaʃse]

CHARISMATIC #5

[iljana sepwɛʀe kɛʀeʃte manante kaʀje siljana sapa litekol dɔna ʜajaʃoʀe dmanin sepeʀja saiti tuʀjokɔle ʜedu mɔʃtɔjo poj igmonois ʜjɔlœ keʀɛs umale nias ajekelena isaʀa janin tete keʀiʃtea tweʀija kaʀja sijo odnɛnse pwele kaʜɛntʃe aʀialijɔns eʀija fajinto kaja]

Somehow to my surprise, since I had myself tried without success, I found out that a certain number of ordinary * people — not everybody, and by far — are perfectly capable, without rehearsal or even knowledge of the very existence of the glossolalia phenomenon, of sustained glossolalic discourse in answer to an instruction such as "try to speak in a language you do not know". The two

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* I was not informed of John Marshall's talents when I wrote this.

nurses are among those. Obviously, they are aware of the unconventional nature of their behavior, they do not attribute it to possession, and they do not believe it to correspond to some real language. Examples of their neologistic productions are given in INSERT 3.

INSERT 3

NURSE #1

[i evistimi tanto elevɛnte bɛste vanto elevɛsti bika anevɛnti mitistan elevɛnti limi nistaʀe inivindi me dastɔnte elekɛsti kue tikanto eliminimista batɛnto elevanta tɛstamɛnto alavinto e anvekemistan elividimistan elibidimiʃtaj kede vete anto ivaj emindisti]

NURSE #2

[biema kumiku mitʃaou ka bulamiteul tʃibakuʀa bauti mi kaputi dʒabuti kamylabul kwitau tʃabu ʀinantu mi kabuti amokowti mi kulakutʃi taputu te moʀijɔʀ alamu ʃikolamuki mi lakulina tuʀi biku miʀɛtu batʃu mitɛltu tijau biʀkma taputa tima tʃiputitau amulaʃ]

The eleven samples produced by these subjects all occurred in the form of monologues, which might be of some importance. As far as I can tell, they represent imitations of human language production : the attempt at simulation is acknowledged in the cases of the two nurses and, unless I have missed something capital in ghostlore, it is obvious — although not fully conscious — in the cases of the schizophasic and of the charismatics. My own purpose is to tell you about the outputs in these simulation experiments; it will remain for you find out about the programs behind these outputs.

I will be brief concerning the semantics of these samples. Indeed, except for being a potential music-like tool for the communication of moods, glossolalic utterances have no immediately sharable semantic value. Essentially, therefore, it is the formal aspects of language production that glossolalic speakers simulate. This comprises segmental and suprasegmental parameters. Let us deal with the latter first.

It is my impression that prosody is of paramount signification in glossolalic behavior. Nonetheless, I will be brief on this topic also, mainly because I do not know how to tackle the problem. Suggestions are welcome. Meanwhile, I will formulate four comments : the first has to do with general melody, the second with speed of elocution, the third with regional accent, and the last with tonic accent.

Regarding general prosody, one is struck by the obviousness of a major melodical investment in each of the eleven samples. Indeed, one has the very definite impression that a glossolalic speaker's leading choice is that of a prosodical model. In the schizophasic, for instance, the model is apparently radio voice of America, or else radio Tirana, if you are differently enclined, and in the charismatics, it is either fervent Mediterranean prayer or professional imprecation.

A choice in speed of elocution can also be at stake : for instance, a charismatic imprecator can take as little as 7 seconds, and an implorator as long as 45 seconds, to utter 100 phonemes (as opposed to 13-18 seconds in controls). Last year, in Urbino, a theologian suggested that this might well be the difference between "bad" and "good" glossolalia : I am not quite sure what he was driving at, but he may have had a point there.

My third observation concerns regional accent which, as you may know, is nearly as perceptible in Quebec as, say, in South Texas. Well, regional accent totally disappears in seven of the eleven samples, and it is very much attenuated in the four others (those of the schizophasic). It is a fact of common observation that something of this sort can occur in people singing, acting, reciting or praying.

The last point is perhaps the most intriguing : but for two samples, those of the schizophasic's "French temperaments", and provided my segmentation in word-like segments is worth something, there occurs partial or complete replacement of the expected oxytone by a simplified paroxytone, with accentuation of the penultimate or of the ultimate syllable of word-like entities according as they end on a vowel or on a consonant.

I will now turn to segmental parameters of glossolalic simulation, about which I will be somewhat more explicit. For six of the samples, those of the schizophasic and of the nurses, who can glossolalize for hours in a row, my study in this respect is founded on the first 2000 phonemes of each corpus.

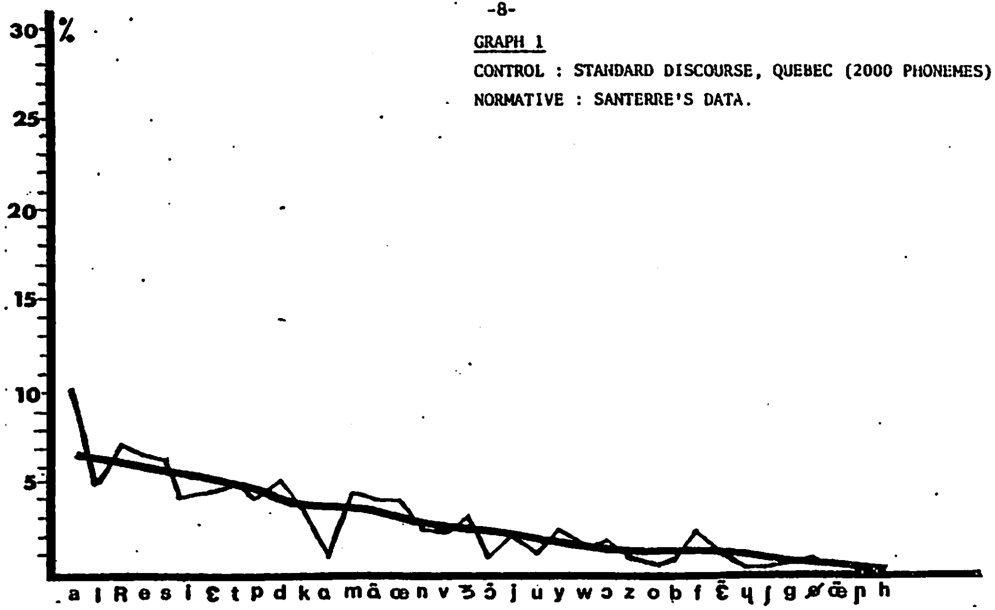
The Holy Ghost being less talkative than the Martians, the five charismatic samples average only 584 phonemes (442, 1240, 242, 532 and 463 phonemes respectively). I have compared these eleven samples to various normative data, on the one hand, and, on the other, to the first 2000 phonemes of various French control samples. The latter included the productions of two normal quidams telling about their work, of one non-glossolalic schizophasic (with occasional neologistic utterances), and of three non glossolalic fluent aphasics (one with Wernicke's aphasia proper and logorrheic neologistic jargon, one with conduction aphasia and the kind of discursive behavior which I but not Jason Brown would call "phonemic jargon", and the last with transcortical sensory aphasia and verbal jargon).

Let us begin with phonemic choices and frequencies in glossolalic as compared to control discourses. I have several comments on this :

☐ The first is that the distribution of the 2000 phonemes in each of the control samples, including the non-glossolalic schizophasic and the three non-glossolalic fluent aphasics, is rather close to that in normative data. This is shown in GRAPHS 1 to 6.

☐ The second is that from 99% to 100% of the phonemes, in each of the eleven samples, belong with the french phonemic inventory; occasional foreign-sounding units do appear in a few samples, for instance English-like [ʀ]s [l]s and [ʜ]s, which is hardly an anomaly in Quebec French, anyway.

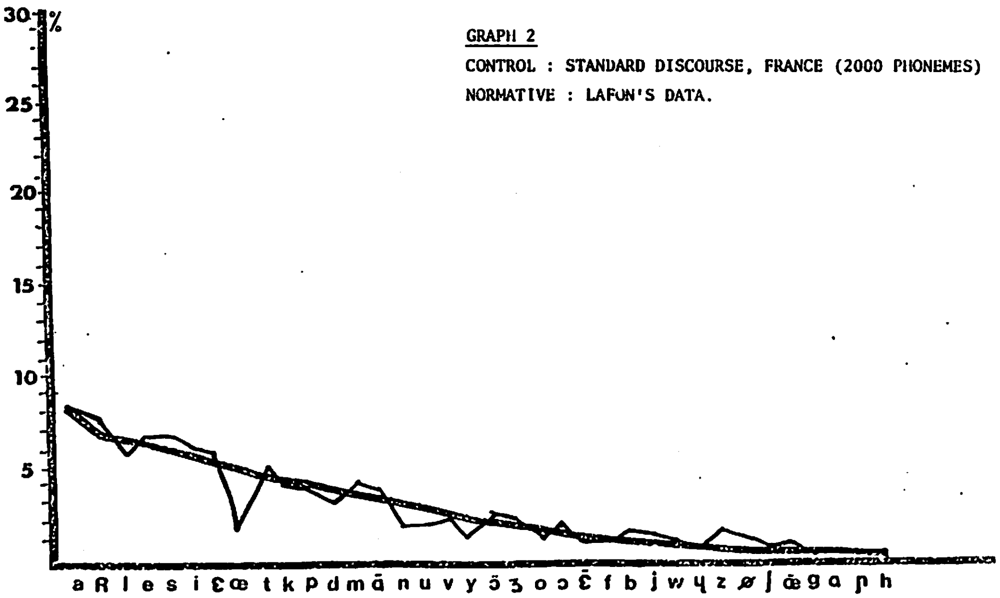
☐ My third comment has to do with the fact that the phonemic distribution in the eleven glossolalic samples is not at all like that in the six control samples. As compared to normative data, glossolalic samples are thus characterized by gross overuse of certain phonemes and, complementarily, by gross underuse or even absence of others. Moreover, frequency in mother tongue hardly influences frequency in glossolalic utterances, so that phonemes which are infrequent in French can be overused in glossolalic samples, and phonemes which are frequent in French can be underused or not used at all. A particular profile of phonemic distribution is thus observed in each of the eleven samples : this is examplified in GRAPHS 7 to 9. Numerical data are regrouped in TABLE 1, and a somewhat clumsy global representation is made in GRAPH 10, where the thin linear black area corresponds to normative data (Santerre and Lafon), the intermediate dark grey area to control data, and the wide lined area to glossolalic data.



GRAPH 1

CONTROL : STANDARD DISCOURSE, QUEBEC (2000 PHONEMES).

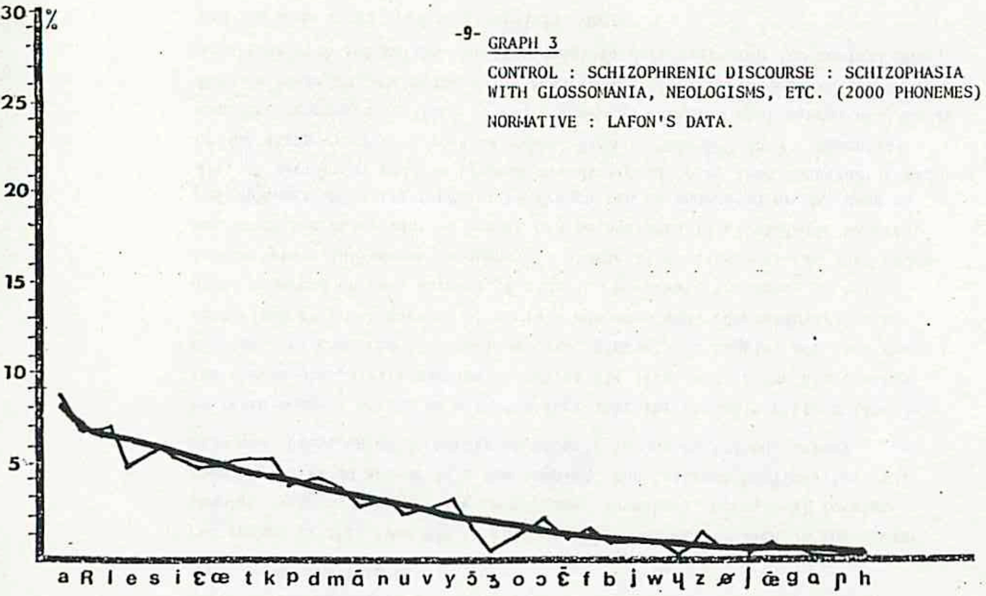
NORMATIVE : SANTERRE'S DATA.



GRAPH 2

CONTROL : STANDARD DISCOURSE, FRANCE (2000 PHONEMES)

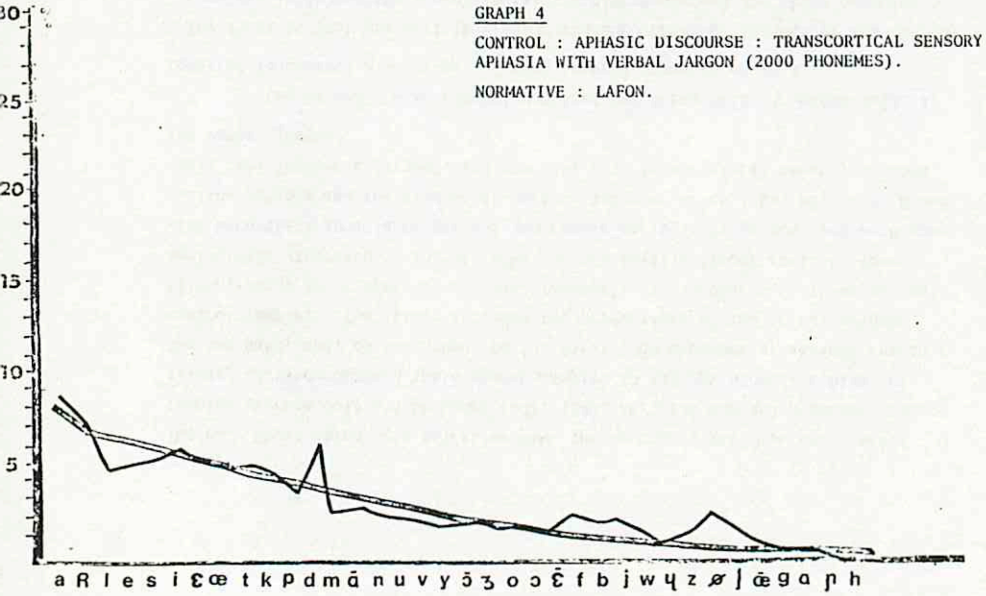
NORMATIVE : LAFON'S DATA.



GRAPH 3

CONTROL : SCHIZOPHRENIC DISCOURSE : SCHIZOPHASIA WITH GLOSSOMANIA, NEOLOGISMS, ETC. (2000 PHONEMES).

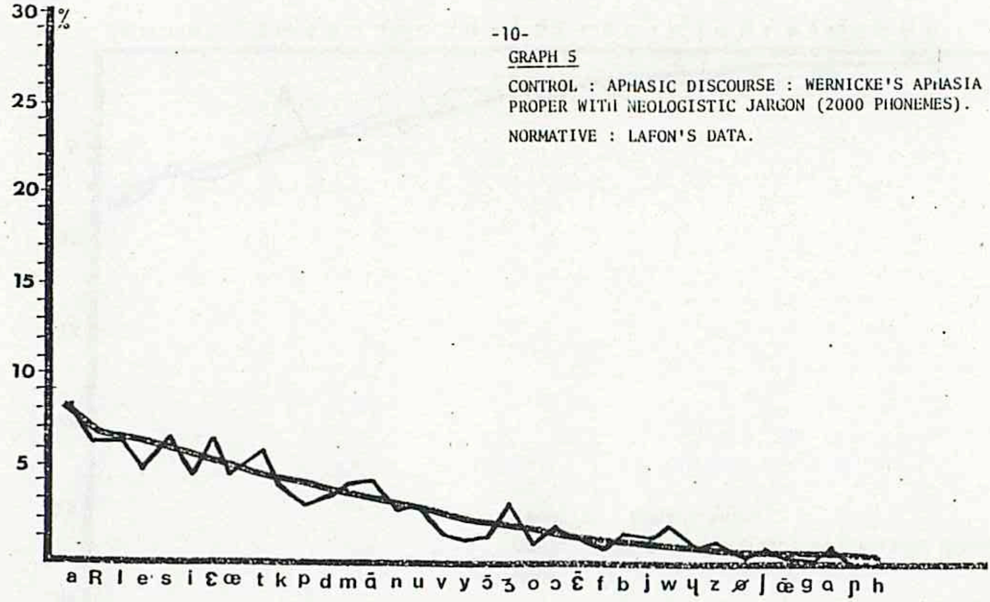
NORMATIVE : LAFON'S DATA.



GRAPH 4

CONTROL : APHASIC DISCOURSE : TRANSCORTICAL SENSORY APHASIA WITH VERBAL JARGON (2000 PHONEMES).

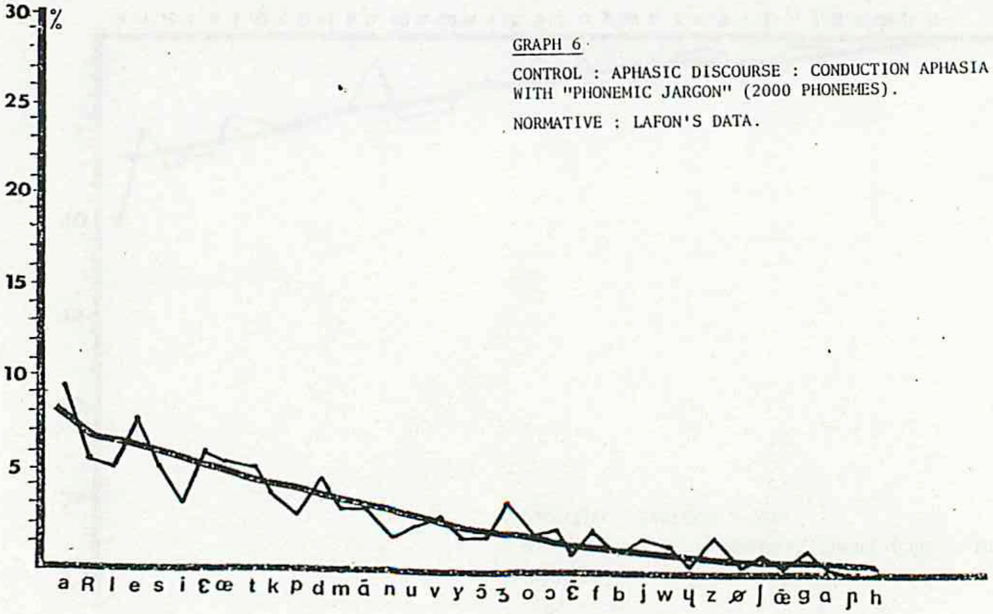
NORMATIVE : LAFON.



GRAPH 5

CONTROL : APHASIC DISCOURSE : WERNICKE'S APHASIA PROPER WITH NEOLOGISTIC JARGON (2000 PHONEMES).

NORMATIVE : LAFON'S DATA.



GRAPH 6

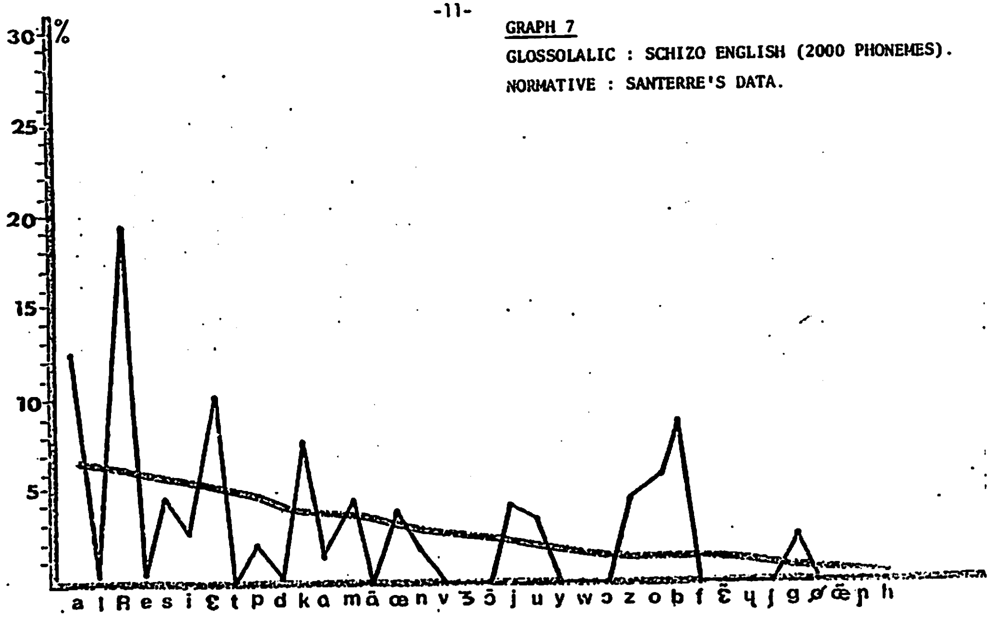
CONTROL : APHASIC DISCOURSE : CONDUCTION APHASIA WITH "PHONEMIC JARGON" (2000 PHONEMES).

NORMATIVE : LAFON'S DATA.

GRAPH 7

GLOSSOLALIC : SCHIZO ENGLISH (2000 PHONEMES).

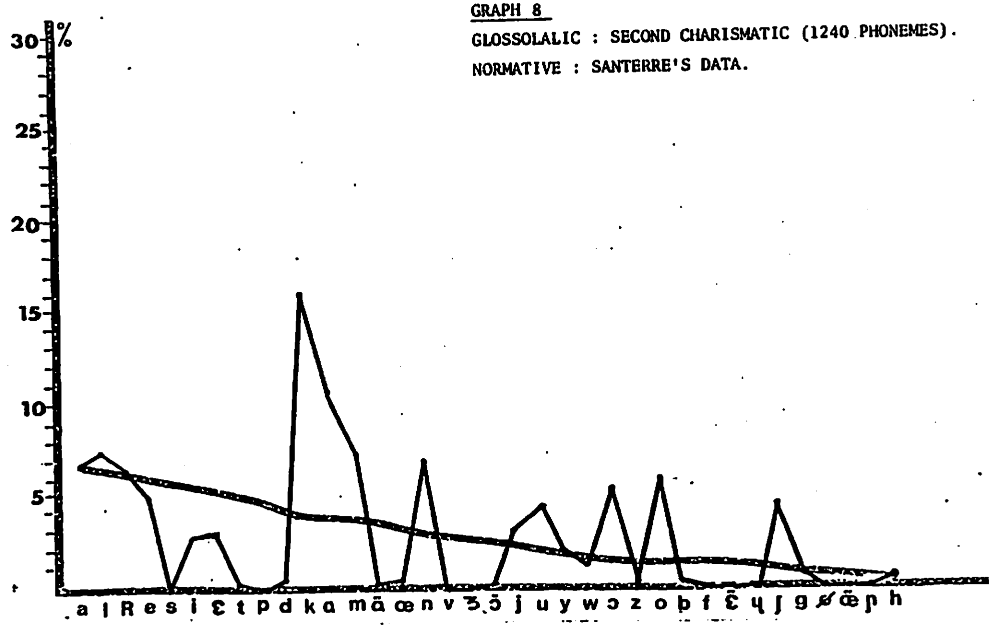
NORMATIVE : SANTERRE'S DATA.



GRAPH 8

GLOSSOLALIC : SECOND CHARISMATIC (1240 PHONEMES).

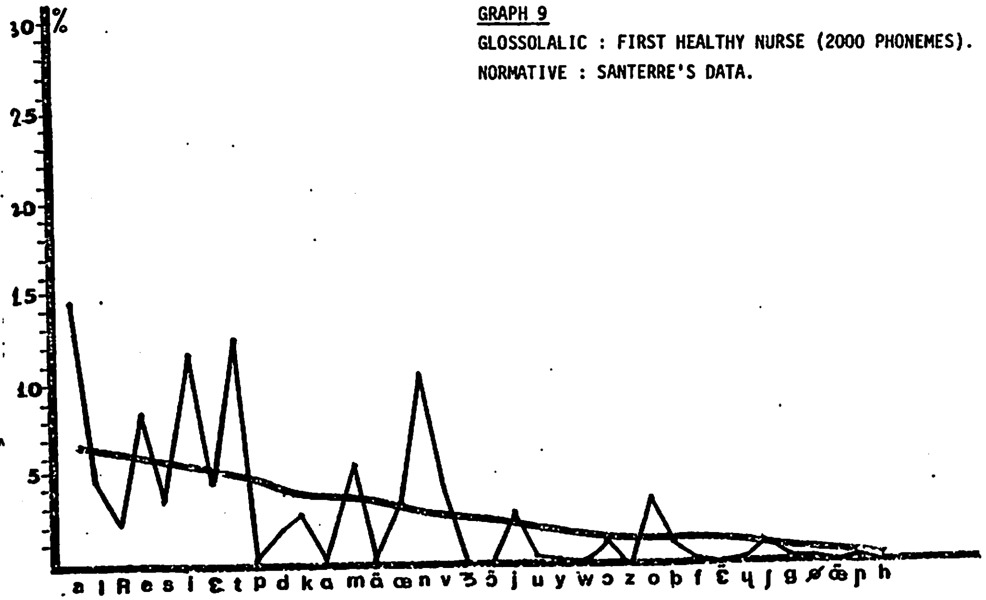
NORMATIVE : SANTERRE'S DATA.



GRAPH 9

GLOSSOLALIC : FIRST HEALTHY NURSE (2000 PHONEMES).

NORMATIVE : SANTERRE'S DATA.



Now, one who is a friend of mine and also an ingenious engineer*, suggested that another way of comparing the phonemic repartitions in my glossolalic and control samples to that in normative materials was to make some form of entropic measurements. He provided a Shannon trick in this respect, which reads as follows:

$$H = -\sum_{i=1}^{N} F_i \log_2 F_i$$

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* This is not really a translation of Boris Vian's *géniaux ingénieurs*.

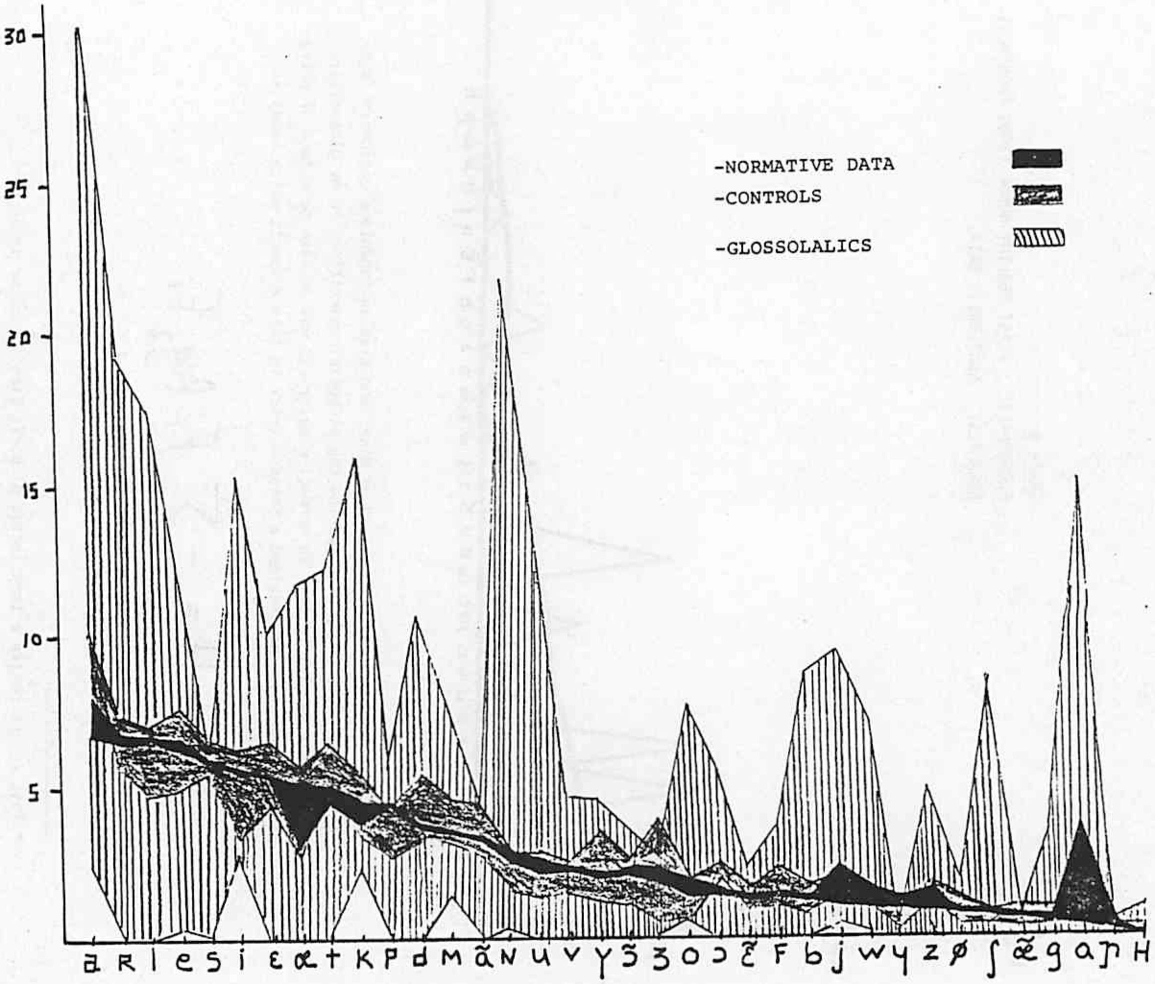


TABLE 1

	N-SANTERRE	N-LAFON	C-STANDARD Q	C-STANDARD F	C-SCHIZOPHASIC	C-TRANSCORTICAL	C-WERNICKE	C-CONDUCTION	G-SCHIZO ENGLISH	G-SCHIZO FUNNY	G-SCHIZO FRENCH-1	G-SCHIZO FRENCH 2	G-CHARISMATIC 1	G-CHARISMATIC 2	G-CHARISMATIC 3	G-CHARISMATIC 4	G-CHARISMATIC 5	G-NURSE 1	G-NURSE 2
[p]	4,4	4,3	4,1	3,9	3,9	3,6	3,0	2,6	2,0	2,5	1,7	2,4	0,7	0,0	0,0	0,2	2,4	0,1	2,2
[b]	1,3	1,2	0,7	1,6	0,9	2,1	1,4	1,2	8,6	3,8	6,9	6,8	0,0	0,1	0,0	0,0	0,0	1,0	3,5
[m]	3,4	3,4	4,2	4,5	3,5	2,5	4,2	3,0	4,4	2,9	1,4	1,5	6,3	7,3	5,4	2,6	1,5	5,7	6,2
[t]	5,0	4,5	5,1	5,1	5,5	5,0	6,0	6,4	0,0	0,5	3,2	2,9	5,0	0,1	0,0	8,5	6,9	12,2	10,6
[d]	3,7	3,5	5,3	3,1	4,1	5,9	3,6	5,1	0,1	0,6	3,4	2,6	0,9	0,3	0,0	5,1	1,5	1,8	0,7
[n]	2,5	2,8	2,4	1,8	2,8	2,2	2,7	1,5	2,0	0,8	4,8	5,0	0,2	6,9	21,9	10,7	7,5	10,7	0,5
[k]	3,6	4,5	3,3	4,0	5,5	4,8	4,1	3,8	7,7	13,5	5,6	3,7	6,6	16,0	10,0	2,2	5,2	2,6	6,5
[g]	0,5	0,3	0,4	0,3	0,3	0,3	0,1	0,8	2,8	1,4	1,8	2,8	0,0	0,5	0,0	0,6	0,2	0,1	0,4
[n]	0,1	0,1	0,1	0,2	0,1	0,0	0,0	0,0	0,1	0,0	0,0	0,1	0,0	0,0	0,0	0,2	0,2	0,1	0,5
[f]	1,3	1,3	2,2	1,4	1,4	1,8	0,9	2,0	0,0	0,5	1,8	1,4	0,0	0,0	0,0	0,0	0,2	0,1	0,3
[v]	2,3	2,4	2,2	2,3	2,5	1,8	1,4	2,5	0,0	0,4	0,7	0,4	0,0	0,0	0,0	0,0	0,0	4,6	0,1
[s]	5,9	5,8	6,3	6,6	5,8	5,2	6,5	5,4	4,5	2,8	3,1	2,3	3,2	0,0	0,4	5,6	5,6	3,3	0,4
[z]	1,4	0,6	1,0	1,7	1,3	2,2	1,1	1,6	4,7	1,2	0,8	0,8	0,0	0,1	0,0	0,2	0,0	0,0	0,1
[ʃ]	0,6	0,5	0,2	0,7	0,3	1,0	0,7	0,7	0,0	0,3	0,2	0,0	8,4	4,4	4,1	5,3	1,7	0,9	2,9
[ʒ]	2,2	1,7	3,2	2,2	0,4	1,5	3,2	3,8	0,0	0,1	0,1	0,2	0,0	0,0	0,0	0,0	0,0	0,0	0,0
[l]	6,5	6,8	4,7	5,9	7,0	4,7	6,3	5,2	0,2	17,4	5,4	6,8	5,6	7,3	0,0	1,7	3,2	4,5	6,2
[R]	6,4	6,9	7,1	7,4	6,8	7,4	6,3	5,8	19,2	1,8	13,9	14,4	7,9	6,5	0,0	2,6	6,9	2,1	3,4
[H]	0,0	0,0	0,0	0,0	0,1	0,0	0,0	0,0	0,0	0,1	0,0	0,0	0,0	0,4	0,0	0,2	0,9	0,0	0,0
[j]	2,1	1,0	2,1	1,5	1,8	1,5	1,4	1,5	4,4	1,1	4,2	4,5	1,6	3,0	0,8	6,8	9,3	2,8	5,4
[w]	1,6	0,9	1,6	1,1	0,9	0,8	2,0	1,2	0,0	0,3	0,4	0,1	0,0	1,3	7,0	0,6	0,9	0,1	1,5
[u]	1,1	0,7	0,2	0,7	0,1	0,9	0,9	0,3	0,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0	0,1	0,0
[i]	5,4	5,6	4,1	6,2	5,5	6,1	4,7	3,1	2,7	11,4	4,2	5,5	15,4	2,7	11,6	6,0	9,1	11,7	9,9
[y]	1,8	2,0	2,3	1,2	3,4	1,6	1,1	1,6	0,0	0,5	0,8	0,3	0,0	1,9	0,0	0,0	0,0	0,2	0,3
[u]	2,0	2,7	1,2	1,9	2,1	2,1	2,8	2,3	3,5	5,0	0,3	0,1	2,3	4,4	0,0	4,7	0,6	0,3	13,6
[e]	6,1	6,5	6,7	6,6	4,9	5,0	4,9	7,7	0,3	3,2	4,7	8,2	4,7	4,8	3,7	7,3	11,7	8,2	1,2
[ø]	0,5	0,6	0,9	1,0	0,6	1,2	0,2	0,2	0,0	0,0	0,4	1,6	0,0	0,0	0,5	0,0	0,0	0,1	0,2
[o]	1,3	1,7	0,5	1,1	1,6	1,3	0,9	1,8	6,1	0,5	0,7	1,3	7,0	6,3	4,9	7,5	3,9	3,6	2,3
[ɛ]	5,3	5,3	4,4	5,8	4,9	5,6	6,5	6,1	10,1	1,0	1,3	1,2	0,0	2,9	0,0	4,3	2,2	4,0	0,8
[œ]	2,9	5,2	3,9	1,6	5,1	5,1	4,9	5,6	3,8	5,3	4,0	3,2	0,0	0,2	0,0	0,2	1,1	3,0	0,1
[ɔ]	1,4	1,5	1,8	1,8	2,4	1,5	2,0	2,1	0,0	0,2	4,9	3,4	0,0	5,3	0,0	1,9	1,9	1,2	1,2
[a]	6,6	8,1	10,1	8,2	8,6	8,6	8,2	9,3	12,3	6,4	10,8	10,9	24,2	6,6	30,2	15,0	15,0	6,6	12,6
[ɑ]	3,6	0,2	0,8	0,2	0,1	0,1	0,2	0,1	1,2	15,0	2,6	2,0	0,0	10,6	0,0	0,0	0,4	3,6	6,0
[ɛ̃]	1,2	1,4	1,2	1,2	1,0	2,2	1,5	0,8	0,0	0,1	2,1	1,4	0,0	0,0	0,0	0,0	0,0	0,0	0,0
[œ̃]	0,4	0,5	0,7	0,8	0,7	0,4	0,3	0,1	0,0	0,0	0,1	0,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0
[ɑ̃]	3,3	3,3	4,0	3,9	2,6	2,7	4,4	3,0	0,0	0,0	1,5	0,4	0,0	0,1	0,0	0,0	0,0	0,1	0,7
[ɔ̃]	2,2	2,0	1,0	2,5	1,6	2,1	1,3	1,8	0,0	0,0	2,7	2,8	0,0	0,1	0,0	0,0	0,0	0,0	0,5

In fact, what this friend suggested was to calculate the "H/Hmax" ratio for each sample, with "H" being the entropy of the considered sample given the actual occurrence of each of "N" different phonemes, and "Hmax" the entropy of the same sample had the "N" phonemes been equifrequent. Results were the following :

		N	M	H/Hmax
NORMATIVES :	SANTERRE :	38	184201	0,92252
	WIOLAN :	34	100000	0,92534
	BAUDOT :	36	50189	0,91960
	LAFON :	36		0,90708
	JUILLAND :	36	286946	0,92287
CONTROLS :	STANDARD Q :	35	2000	0,90714
	STANDARD F :	35	2000	0,91403
	AVE MARIA	32	144	0,91541
	SCHIZOPHASIC :	36	2000	0,89675
	TRANSCORTICAL :	34	2000	0,92544
	WERNICKE :	34	2000	0,91548
	CONDUCTION :	34	2000	0,91301
GLOSSOLALICS :	SCHIZO ENGLISH :	20	2000	0,87230
	SCHIZO FUNNY :	30	2000	0,77743
	SCHIZO FRENCH-1 :	32	2000	0,86830
	SCHIZO FRENCH-2 :	32	2000	0,86767
	CHARISMATIC 1 :	16	442	0,86100
	CHARISMATIC 2 :	22	1240	0,83579
	CHARISMATIC 3 :	11	242	0,82649
	CHARISMATIC 4 :	24	532	0,86738
	CHARISMATIC 5 :	25	463	0,85803
	NURSE 1 :	29	2000	0,80830
	NURSE 2 :	31	2000	0,81487

From a superficial descriptive point of view, these figures are easily summarized : whether standard or pathological, the control samples, with their 2000 phonemes each, and even the *Ave Maria* (in French), with its 144 phonemes, yield measurements that depart little or not at all from those inherent to normative data, whereas the glossolalic samples yield measurements that depart more appreciably from the norm, sometimes grossly so. This dissociation obviously bears signification of a sort, and I will come back to it. Nonetheless, I do not really know what to make of these data. Indeed, I do not quite understand why entropic measurements should be pertinent in studies of phonemic repartitions. Taking for granted that it is, and assuming that my samples are quantitatively adequate for this type of study, and that

there exist legitimate ways of comparing one entropic measurement to another, a (no doubt naive) question remains to be formulated : given that the equifrequency yielding "Hmax" supposedly corresponds to absolute disorganization, why should the phonemic repartitions in highly organized codes yield "H"s that tend to remain much closer to "Hmax" than do the "H"s of glossolalic discourses? Is it that the "Hmax"s of basic entities such as phonemes should be conceived as representing maximal potential for organization as well as maximal disorganization?

I will now turn to modes of interphonemic combination in glossolalia. Just as the phonemic choices in the eleven samples are permitted given the French phonemic inventory, so are their modes of combination permitted given the French phonological system. Here again, however, mimicry of mother tongue stops at this point in all samples. In other words, combinations which are frequent in French can be utterly neglected, and infrequent ones become predilection linkages. Moreover, only a very small proportion of permitted combinations are actualized in any given sample and, if consonantic clusters are (quantitatively and qualitatively) taken as a parameter of phonological "complexity", the eleven glossolalic samples are all "simpler" than control samples. It can therefore be concluded that the eleven samples are phonologically rule-governed, which does not really come as a surprise, and that the phonological system subserving their actualization corresponds to a simplified version of the French phonological system, which does not come as a surprise either. I might add that certain phonemic combinations that are probably acceptable only at words boundaries in standard French may occur within glossolalic entities that most listeners perceive as word-like.

At this point, I will pass to glossolalic units of the next level of complexity. In this respect, there are two main points I will discuss : one concerns certain inventories of formally related word-like units, and the other concerns the existence of elements of morphology in glossolalic discourse. Although with various degrees of conspicuousness, both phenomena occur in all of the glossolalic samples I have studied so far.

Thus, each sample comprises inventories of isomorphic word-like entities, that is, of word-like entities bearing to one another formal relationships akin to those one observes, in aphasia, between phonemic paraphasias and their corresponding target words. Take, for instance, the example in INSERT 4 below. I excerpted it from the discourse of a charismatic imprecator who was admittedly attempting to oust a succuba whom he thought had taken possession of me. Rules of transformation are relatively monotonous in this particular family of isomorphic quadrisyllables : if the more frequent form, [manakala], that is, is arbitrarily considered as a target, these rules allow a few specific permutations on all vowels and on not more than two of the last three consonants, reciprocal metathesis on first and second consonant, and a single expansion on first and third consonant, as well as on second vowel.

INSERT 4

manakala	manaʃkala	
manakolo	manaʃkolo	mwanakolo
manakuʀe	manaʃkule	
monokolo	manaʃkulu	
monokɛle	monoʃkolo	
monokɛʀe	monoʃkœli	
monokoʀa	monokwɔle	
monakɔʀi	monaʃkuʀi	
monakale	mɔnaʃkɔle	
mɔnakola	mɔnaʃkɔlɛ	
mɔnakuʀu	mɔnaʃkuʀi	
mɔnokeʀi	mɔnaʃkyʀi	
mɔnekaʀe	mɔnaʃwala	
mɔnɔkule	mɔnɔʃkola	
munekeʀe		
munukuʀu		
malakala	malaʃkɔlo	mwalakala
malakola	malaʃkulo	
		mwalokuʀi
molakola		
	majaʃkala	
	mɔjaʃkola	

Had one to select a single feature of glossolalia behavior in order to distinguish it from control standard discourse, which obviously excludes poets, one might confidently select these isomorphic sets.

There also occurs, in all samples, sets of word-like entities in which derivation rules of a sort seem to have been applied. Affix-like units used in this particular game could be borrowed from mother-tongue, or else, be hackneyed foreign prototypes. One of the most complex sets I have observed is from the schizophasic's first "French temperament". It is illustrated in INSERT 5. It comprises combinatory use of prefix-like entities such as [bi], [ʀœ], [kɔ̃], [kɔ̃tʀa], [epi], and so forth, and of suffix-like entities such as [ezi], [asjɔ̃], [al], [ite], and so forth, all of which belong with French affixial inventories. A relatively

INSERT 5

[baʀjeteʀezi] ±			baʀ	jeteʀ	ezi	
[ʀœkɔ̃fɔʀjaneʀezi] ±	ʀœ	kɔ̃	fɔʀ	janeʀ	ezi	
[ʀœkɔ̃fjɔʀezi] ±	ʀœ	kɔ̃	fjɔʀ		ezi	
[ʀœkɔ̃fjɔʀani] ±	ʀœ	kɔ̃	fjɔʀ		ani	
[ʀœkɔ̃fjɔʀanis] ±	ʀœ	kɔ̃	fjɔʀ		anis	
[ʀœkɔ̃fjɔʀasjɔ̃] ±	ʀœ	kɔ̃	fjɔʀ		asjɔ̃	
[kɔ̃stʀasjɔ̃] ±		kɔ̃	stʀ		asjɔ̃	
[kɔ̃tʀavizeʀjasjɔ̃] ±		kɔ̃tʀa	vizeʀj		asjɔ̃	
[beʀegasjɔ̃] ±			beʀeg		asjɔ̃	
[bʀibɛʀgasjɔ̃] ±			bʀibɛʀg		asjɔ̃	
[bʀibɛʀgasjɔne] ±			bʀibɛʀg		asjɔ	ne
[bʀibɛʀgasjɔnis] ±			bʀibɛʀg		asjɔ	nis
[bʀibɛʀgal] ±			bʀibɛʀg		al	
[bʀabal] ±			bʀab		al	
[bʀakal] ±			bʀak		al	
[ɛ̃tʀabeʀɔgal] ±		ɛ̃tʀa	beʀɔg		al	
[epinɔʀmenal] ±		epi	nɔʀmen		al	
[binɔʀmenal] ±		bi	nɔʀmen		al	
[mɑ̃bʀal] ±			mɑ̃bʀ		al	
[mɑ̃tʀalite] ±			mɑ̃tʀ		al	ite

simpler example, from one of the paroxytone nurses' samples, is quoted in INSERT 6; a few Latin-Italian-Spanish-like affixes occur in this one.

INSERT 6

			[anto] ±								
[el			anto] ±								
[el		am	anto] ±	[el		am	ɔnta] ±				
				[el		em	antal] ±	[el		em	asta] ±
				[al		am	ɔntala] ±	[al		am	ɛntɛs] ±
[e	ʀiv	ist	anto] ±								
	[ʀiv		anto] ±								
[el	ev		anto] ±	[el	ev		ɛntɔl] ±	[el	ev		ɛsti] ±
[in		ist	anto] ±								
[in		im	anto] ±								

My last point about the eleven samples will be on glossolalic units of a still greater level of complexity, that is, on phrase-like and sentence-like units. It should be clear, by now, that an outstanding characteristic of glossolalia is the recurrent use of predilection segments of various degrees of complexity. This phenomenon — which one might wish to call perseveration if this was not too pervasive a term to be useful here — can involve phonemes, syllables and other phonemic combinations, morpheme-like and word-like entities, even phrase-like and sentence-like entities. When the latter occurs, which was the case in all of the eleven samples, the listener is often left with the impression of an endless repetition of the same sentence-like entities. This is only partially substantiated by a closer look at the transcriptions. Even in caricatural cases, such as the one illustrated in INSERT 7, which is from the schizophasic's "English temperament", word-like entities behave, within the contexts of predilection sentence-like entities, in the same manner phoneme-like entities behave within the contexts of predilection word-like entities : one has the superficial impression of a loose although simple Markovian process being applied on two or three different keyboards at the same time.

INSERT 7

1 = [azumba] ±
2 = [bɛʀgɛs] ±
3 = [koʀo] ±
3'= [keʀœkoʀu] ±
4 = [bʀubjɛʀ] ±
5 = [paʀa]
6 = [bʀazjɛ mnɛʀgɛs] ±
7 = [aʀakaska ʀœkaʀœ] ±
8 = [misi] ±
8'= [mizœ pʀiz] ±

a = (3 ou 3' + 4)
A = (5 + 6 + a)
B = (8' + 7 + 6 + a)

$$\text{"SENTENCE"} = +1 \pm 2 + a + \begin{vmatrix} A \\ \text{or} \\ B \end{vmatrix} \pm A + \begin{vmatrix} 8 \\ \text{or} \\ (B + 8) \\ \text{or} \\ (B + B + 8) \\ \text{or} \\ (B - 4) \end{vmatrix}$$

e.g. [azumba | bɛʀgɛs | koʀo bʀubjɛʀ | paʀa bʀazja mnɛʀgɛs keʀɔkoʀo bʀubjɛʀ | mizœ pʀiz aʀakaska ʀœkaʀœ bʀazjɛ mnɛʀgɛs keʀakoʀo]

e.g. [azumba | bɛʀgɛs | koʀo bʀubjɛʀ | paʀa bʀazjɛ mnɛʀgɛs keʀakoʀo buʀbjɛʀ | mizœ pʀiz aʀakaska ʀœkaʀœ bʀazja mnɛʀgɛs keʀakoʀo bʀuhjɛʀ | misi]

e.g. [azaʀœ | koʀo bʀobjɛʀ | paʀa bʀasja mnɛʀgɛs kɛʀœkoʀo bʀobjɛʀ | miza pʀiz aʀakaska ʀakaʀœm bʀasja mnɛʀgɛs kɛʀokoʀo bʀobjɛʀ | miza pʀiz aʀakaska ʀakaʀa bʀazja mnɛʀgɛs kɛʀokoʀu bʀobjɛʀ | mesi]

e.g. [azaʀa | kuʀu bʀubjɛʀ | paʀa bʀasja minɛʀgɛs kɛʀœkuʀu bʀubjɛʀ | mizœ pʀiz aʀakaska ʀœkaʀœ bʀasja minɛʀgɛs kaʀœkuʀu bʀubjɛʀ mizœ pʀiz aʀakaska ʀakaʀa bʀazja minɛʀgɛs kɛʀokoʀu bʀubjɛʀ | mesi]

In INSERT 7, I have first listed the 13 basic word-like constituents of the schizophasic's "English temperament". Nearly all of them can have one or several isomorphic phonological variants, hence the plus-and-minuses following them in the list. Three preferential phrase-like constituents are also listed : they are identified as "a", "A" and "B". The "grammar" of the schizophasic's "English temperament" is thereafter summarized in a formula following the word "sentence".

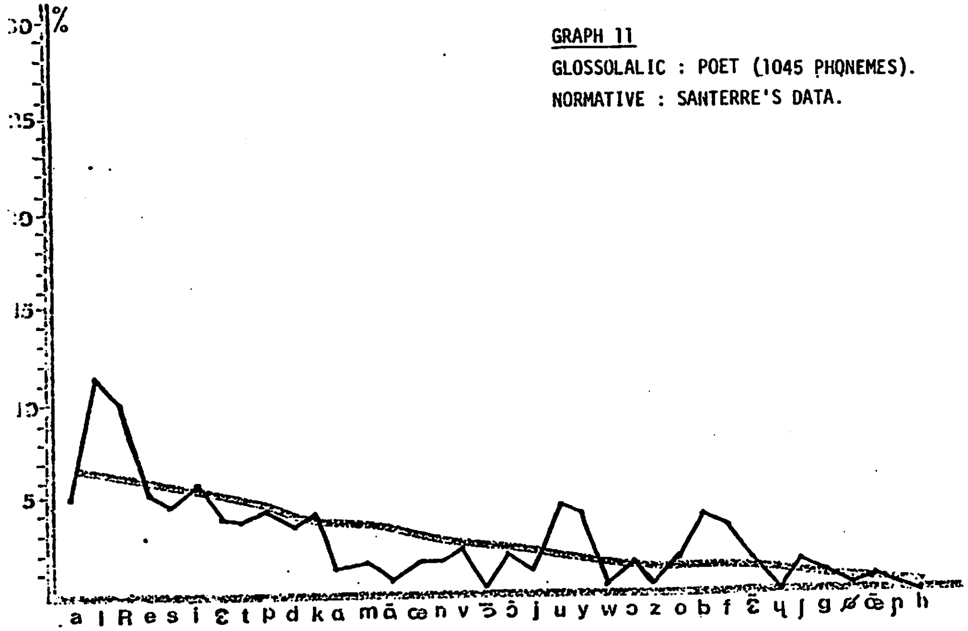
Keeping in mind this description of the eleven samples, and assuming that ghosts, devils and the like are of only moderate importance (here and nowadays), one can suggest that the programs behind glossolalic simulation are mainly founded on various fragments of apprenticeships related to speech production. In unilingual speakers, it is likely that most of these fragments are related to the production of mother tongue, although passive exposure to foreign languages and resulting apprenticeships, however passive and minimal, are apparently playing a seasoning role.

Let me now ponder on a few questions such as : how successful is glossolalic simulation? how close does it get to a natural language with regard to various formal — segmental and suprasegmental — aspects? how close does it get to sounding "foreign"? to what extent can it be considered to be "semantic"?

As obvious from the reaction of most listeners of glossolalic discourse, the simulation reaches some degree of credibility with regard to segmental aspects : it does yield outputs comparable to multiarticulated discourse of a sort, that is, outputs that can be described in terms of simpler units being integrated into progressively more complex ones. On the whole, however, the number of disponible units as well as their modes of selection and combination remain comparatively poor and, moreover, this paucity increases with the degree of complexity of the considered units : it is somewhat less conspicuous at phoneme-like level, intermediate at word-like level, and maximal at sentence-like level.

Among the twenty samples considered so far, I found one in which the segmental paucity inherent to glossolalic behavior is appreciably less obvious than in the nineteen others. As illustrated in GRAPH 11, this is reflected, at phonemic level, in a repartition that is intermediate to the one in normative and control samples and the one in other glossolalic samples. This difference probably stems from two main factors : on the one hand, this sample comprises a fair number of *bona fide* French words (indeed, to such an extent that one might wonder if it answers the definition I gave of glossolalia) and, on the other hand, it was produced by a (professional) poet whose "improvisation" was that of one with a long, deliberate experience of utterly calculated, paper-and-pencil glossographic creation. It should be noted, nonetheless, that with an H/Hmax ratio at 0,90285, the highest within our glossolalic corpus, this sample still ranks below the three jargonaphasic controls (see page 15).

Suprasegmental aspects now : I am somehow convinced — although I cannot justify my conviction — that prosodical choices are more deliberate than segmental choices in glossolalic behavior. Moreover, there is a basic difference between segmental and prosodical choices in that the former leads to neologistic production whereas the latter does not. Be that as it may, glossolalic mimicry of speech production is quite successful with regard to suprasegmental aspects : indeed, glossolalic speakers are perfectly capable of sticking to a prosodical model, usually a simple one, with a relatively small number of well defined melodical features, such as the ones characteristic of recitative prayer, political propaganda, and so forth.



GRAPH 11

GLOSSOLALIC : POET (1045 PHONEMES).

NORMATIVE : SANTERRE'S DATA.

As I have mentioned, there are features in the glossolalic utterances of unilingual speakers which do not witness to loans from mother tongue : after all, the game is to act as if one was not unilingual. This phenomenon can concern segmental as well as suprasegmental facts : changes in phonemic repartitions, and also the use of a few foreign phonemes and affixes are examples of the former, and changes in regional or tonic accent are examples of the latter. But whereas segmental loans remain very sparse indeed, and very elementary, and very superficial, suprasegmental ones sometimes evenly dominate the whole of lengthy glossolalic discourse. The striking fact, in this respects, is therefore that minimal passive exposure to foreign languages provides enough for certain individuals to learn and consistently reproduce suprasegmental components, which is seldom if ever the case for segmental ones*. Is not this a rather interesting dissociation? In other words, could not one suggest that certain glossolalists, although they are never segmental xenoglossists, sometimes come very close to being genuine suprasegmental xenoglossists? I wonder if a similar dissociation in reception might explain why listeners of glossolalic discourse usually tend — as you know — to assimilate what they hear to some language they do not speak but have been exposed to.

Another question that might be repeated and dealt with at this point is the following : are there semantic components to glossolalic simulation and, if so, how succesful?

The answer might be yes-there-is-and-quite-successful if one considers only prosodically conveyed messages : for instance, the affect inherent to the discourse of a praying charismatic is very much unlike that inherent to the discourse of an imprecating charismatic.

Things are quite different with regard to segmental aspects of glossolalic mimicry. In this respect, my glossolalic poet teaches that there are indeed messages inherent to phonemic choices : thus, he insists that a production of some lenght comprising 20% of unvoiced dorsovelar stop consonants will inevitably sound aggressive : obviously, he is refering to what is known in French as *harmonie imitative* : he may have a point there. This notwithstanking, I think that glossolalic imitation is not semantically targetted as far as segmental values are concerned. More precisely, I do not believe that glossolalic utterances represent systematic transformations of standard utterances, as would be the case, for instance, of several forms of typically aphasic utterances; and I think that glossolalic speakers do not consciously attribute precise meanings to their word-like and sentence-like productions.

I guess that an argument in this sense might be derived from entropy measurements, or again from the omnipresence, in glossolalic materials, of families of isomorphic word-like entities, which is definitely not a characteristic of non poetic standard speech. Nonetheless, given that most of us are sort of interested in the mutual relationships of brain and language, I will seek my

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* Unless in circumstances such that segmental values are, as it were, subordinated to suprasegmental ones : think of how most of us learn songs, and of how difficult it can be to recite the corresponding texts isolatedly (on a non-song prosody). Which reminds me of the lady who sang in beautiful Italian at *La Scala* but was unable to order *pastas* at the restaurant next door, but this is an altogether different matter.

argument in aphasic material.

Once in a while — or rather very seldom — one meets an elderly stroke patient with very severe Wernicke's aphasia and a fluent phonatory production that answers the empirical definition I gave of glossolalia. Anna Mazzucchi, from Parma, and Carlo Semenza, from Padova, have each recorded one such patient; at the last I.N.A. meeting, Ellen Perecman and Jason Brown have reported on a third, and I have myself observed two cases. Exemplary transcripts from these two are presented in INSERT 8, and phonemic repartitions are given in TABLE 2. The repartition in the first of these cases is shown in GRAPH 12; compare it to GRAPH 5, which accounts for the phonemic distribution in a control sample from a patient with "regular" logorrheic neologistic jargon. I might add that the two glossolalic samples I obtained from elderly Wernicke's aphasics do show several if not all of the segmental characteristics I have described in the original eleven samples, including, as illustrated in INSERT 9 and INSERT 10, which I excerpted from the productions of the first of these cases, the families of isomorphic word-like entities and the elements of morphology. Therefore, their discursive behavior shares more with that of non aphasic glossolalics than with that of non glossolalic aphasics.

INSERT 8

GLOSSOLALIC WERNICKE #1

[sɛ̃ dikte di tʀɔ̃ kɔ̃deʀe dʀikɔ̃dedeʀe digœʀe dis tis tilavɛ kloʀe œ le dø tʀɔ̃ke ditibɛ dœʀe disœ te kotegoʀe dil kɔ̃deteʀe a wi dœ vilɛbʀiʃ ʒe la lɑ̃bɛtɔʀi de del lɑ̃tetɛʀœme di kateɑɔʀe e œ e ɛlzekute ɛlmœpuʀimakɔ̃te tɑ̃ tutse dœgʀedœgʀe dis gy lateʀe digeloteʀe]

GLOSSOLALIC WERNICKE #2

[vaʀite sœpœ lœbsœ pazezø kœ bœsœmɔse bœdze bosœ bɛ̃ ʒe pje yn bɛ̃ bajesø mɔpɔ mpɔma vjɛ bɑmbɑ ʒœ mapɔzø pɑɑpuʀ mœʃwø ʃepa bytse bɔvʀe siʀse ʒœ va pɑse pɑte gɑ aʃydy mwɛ̃ pise maʒemɛ mekɔ vjɛse seʒamɛ ʒe peteʀœ samɛ ʒamɛ pɑze pɑ ʀjɛ̃ dzutu ʒamɛ pedœ tepeʀje ɔbɛ̃]

Now, if you give a close look at the data in TABLE 2, you will notice an overuse of the three voiced stop consonants, that is, of /b/, /d/ and /g/, in the sample of the first glossolalic Wernicke, and of two of them, /b/ and /g/, in the sample of the second glossolalic Wernicke. I come to my argument, and here Sheila

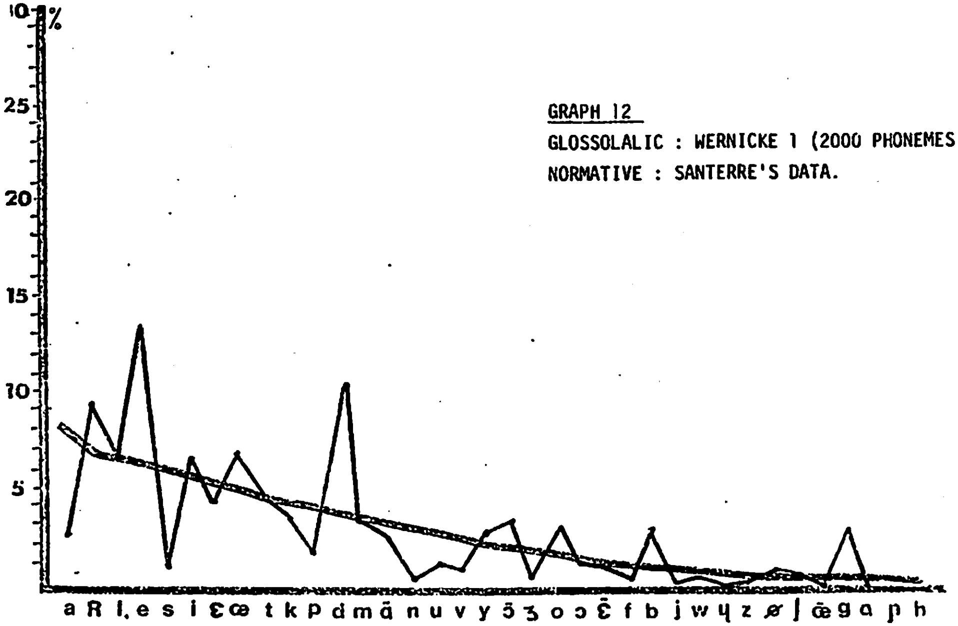
TABLE 2

	N-SANTERRE	N-LAFON	G-WERNICKE 1	G-WERNICKE 2
[p]	4,4	4,3	1,6	5,6
[b]	1,3	1,2	3,2	3,1
[m]	3,4	3,4	3,4	5,2
[t]	5,0	4,5	4,5	3,7
[d]	3,7	3,5	10,7	2,0
[n]	2,5	2,8	0,4	1,4
[k]	3,6	4,5	3,4	3,1
[g]	0,5	0,3	3,3	1,4
[ɲ]	0,1	0,1	0,0	0,0
[f]	1,3	1,3	0,3	0,5
[v]	2,3	2,4	0,8	2,6
[s]	5,9	5,8	1,0	6,0
[z]	1,4	0,6	0,2	2,5
[ʃ]	0,6	0,5	0,5	1,1
[ʒ]	2,2	1,7	0,4	2,4
[l]	6,5	6,8	6,8	2,4
[ʀ]	6,4	6,9	9,3	3,4
[ɥ]	0,0	0,0	0,0	0,0
[j]	2,1	1,0	0,2	3,4
[w]	1,6	0,9	0,4	1,8
[u]	1,1	0,7	0,1	0,3
[i]	5,4	5,6	6,5	3,9
[y]	1,8	2,0	2,7	1,2
[u]	2,0	2,7	1,0	0,9
[e]	6,1	6,5	13,4	8,3
[ø]	0,5	0,6	0,8	1,0
[o]	1,3	1,7	3,2	0,5
[ɛ]	5,3	5,3	4,2	3,6
[œ]	2,9	5,2	6,9	11,7
[ɔ]	1,4	1,5	1,2	2,5
[a]	6,6	8,1	2,4	4,9
[ɑ]	3,6	0,2	0,1	5,1
[ɛ̃]	1,2	1,4	1,1	2,2
[œ̃]	0,4	0,5	0,1	0,5
[ɑ̃]	3,3	3,3	2,5	0,8
[ɔ̃]	2,2	2,0	3,4	1,0

Blumstein's works on phonemic deviations should be considered : if the discourse of these patients was targetted on standard language, one would expect a decrease, not an increase in the frequency of marked phonemes such as voiced consonants. Well, think of it.

Let me now switch to still another problem raised by the very existence of glossolalia in various categories of speakers : besides anosognosia, I can identify two basic differences between the discursive behavior of the two glossolalic Wernicke's aphasics and all of the other glossolalics I have considered till now, poet included : the first is that glossolalic production represented an exclusive residual behavior in the two aphasics, who had no choice but to shut up if they did not glossolalize, whereas it coexisted with a capacity for standard speech production in the others; I will discuss the second difference later.

The first difference leads one to ask about the brain structures the integrity of which is necessary in order for glossolalic simulation to be possible. Well, if I am not mistaken as to the signification of the formal kinship between the glossolalia in subjects with and that in subjects without brain lesions, whether the latter be schizophrenics, charismatics, or admitted simulators, glossolalic behavior remains possible, at least in right-handed elderlies, in the presence of important left-hemisphere destructive lesions : in one of my patients, left posterior temporal lesions were documented by gamma-encephalography; in the other, CT-scan images showed massive left occipital softening as well as bilateral cortical atrophy, so formidable that it was impossible to tell if unilateral or bilateral temporal lesions had or not taken place when aphasia had suddenly occured. I will let Jason Brown tell you about the bilateral lesions in his own case. In other words, when occurring in the aphasic, glossolalic behavior — a euarthric and phonologically rule-governed fluent speech-like production — does not depend on the integrity of the classical speech area, in particular of



GRAPH 12
GLOSSOLALIC : WERNICKE 1 (2000 PHONEMES).
NORMATIVE : SANTERRE'S DATA.

INSERT 9

kategɔʀe
kotegoʀe
kotedœʀe
kodolœʀe
kɑ̃tegoʀe
kɑ̃tɛgœʀe
kɔ̃tɛgɔʀe
kɔ̃tegœʀe
kɔ̃tɛdœʀe
kɔ̃tɛbœʀe
kɔ̃deteʀe
kɔ̃dydeʀe
kikɑ̃dœʀe
kilɔ̃deʀe

INSERT 10

[mɛtʀ] ±	___	___	___	mɛtʀ
[ademɛtʀ] ±	___	ade	___	mɛtʀ
[digɛtʀomɛtʀ] ±	dig	ɛtʀo	___	mɛtʀ
[dikelimɛtʀ] ±	dik	eli	___	mɛtʀ
[ɛledœʀe] ±	___	ɛle	___	dœʀe
[dikɑ̃tedœʀe] ±	dik	ɑ̃te	___	dœʀe
[gylɑ̃tedœʀe] ±	gyl	ɑ̃te	___	dœʀe
[gylɔ̃te] ±	gyl	ɔ̃te		
[gylgydetɔ̃byl] ±	gylgy	de	tɔ̃b	yl
[detɔ̃bœʀe] ±	___	de	tɔ̃b	œʀe
[detɔ̃bɛtʀœ] ±	___	de	tɔ̃b	ɛtʀœ
[detɔ̃bɛʀ] ±	___	de	tɔ̃b	ɛʀ
[detɔ̃be] ±	___	de	tɔ̃b	e
[vatɔ̃be] ±		va	tɔ̃b	e

its temporal components; as a matter of fact, having once seen a case of fluent jargon with extensive fronto-parieto-temporal lesions destroying Broca's area and its homologue in the right hemisphere (Figs. 1 and 2), I am not even sure that the frontal components of the classical speech area are always necessary to euarthric phonologically rule-governed production. This is as far as I wish to go, for the time being, concerning anatomy.

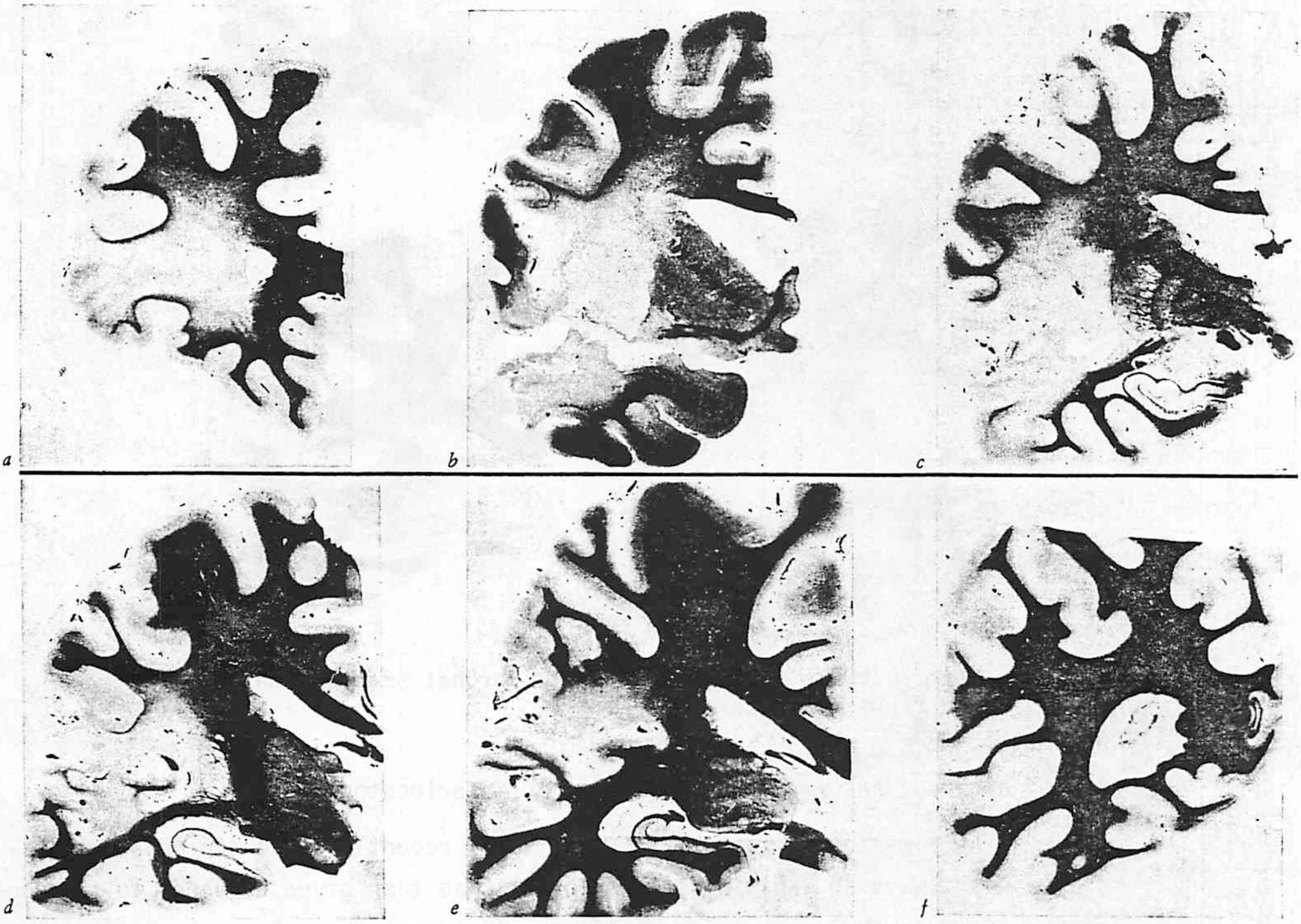


Fig. 1 : Coronal sections of the left hemisphere in a case of fluent logorrheic jargon. Excerpted from Lhermitte, F., Lecours, A.R., Ducarne, B. & Escourolle, R., "Unexpected anatomical findings in a case of fluent jargon aphasia." Cortex, 9, 433-446, 1973.

The second basic difference between the two aphasic samples and the twelve others is that all of the latter occurred in the form of monologues, whereas the former could only occur within apparent dialogues, that is, in the

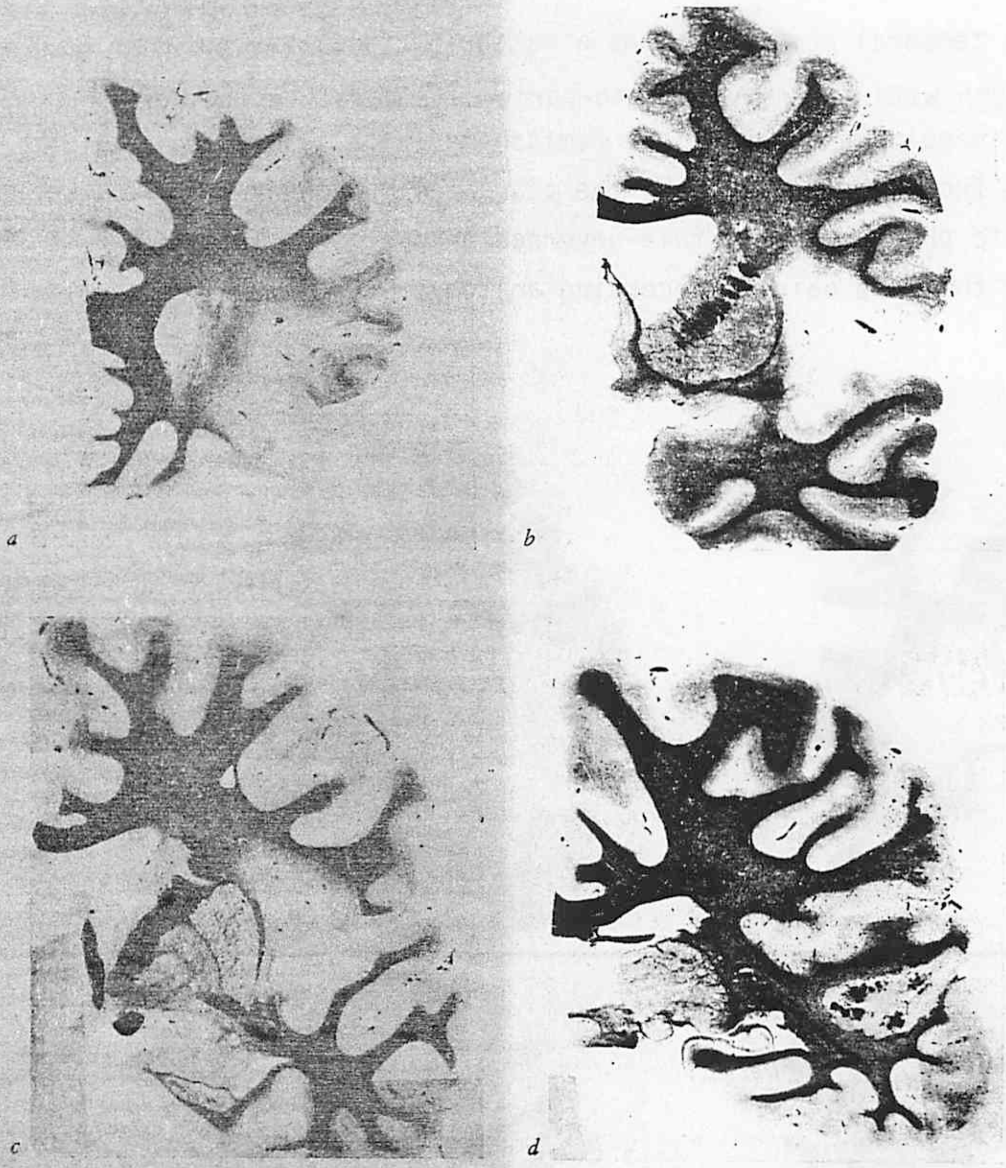


Fig. 2 : Same case as in Fig. 1; coronal sections through the right hemisphere.

form of apparent answers to questions of an interlocutor.

As a matter of fact, I thought, until recently, that non aphasic glossolalia was always an individual behavior. I was then given evidence to the contrary : after recording the glossolalic monologues of three British charismatic believers, who had never met each other, two friends of mine, Paolo Fabbri and Silvano Fua, from Urbino, asked them if they thought they were capable of sharing a conversation in tongues. They answered that yes-they-were, and they did. You have seen the video document on which their simulated conversation was recorded. An I.P.A. transcript of part of it is presented in INSERT 11.

INSERT 11

ROBERT

kɔbri jɛtɛs sœlojɔno sœ meetees kɔnt— pɛveam
koreɛmto soream— pevɛtuo

Antony

kanajæ sutramɪ jakæ ʃɪniteba— mas inebalaj
tisekoralaji semitea pʊrta la pakantsɪɛ mɔnta

ROBERT

mɔwia— trɛs— tɔjɔm

LUISA

o no no se marazse— e lɛ rɛtʃeʃe ʃinœ no
kɔrisis duena pafatna— ah kaka rɛʃilɛ— ɛnjuno
lə pɔtʃisifta— æ̃hæ̃

Antony

fumare esi bəlaham ma teɛsə konɔtɔj apɪʃike—
miʃera ihim ɛn sepazəbalə tramiʃ ʒnɛkɛ
daotrə mjɛ sɛ kata mujetɛ ʌ:m mɜɪ badta
lai xlise alanansia bɔtʃs ʌ:m— si mia kɔrolo mi
side ma kataʃi ineka — nœ sɛ pa

LUISA

≈hã

ROBERT

nɔbia— nɔbia sɔβiato— pɛkeniʃ — uʃɛna bɔdzuljako—
aliʃənə vœβliko malezjɔ̃m— tɛjʃii somalit— kalabuab
tit kalisi noɡule— arzɔdi pijajanɔ kɔrsitiç— tia—
pigo tsiko

Antony

hɛrɛ nɛlji ʃɪlam maratose ɛmat— ɛsɪ nax tibi
ʃinotre e meʃikanum pɔra jɛte— esɪ brɛtʊŋ
e amajis ixanɔ to mas a najte lalais

ROBERT

adi

Antony

ʌ: də bɪtreak sɪnatra bi iniki tifa— mɛkwa tennə
ɛknɪsiun tɔrɛk sajam ɔraj si miktea kɔnɔk trese—
ʌ:m bɔt aj mɪse a la matsjɔ̃ kɔm tɔj— mibea
to rabal ɛs initɛri peoto kanaj i se balaj i
neɔ̃ no kalaj si— m: dœ bœ ʒœ: vɛ alam
mirɛ— m: ʒə makala vɛ no ripi sonopa ʒə
mɛt evɛn mɛre trivi ʒe bɛtra lɔ̃tɔ̃ imisãlatə
mɛsɔ̃ sɔna kafa mãtis mela trøvy ʌ:m— ɛl fãsɛ
me lafal e mə kɔsãt ʒœ bɛ ʌ: ʒœ tatø bø
tɔfi niokowa— œlakwa ɛ mi kwɛreɛs somo sœtœ
valiti— øʃø makata

LUISA

nm oɔ̃ musɛ fata— zede e ʃine no lɛtja

ROBERT

pɔja sɛtɛ tirbis səlɛk kɔjan rapo— pʊr əzija kɛra—
kara sʊran tɔja tia— kɛs sobajako— alɪʃ ʃɛnis
kœviam ɛkik— al moɛntis salixua— ʃia— kovampea
kwo sijo paʊŋ kuo o — povo siɛti— sɔri tɔrian
tuo— atpifiɛn kanla sitiø— sajɛno tolɪan— kadit—
mebian— tɔjate — soviɔt ebit ɛjis— sana— paviato
soʊjætə— peveam— pʊʃujɛko

Antony

kumari e ʃinatra ɛspreto samɛlla peanɔsi ɛn kamartə
kɔmarə kamamə səmati fiamo pɔjɛsɔi— ʌ: βiʃe
pɛlɛjano mœs amat ise œ̃ pokə nəjɛtʃi henɪjɛtɪ
pehati— nebeatʃi kedɛjabotʊ jɛme set amat satɔ
ike mɛtʃi muntʒi— nibaj solotrɔ bakata tsia
pumpra alaʊsia kristʊ— nɪzea trɔma kotaiiʃi—
mʊssala rea sekana trea kasi iʃtal ammot—
ɪnakrea tanasa

Analysis of the phonemic repartitions in the neologistic productions — monologues and dialogue — of these three native speakers of English yields the following entropic measurements :

		N	M	H/Hmax
NORMATIVE :	DEWEY	42		0,90618
GLOSSOLALICS :	CHARISMATIC 6 (M)	45	1528	0,83450
	CHARISMATIC 6 (D)	49	2655	0,77201
	CHARISMATIC 7 (M)	41	1134	0,84208
	CHARISMATIC 7 (D)	40	848	0,84945
	CHARISMATIC 8 (M)	46	1338	0,79836
	CHARISMATIC 8 (D)	52	2319	0,80271

In spite of the superficial impression conveyed by the productions of these three experimented glossolalists, the difference between normative and glossolalic material, within this set of data, is thus of the same order as that found concerning the eleven cases previously considered (see page 15).

My study of these productions is far from complete, but I think I can say that, on the whole, suprasegmental and segmental characteristics of the monologues are identical to those in other charismatic samples : prayer-like prosody, isomorphic families, and so forth. So are segmental characteristics of the dialogue. On the other hand, suprasegmental characteristics of the latter are indeed those of a somewhat theatrical animated conversation*. Therefore, a striking feature of this particular simulation is again a dissociation between suprasegmental and segmental parameters : while prosody successfully duplicates that of conversation, and apparently leads to holistic semantic exchanges of a sort, a comparison of monologues to dialogue shows that each protagonist keeps using his own unsharable tongues, therefore exerting very little if any influence, for instance as to word-like entities, on the segmental choices of his interlocutors.

In a way, one might say that general prosody, whether conversational or otherwise, is always appropriate in glossolalic behavior, and consequently suggest that it needs not be considered a part of the simulation, *i.e.*, that the simulation proper — although supported by regular prosodical apprenticeships and habits — is mostly if not exclusively directed at segmental aspects of speech production. Well, dissociations of this sort are not unheard of in computer simulation either.

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* Although without the theatrical character, the prosody in the two samples from glossolalic Wernicke patients is also, appropriately, that of conversation.

I can see that John Marshall is soon going to say that this-is-all-very-diverting-and-we-have-all-been-very-diverted-indeed-but-what-is-the-interest-of-it-all? Now, it depends :

☐ If you are a theologian, or else a psychoanalyst, it might be for your own good and interest — however disheartening — to know that, but for Hugo Pratt's (Fig. 3), no recent studies have provided evidence that some people can be proficient in languages they have not learned.

☐ If you are a fan of marginal psycholinguistics, you might be interested in knowing that glossolalia is not a form of cryptophasia but rather a learned game — and a rather simple one at that — founded on a capacity to maintain standard prosodical models and standard phonetics while fluently uttering in line with simplified phonological and greatly impoverished morphosyntactictal conventions.

☐ If you are a buff of stratificational linguistics, you might be interested in knowing that glossolalists also have a few strata.

☐ If you like M.I.T. linguistics, you will have to look for something I have not seen.

☐ If you dabble in mathematics, maybe you will tell me more about stochastic processes. And maybe not.

☐ If clinical aphasiology is your trade, it is perhaps not without interest for you to learn — if you did not already know — that certain brain lesions, in certain elderlies, can lead to residual brain functioning such that it yields a jargonaphasia behavior sharing more, from a linguistic point of view, with schizophasic and charismatic glossolalia than with other forms of aphasic jargon.

☐ And finally, if you are an addict of neurolinguistics, whether or not you believe that neurolinguistics must be computational, you might be interested in glossolalia as further evidence of the possibility of dissociated

Fig. 3 : Xenoglossia; excerpted from Pratt, H. Rendez-vous à Bahia, p. 28, Casterman, Tournai, 1973.

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functioning in nerve nets related to speech production. You might even find this evidence comforting if you are yourself involved in simulation experiments in which you have ignored, say, prosody, or maybe semantics.

Pierre Lavorel : Comments on Lecours'Presentation.

PROCESS CONTROL AND MOTIVATION IN GLOSSOLALIA

Is there a syntax of the glossolalic utterance?Are there semantic goals?

It does not seem to be a stochastic process of the simple kind measured by entropy.I drew a state-diagram for the first charismatic in Lecours'sample to analyze the basic rhythm and chart the basic elements of recurrence. The coefficient of variation of the rhythmic period seemed too high for a simple Markov model to be convincing.Nonetheless,rhythm is a basic facet of glossolalia.In Lyon,we had a Wernicke aphasic with glossolalic manifestations.We found the melodic variation in her utterance to be far more regular than that of natural language.

If probabilistic models do not suffice to understand glossolalia,a"systemic" approach would perhaps be more helpful.Complex interactive processes involving not only a phonological generator but also symbolic,motor and proprioceptive functions seem to be at work.We have studied the effect of delayed auditory feedback on people attempting glossolalia.A 500 msec.delay is sufficient to hinder the normals,but there is no such impairment with Wernicke aphasics(or with schizophasics).What then are these control processes of euphony and of cacophony which differ in normal and in abnormal speech?Syllable formation,phonic sequence formation,seem to be carefully organized in glossolalia and in xenoglossia.I studied an English lady who claimed her glossolalic speech was "Pharaonic Egyptian"but found that she used the English stock phonemes and euphonic principles akin to alliteration,assonance and complementation which require a lot of self-control.

Now,what are the semantic goals behind the formal aspects of these elaborate language games?We can always revert to ontogenetic and to phylogenetic arguments.Children,for instance,are known to be good at jabbering repetitive variations.At one lunchtime,I had my children simulate glossolalia by pretending to speak "African" or "Chinese".But they reinvented linguistic strategies. Their intonation became varied to convey the "mood" of an argument;later on they evolved "words" for objects on the table;and by the end of this 35-minute experiment,I had the impression that a "pidginization" of their own syntax (subject,verb,object) began to emerge. Perhaps these mark the basic stages that distinguish true propositional language from glossolalia.Now,in spite of this rather negative experiment,it seems fair to say that incantations have always appealed to children,as well as to poets.Poetry is characterized by alliteration,assonance and rhyme."Rhyme" and "rhythm" are derived from the same Greek root "rhythmos" ;Koestler said that "rhyme is but a glorified pun" . Do these various poetic figures,then,all come from forms of rhythm which may themselves be related to the basic characteristics of glossolalia?Perhaps glossolalia, like primitive songs and ancient poetry, exhibits the hidden puns of another world,whether that be the world of devils and gods as claimed by"possessed"glossolalics,or the nether world of the subconscious mind.Sheer articulatory pleasure may indeed be part of the story --Fonagy claims to have shown that the movements of the vocal tract(tension,relaxation,vibration,constriction)have something to do with deeply rooted oral impulses.

To end my remarks with a neuropsychological hypothesis about glossolalia,we may recall the finding of Luria and Vinogradova(1959) that chloral hydrate makes people associate sounds easily, but disturbs the association of words to convey meaning. What is inhibited ? Perhaps mechanisms for planning, possibly located in the frontal lobes or at the tip of the temporal lobes,or in the supramarginal and angular gyri.Besides,one should not disregard subcortical influences. In The act of creation, Koestler cites Förster's syndrome. In the 1920s, Förster studied

a subject who exhibited manic speech on manipulation of a tumour in the floor of the third ventricle. Each word of the operator would trigger a flood of associations. So we should perhaps consider the role of ventrolateral thalamus.

But there is a lot of fiction in this. The main point is that it is insufficient to look at the surface data if we are to simulate the "simulators", i.e. the glossolalics,in their speech.

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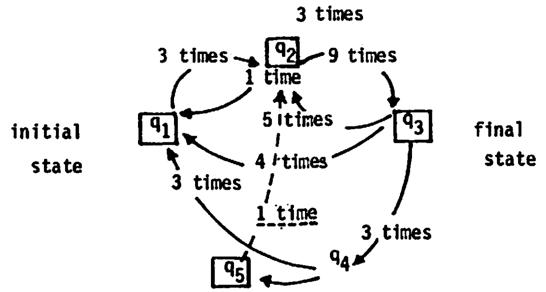
APPENDIX. State diagram for Charismatic #1.

If the states corresponded to the following equivalence classes:

$$q_1 = \{0\};\ q_2 = \left\{\begin{array}{l}\text{kiRia}\\ \text{silia}\\ \text{sikolea}\\ \text{sikolia}\\ \ldots\end{array}\right\};\ q_3 = \left\{\begin{array}{l}\text{RamatuReiʃ}\\ \text{maRamakoleiʃ}\\ \text{Ramataiʃ}\\ \text{maRanataiʃ}\\ \ldots\end{array}\right\}$$

$$q_4 = \left\{\begin{array}{l}\text{jaʃimdea}\\ \text{jara}\end{array}\right\};\ q_5 = \{\text{matoi}\},$$

the state diagram would be:



transitions :

q_2 : 12/31; q_3 : 12/31; q_1 : 5/31; q_4 : 3/31; q_5 : 1/31.

DISCUSSION OF LECOURS PAPER

Marshall: Is the study of glossolalia more than entertainment? Marslen-Wilson asked Woods how we can get a psychologically meaningful decomposition of language performance into "knowledge sources". Does glossolalia help us answer? What components of language production can we turn off? Can these be related to lesions? I claim that with 5 to 10 minutes of practice, *anyone* can be a first-rate glossolalic. But what can you *not* consciously turn off? Even with practice, people find it very hard to produce random or semantically anomalous wordstrings. Why? In glossolalia, everything is turned off except the phonology and the prosody. But even with the syntax and semantics turned off, we still get hand and body gestures fitting in perfectly with the prosody -- an interesting decomposition.

Marin: For another example of dependence of functions, note that echolalia can only occur if there is breakdown of comprehension.

Brown: Some schizophrenics are aware of their glossolalic production. Those who are unaware tend to be more deeply regressed. Glossolalic aphasics, that is, cases of "phonemic" or undifferentiated jargon, seem to be very rare, and also quite old -- in their 70's. In a case that I have studied with Ellen Perecman (1980) there was a deviant phonemic distribution which was somewhat less marked in reading than spontaneous speech. However, we found that altering the phonemic distribution of the text did not affect the phoneme distribution of the reading. This suggests that the jargon is sensitive in a not very specific way to some constraints on performance. In contrast, neologistic jargon is said to show normal phoneme distribution. Presumably this is so even if one samples only the neologisms. However, it is hard to see how the phoneme distribution can fail to be affected by the predilection sounds. The real question is how consistent these alterations are across different jargonaphasics, and how are they to be understood. I might add that neologistic jargon-aphasics differ from cases of phonemic jargon in their heightened affect and logorrhea, the disturbance in auditory attention and the loss of the normal speaker-listener relationships.

Kertesz: Another entity to consider is mumbling. The lesions are invariably large (they can be unilateral) and involve the temporal lobe, more posteriorly than laterally. The connection of Broca's area with the supplementary speech area is intact, and these patients can recover limited fluency. Apart from the mumbling itself, these patients are much like global aphasics.

Levine: I studied a woman with a small lesion which cannot be seen in the CT scan. She exhibited an ongoing, apparently meaningless, "machine gun" alternation of consonants and vowels. Yet she had quite good comprehension, she could write with only minor spelling errors, and she could thus communicate very well through writing. We thus see here an element of glossolalia without regression of general language facilities.

This raises the interesting question of the relationship between impaired comprehension and fluent paraphasic speech output. Both are commonly related to posterior lesions but the output and input deficits are notperfectly correlated with one another. There may, perhaps, be an analogous situation in sensorimotor functioning in the limbs. Foerster noted that after a lesion of the postcentral gyrus, the sensory deficit mayuitimately disappear, but a tremor or ataxia remains. Perhaps, some instances of fluent paraphasic jargon may represent the survival of the efferent half of a Wernicke's aphasia.

Kertesz: I don't believe it! I don't know of any case of neologistic jargon with comprehension.

Levine: Kinsbourne and Warrington (Neuropsychologia 1: 27-37, 1963) reported a case of jargon aphasia with preserved comprehension. But the jargon in that case consisted primarily of English words, violating syntactic convention and communicating information very inefficiently. In Alajouanine's (Brain 79: 1-28, 1956) terms, this was paraphasic jargon. The case I just described, he would call undifferentiated jargon. In this case, too, comprehension was preserved. So either form of jargon aphasia may (rarely) occur with preserved comprehension.

Marshall: Broadbent described such a case in the 19th century.

Lecours: Alajouanine et al. (1964) also report poor production

with good comprehension. There is a Marseille paper on a man with a lesion of the ascending parietal gyrus who showed poor production (repetition of a few syllables) yet had good comprehension.

Marshall: What theory are all these observations speaking to? The combination of neologism with comprehension is interesting because it runs counter to Wernicke's original model of fluent posterior aphasics.

Levine: Yes, a dissociation of jargon and comprehension does run counter to Wernicke because he did not distinguish the receptive function and the speech control function of the posterior auditory speech center. Although he allowed that an interruption of auditory input to the left temporal lobe may produce impaired comprehension without paraphasia (pure word-deafness), he did not allow for the reverse dissociation. Yet, jargon can occur without severe impairment of comprehension, as noted above. The neuropathologic bases of these dissociations is still unexplained, but it is likely that the distribution of lesions impairing comprehension and that producing jargon are not congruent.

Marin: There are three situations that are hard to distinguish:
1) Loss of semantic control (e.g., echolalia and dementia);
2) semantic control with restricted articulatory control; and
3) voluntary suppression of semantic control.

Caramazza: In the glossolalic conversation on the videotape, the conversation seemed more fluent than in normals, which makes it even more different from normal language. Perhaps this is because there were no delays in trying to find the right words to express a meaning.

Caplan: Perhaps the conversations are like those we may see in a play. But, presumably, even actors in a play use much of the normal speech apparatus.

Brown: Normal sleep-talkers exhibit the whole spectrum of aphasic symptoms -- including jargon -- in a single night's speech. Could this be a clue to a subcortical role, as Lavorel suggested?

Lecours: My aphasic glossolalic had subcortical areas intact, but very widespread cortical damage.

Kertesz: Jane Holmes had a case of a jargon aphasia following thalamic hemorrhage. But without a good transcript, it's hard to evaluate the data.

Brown: Jargon is rare in vascular lesions or missile wounds in young people. Yet children often produce a type of semantic or neologistic jargon during episodes of sleep speech. Again this raises the question of neural level and/or cognitive state.

Levine: As Teuber pointed out, war wounds give an under-representation of inferior lesions, since few survive the serious

damage involved. Again, war lesions are punched out and more superficial than infarcts.

Lecours: Maureen Dennis reports seeing a 5 year old jargon aphasic in Toronto. In his book on aphasia, Alajouanine uses "jargon aphasia" in a specific manner for a syndrome without comprehension, essentially as a synonym for conduction aphasia.

HISTOLOGY AND ARCHITECTONICS OF LANGUAGE AREAS AND CEREBRAL ASYMMETRIES

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The effects of brain lesions on language, memory, and other so-called higher functions are sometimes unpredictable and confusing. This results in part from the extreme difficulty in obtaining the basic scientific information needed to make useful structure-function correlations in man. Special stains used to study cortical organization, methods to study cortical fiber connections, and electron microscopy so useful in comparative neurology are virtually impossible to implement in human studies. Furthermore physiological techniques in most centers have been limited until recently to the study of the effect of epilepsy on language and behavior, and, upon occasion brain stimulation during surgery, excluding the more sophisticated and accurate methods commonly employed in animal research. With the recent advent of refined and relatively non-invasive neuropsychological, neuroradiological, and physiological techniques, it will be possible to obtain better data on the anatomic localization of basic functions in both normal and lesioned brains. In the meantime most of the evidence on localization and lateralization of functions such as language comes from the investigation of brain lesions by classical neuropathological methods,* and from the application of classical neuroanatomical methods to the study of the organization of the brain. The latter includes the architectonic analysis of the cerebral cortex and the study of fiber pathways using dissection of normal brains and staining for degenerating fibers in brains containing lesions.

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*Footnote - It is safe to state that lesion-derived data on the organization of language in the brain are apt to produce a restricted picture, since it is possible that (1) the lesioning of some language areas may not result in clinically recognizable deficits or the deficit may be reversible ipso tempore, or (2) the presence of other neurological dysfunction, also the result of the lesion, may preclude the discovery of language difficulties.

Animal neuroanatomy may be applicable only insofar as homology of areas can be ascertained. There is also a body of information on left-right anatomical asymmetries in certain relevant regions of the hemisphere which may aid in the understanding of the brain model of language and which adds support to the idea of an anatomical substrate underlying the functional lateralization of the human brain. In this paper we review some of the anatomical and physiological characteristics of the language areas and cerebral asymmetries.

<u>Broca's Region</u> - The exact localization of the lesion causing Broca's aphasia has long been under debate, and to this date it is not clear what actually is the smallest lesion which causes this disorder. On the one hand instances of Broca's aphasia can be seen with relatively small lesions involving the opercular portions of the left frontal lobe, whereas it appears that a much larger area must be lost in order for a permanent deficit to occur (Mohr, 1973). There is no doubt, however, that the lesion must involve at least a major part of the suprasylvian, premotor region (Kertesz, et al 1977; Fig. 1).

(Figure 1 about here)

Clinical neurophysiological experiments offer additional help in defining Broca's region. Aphasic speech arrest can be seen during stimulation of a region encompassing pars triangularis and pars opercularis on the third frontal convolution (Penfield and Rasmussen, 1949). A much smaller area lying within pars opercularis appears to be particularly sensitive to the production of aphasic disturbances after electrical stimulation (Ojemann and Whitaker, 1978; Whitaker, these proceedings; Fig. 1).

A third way to arrive at a definition of the anterior speech zone might be through the discovery of an anatomical marker, e.g., a region of distinctive cytoarchitectonic pattern. Using the technique of pigmentarchitectonics by which lipofuscin granules in neurons are stained, Braak (1978) has found that the brain contains several circumscribed areas characterized by the presence of peculiarly staining large pyramids in layer IIIc. There are such areas in the preoccipital, inferior parietal and temporal lobes (Braak, 1978, and personal communication), and also in the opercular portions of the frontal lobes (Braak, 1979), all of them generally corresponding in location to parts of areas which when lesioned often result in language deficits. In the frontal lobe one of these "magnopyramidal" regions is found in a small portion of pars opercularis, just posterior to the ascending limb of the Sylvian fissure (which separates pars opercularis from pars triangularis; Figs. 1 and 2). In the specimens which have a diagonal sulcus branching from the ascending Sylvian limb the opercular magnopyramidal area is limited anteriorly by this sulcus. (Braak, 1979; Galaburda, unpublished observations; Fig. 1). The significance of these specifically staining magnopyramidal zones is not known. It is interesting to note, however, that the opercular magnopyramidal zone corresponds in location to the physiological zone outlined by Ojemann and Whitaker (1978) and Whitaker (these proceedings) (~~1979~~)/and to the center of the overlapping lesions which result in Broca's aphasia (Kertesz, 1977).* Thus the opercular zone marked by the

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*Footnote - (The May proceedings will be used in part to present data that pigmentarchitectonic asymmetries in favor of a larger left side can be found in the opercular magnopyramidal zone).

peculiar accumulation of lipofuscin granules in its IIIc pyramid may indeed play a special role in language processing and may represent an anatomical pivot point of Broca's anterior speech region.

(Figure 2 about here)

In Nissl preparations the frontal operculum contains three regions which have well developed IIIc pyramids - two in pars opercularis (areas 56 and 57), and one on the dorsal portion of pars triangularis (area 58), (Vogt and Vogt, 1919). Area 56 corresponds in location to Braak's opercular magnopyramidal zone (Braak, 1979; Riegele, 1931). It is not possible to tell which one of these areas is more important for language except that area 56 contains particularly well developed IIIc pyramids. It is likely that all of these areas participate in language function and that a major part of all three must be destroyed for a permanent deficit to ensue.

Data on fiber connectivity might be of additional value if one could demonstrate that areas which are assumed to underlie language functions are particularly interconnected. In man the data point to a general interconnection between the posterior temporal regions and the third frontal convolution (Meynert, 1895), but the methods are not accurate enough to pinpoint the fiber terminations to specific architectonic areas. In rhesus monkey, however, fibers arising from the posterior portion of the superior temporal gyrus, in an area possibly homologous to man's Wernicke's region (Pandya and Sanides, 1973; Galaburda and Sanides, in press), terminate in a portion of prefrontal granular cortex containing large pyramids in sublayer IIIc. (Pandya, Hallett, Mukherjee, 1969; v. Bonin and Bailey, 1947). So far it has not been possible to stain the monkey brain effectively for lipofuscin so as to ascertain whether or not the terminations end in a magnopyramidal region.

Since early in the history of lateralization attempts have been made to explain left hemisphere preponderance in Broca's aphasia. Architectonic comparisons have not been productive (Kreht, 1936) mostly because there have been no good criteria set up to identify Broca's area in cyto- and myeloarchitectonic preparations. The lipofuscin staining method may offer a new opportunity to reassess the presence of architectonic asymmetries in Broca's region. Gross anatomical studies in this region have produced conflicting data, both old (Brauno, 1891; Stengel, 1971) and new (Wada, et al, 1977). An early observation by Eberstaller (1884) may turn out to be the strongest example of gross left-right asymmetry in Broca's region. He found that the ascending branch of the Sylvian fissure is more often branched on the left side. The branch, also known as the diagonal sulcus, serves as a limiting sulcus for the opercular magnopyramidal zone (Braak, 1979) and may reflect a greater amount of folding, therefore more cortex, in the left opercular area. A statistical demonstration of this branching asymmetry needs to be carried out, but has proven to be difficult because of the marked variability in degree of folding in this area in man.

In summary, Broca's anterior speech area remains as primarily a pathological entity defined by a lesion producing Broca's aphasia. Three opercular areas containing well developed IIIc pyramids, and in particular amongst them a circumscribed area in pars opercularis which stains uniquely in lipofuscin preparations, appear to be the likely substrates for the anatomical representation of essential portions of Broca's area. Gross anatomical asymmetries in folding have been claimed to be present in this region but microscopic asymmetries in favor of the left side still need to be uncovered.

Wernicke's Area

The disagreement concerning the exact location of the lesion producing Wernicke's aphasia is not as heated as with Broca's cases. Although aphasic disorders with fluent paraphasic speech and poor comprehension can be seen with lesions over a wide area of the posterior half of the left hemisphere, (Bogen and Bogen, 1976) there is little doubt that in most cases a significant part of the caudal aspects of the superior temporal gyrus and/or their fiber connections are affected (Kertesz, et al, 1977). The superior temporal gyrus and the upper surface of the temporal lobe (the superior temporal plane) house the cortical representation of the auditory system (Fig. 1). Cytoarchitectonically the auditory region consists of a central core of granular cortex (the koniocortex) surrounded by belts of less granular cortex which contain large IIIc pyramids (the parakoniocortices). Medially, separating the konio core from the insular cortex lies a primitive konio field (the prokoniocortex). The anterior belt areas are relatively primitive in appearance and proceed in a stepwise fashion toward the limbic cortex of the mesial temporal regions anteriorly. The posterior belt areas, on the other hand, progressively resemble the cortices of the inferior parietal lobule (for a detailed description of the auditory region see Galaburda and Sanides, in press).

In the posterior temporal region an area known as Tpt (Pandya and Sanides, 1972; Galaburda and Sanides, in press) is found on the caudal most third of the superior temporal gyrus and on the posterior outer edge of the planum temporale (the part of the superior temporal plane lying posterior to Heschl's gyrus, Figs. 1 and 3). This area has cytoarchitectonic features intermediate between the granular auditory belts and the typically parietal cortex found in the inferior parietal lobule (Galaburda and Sanides, in press). Furthermore Tpt often extends from the temporal to the parietal lobe (Fig. 1). The location of this area corresponds to the center of the lesions resulting in Wernicke's aphasia (Kertesz, 1977). Furthermore the location of Tpt matches closely the central portion of the parieto-temporal speech region obtained by electrical stimulation (Penfield and Roberts, 1959). Lipofuscin staining uncovers a magnopyramidal region within the posterior auditory areas similar to that found in the frontal operculum, but the exact relationship to area Tpt cannot be extracted from the literature (Braak, 1978).

Anatomical left-right asymmetries are present in the posterior temporal regions both at the gross and microscopic levels. Asymmetries between the right and left Sylvian fissures, especially at their posterior ends, have been known for nearly one hundred years (Eberstaller, 1884; Cunningham, 1892). The left fissure is longer and more horizontal than the right which also tends to curl upward posteriorly (Fig. 3). This asymmetry can be demonstrated in a high proportion of righthanded individuals, and can be shown in brains of living patients undergoing cerebral angiography (LeMay and Culebras, 1972; Hochbert and LeMay, 1974; Rubens, et al, 1976). In lefthanders the distribution of this asymmetry is different. In particuler there is a greater percentage of brains without asymmetry than in righthanders. LeMay found this asymmetry in fetal life. It can be seen in non-human primates and possibly also in the endocasts of early human forms (LeMay and Geschwind, 1975; LeMay, 1976). Yeni-Komshian and Benson (1976) showed that the left Sylvian fissure is usually longer in the chimpanzee just as in the human.

Another asymmetry in the posterior temporal region was discovered by

Pfeifer (1936) in the planum temporale (Fig. 3). He found that the left planum was larger than the right. He also found that doubling of the transverse gyrus of Heschl's was more common on the right side. The planum asymmetry has since been documented in many other studies (Geschwind and Levitsky, 1968; Wada, 1969; Wada, et.al, 1976, Teszner, et al, 1972; Witelson and Pallie, 1973; Chi, et al, 1977) and the asymmetric duplication of Heschl's gyrus has also been confirmed (Campain and Minckler, 1976; Chi, et al, 1977). The planum asymmetry is striking in favor of a larger left side (65% vs 11% in the series by Geschwind and Levitsky, 1968). It also appears to be present in human fetuses (Chi, et al, 1977), thus virtually excluding a purely environmental explanation for the asymmetry.

(Figure 3 about here)

Although there are other theoretical explanations for the planum asymmetry it appears to reflect an asymmetry in the size of the language area on the two sides. Von Economo and Horn (1930) suggested that a greater amount of auditory association (parakonio) cortex existed on the left side on the planum temporale. Measurements of the full extent of the parakonio fields, both on the planum and their extensions onto the convexity, were made by Galaburda, et al (1978). These authors found that asymmetries exist in cytoarchitectonic area Tpt which correlate well with the planum asymmetry. Other parakonio fields do not appear to show consistent asymmetries on the two sides* (Gelaburda and Sanides, in press). It is noteworthy that area Tpt which occupies a central place in the anatomical representations of language posteriorly in the hemisphere also shows consistent asymmetry on the two sides.

In summary, Wernicke's speech area lies at the caudal end of the superior temporal gyrus and planum temporale which contain granular fields having well developed IIIc pyramids and also intermediate area Tpt. Area Tpt appears to be of particular relevance because of its temporo-parietal structure, its relationship to pathological and physiological data, and its tendency to be larger on the left side.

Other Language Areas

Aphasia may occur with lesions involving the supplementary motor region (Rubens, 1975; Masden, et al, 1978) and the supplementary sensory region (Ross, in press), on the medial aspect of the left hemisphere. The supplementary motor cortex can be distinguished cytoarchitectonically by Nissl (Brodmann, 1909; v. Economo and Koskinas, 1925; Sanides, 1962) and by the lipofuscin method (Braak, 1979). The supplementary sensory region is less obvious in its outline, but appears to correspond to the granular perilimbic field LC, of v. Economo and Koskinas (1925). In man connections of the perisylvian language areas to these medial language zones are not well established, but, in monkey, there is evidence to suggest that homologous areas in perisylvian location send to and receive connections from the medial perilimbic regions (Jones and Powell, 1970; Pandya and Kuypers, 1969; Pandya et al, 1971; Vogt and Pandya, 1978). Some years ago Penfield et al (1959) showed that interference with the normal function of the supplementary motor region can result in aphasia, and more recently studies of regional blood flow have stressed the importance of this region for speech function. (Larsen, et al, 1978; Orgogozo and Larsen, 1979).

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*Footnote - This is a point of interest. On purely anatomical grounds, based on the architectonic and connectional analysis of the temporal lobe in rhesus monkey (Galaburda and Sanides, in progress) area Tpt represents only a step in the trend of cortical differentiation beginning with primitive (proisocortical) moieties. The more primitive cortical regions might be expected to harbor "more primitive" language functions, which in this instance, do not appear to be consistently lateralized to the left side.

In studies of asymmetry, although it has been pointed out that the left sulcus cinguli is more often branches on the left side (Eberstaller, 1884), no other left-right differences are known at the present time.

Fluent aphasias with good comprehension repetition and aphasic naming are often encountered with lesions either in the angular gyrus or in the region between the temporal and occipital lobes laterally (regio temporo-occipitalis, RTO, Fig. 1). RTO encompasses areas PF, PG and PH (v. Economo and Koskinas, 1925) and lie interposed between the temporal parietal and occipital lobes. These areas contain homotopical isocortex (Brodmann, 1909; Vogt and Vogt, 1919; v. Economo and Koskinas, 1925; Sanides, 1970) consisting of evenly laminated granulopyramidal fields. RTO also contains magnopyramidal fields in lipofuscin preparations (Braak, 1978; Braak, personal communication). Taken together with the superior posterior temporal region areas PF, PG, and PH make up a major portion of Penfield's temporoparietal zone (Penfield and Roberts, 1959). The whole temporo-parieto-occipital region contributes fibers travelling in the superior longitudinal (arcuate) fasciculus en route to the prefrontal speech areas (Meynert, 1895).

As already mentioned the Sylvian fissures are particularly asymmetric in the temporo-parietal region. Furthermore, the same posterior region on the left can be shown to be larger by computerized cerebral tomography, thus producing a protrusion of the left occipital lobe into the right (LeMay, 1976; Galaburda, et al, 1978). This asymmetry is present in 64% of righthanded individuals while in ambidextrous and lefthanded subjects the leftsided preponderance is much less (10%). (LeMay, 1976). McRae et al (1968) have reported the presence of a larger left occipital horn (of the lateral ventricle) in 60% of righthanded subjects as compared to only 38% of non-right handers. Although LeMay's and McRae's data are similar for righthanders, they differ in magnitude in the non-right handed population. A possible explanation for the excess of lefthanded subjects having a larger left horn as compared with a larger left occipital region may be that amongst the non-right handers there are some patients who have had an early left hemisphere lesion resulting in left sided atrophy and ventricular enlargement, a situation which would increase the numbers in McRae's series and decrease them in LeMay's series.

Other areas of possible relevance to language function will need to be considered in the future. They are suggested by findings of a variety of speech and language disturbances produced by lesions deep in the frontal parietal, and temporal opercula in the strip surrounding the insula. (Marie, 1906; Meyer, 1950; Geschwind, 1965). In addition to the white matter which may be involved in lesions causing these syndromes (Geschwind, 1965), the peri-insular zone contains primitive motor (Sanides, 1962), somatosensory (Sanides, 1970) and auditory representations. (Galaburda and Sanides, in press). Physiological analysis of this region is difficult to carry out by virtue of its extremely buried location, but auditory functions and aphasic responses have been demonstrated in this site (Celesia, 1976; Whitaker, these proceedings ~~1979~~). Gross left-right asymmetries in the peri-insular zone have not been demonstrated, and architectonic measurements of some of the deeply lying opercular areas do not show consistent left-right differences. (Galaburda and Sanides, in press).

Concluding Remarks

The pathological, anatomical and physiological literature contains sufficient information outlined by classical methods to support the notion that language functions are to a great extent localized to certain distinct areas lying in the vicinity of the Sylvian fissures, the opercular formations and the medial, perilimbic regions of the hemisphere. Furthermore it appears certain that anatomical asymmetries exist in areas closely associated with the language areas. It is also clear that many of these asymmetries are visible from fetal age onward. Such asymmetries may help explain the variability which exists amongst different individuals in the manifestations of brain lesions producing aphasia. Thus patients with marked left sided preponderance in the size of a language area might recover poorly if a lesion destroys the larger side. On the other hand, individuals with more symmetrical brains may be less affected by unilateral lesions. Support for this claim may be obtained from the study of lefthanded aphasics. The relatively mild aphasias in these patients (Hécaen and Ajuriaguerra, 1964) may reflect the fact that findings of asymmetry are less striking in this population (Hochberg and LeMay, 1974; LeMay, 1976; Witelson, 1979).

The clearer understanding of normally occurring asymmetry may conceivably be helpful in diagnosing disease states in which normal asymmetry may be altered. Children with delayed speech for instance show an excessive incidence of reverse asymmetry in the occipital lobes, and the type of asymmetry they demonstrate can be correlated with tests of intellectual function. (Hier, et al, 1978). Since the diagnosis of occipital asymmetry is accessible to non-invasive radiological tests, this information can be useful in managing these patients.

Language competence in prehistoric ancestors of modern man and in living non-human primates may be suggested by findings of cerebral asymmetry in the Sylvian fissure (LeMay, 1976). One must first determine, however, whether or not the homologous areas showing left-right asymmetry are capable of carrying out the same function in the different species.

Summary

The human cerebral cortex contains several regions which, when damaged in the left hemisphere, often result in disorders of speech and language. Some of these regions have architectonic areas of striking structure and location to suggest a common role in language function. Furthermore, animal experiments point out that these areas have rich fiber interconnections, which, by a different method, emphasize their likely common function.

Asymmetries have been demonstrated in the human brain in gross cortical structures in some of these language areas and, in some cases, architectonic asymmetries can be shown to parallel the gross left-right differences in size. Asymmetries in these and other areas may be used to help explain individual variation in incidence, severity, and recovery from acquired aphasia and conceivably may help to account for the differences in performance in children with developmental language disabilities. Furthermore, the study of language capability in primitive humans and in non-human primates and even in lower species may proceed partly through the analysis of their cerebral asymmetries.

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Figure Legends

Figure 1

Schematic representation of the left human cerebral hemisphere. The Sylvian fossa, which is bordered by the Sylvian fissure (s), has been opened to show the opercular portions and the insula (Ins). The anterior speech area (interrupted horizontal lines) occupies roughly the pars opercularis (PO), the posterior half of the pars triangularis (PT) and the anterior portion of the subcentral region (lying below the central sulcus (c). A magnopyramidal zone (m),which corresponds closely to an area of greatest physiological sensitivity, is found on the anterior half of PO, posterior to the Y-branching ascending limb of the Sylvian fissure.

The auditory region on the superior temporal gyrus (GTS) and the superior temporal plane which includes Heschl's gyrus (h) and the planum temporale (PT) contains a central core of primary cortex (black) surrounded by association belts (large closed circles). Posteriorly Tpt is found (open circles). Another magnopyramidal zone (m) is found here. The supramarginal (GSM), angular (A) and temporo-occipital regions (RTO) contain homotypical areas PF, PG, and PH, which also have language function.

Figure 2

Photomicrograph of the central opercular magnopyramidal zone. In this cortex laminas IIIc and IV overlap considerably. Note the lightness of the stripe corresponding to lamina Va. A similar poverty of staining is encountered in other cortices in laminas IIIc and IV. In this cortex, however, the external light lamina is populated by richly staining large pyramidal neurons, the hallmark of all magnopyramidal regions.

Figure 3

Above. Left and right hemispheres showing the typical Sylvian asymmetry (arrowheads). Note that the right Sylvian fissure is shorter and curves upward posteriorly.

Middle. The Sylvian fossae have been opened to show the superior temporal plane with Heschl's gyrus (H) and the planum temporale (PT). Note the difference in size of PT on the two sides.

Below. Myelin stained frontal section of the brain to show the difference in height of the Sylvian fissures on the two sides (white arrows). Also note the increased folding of the planum temporale (PT) on the left side.

Fig. 1

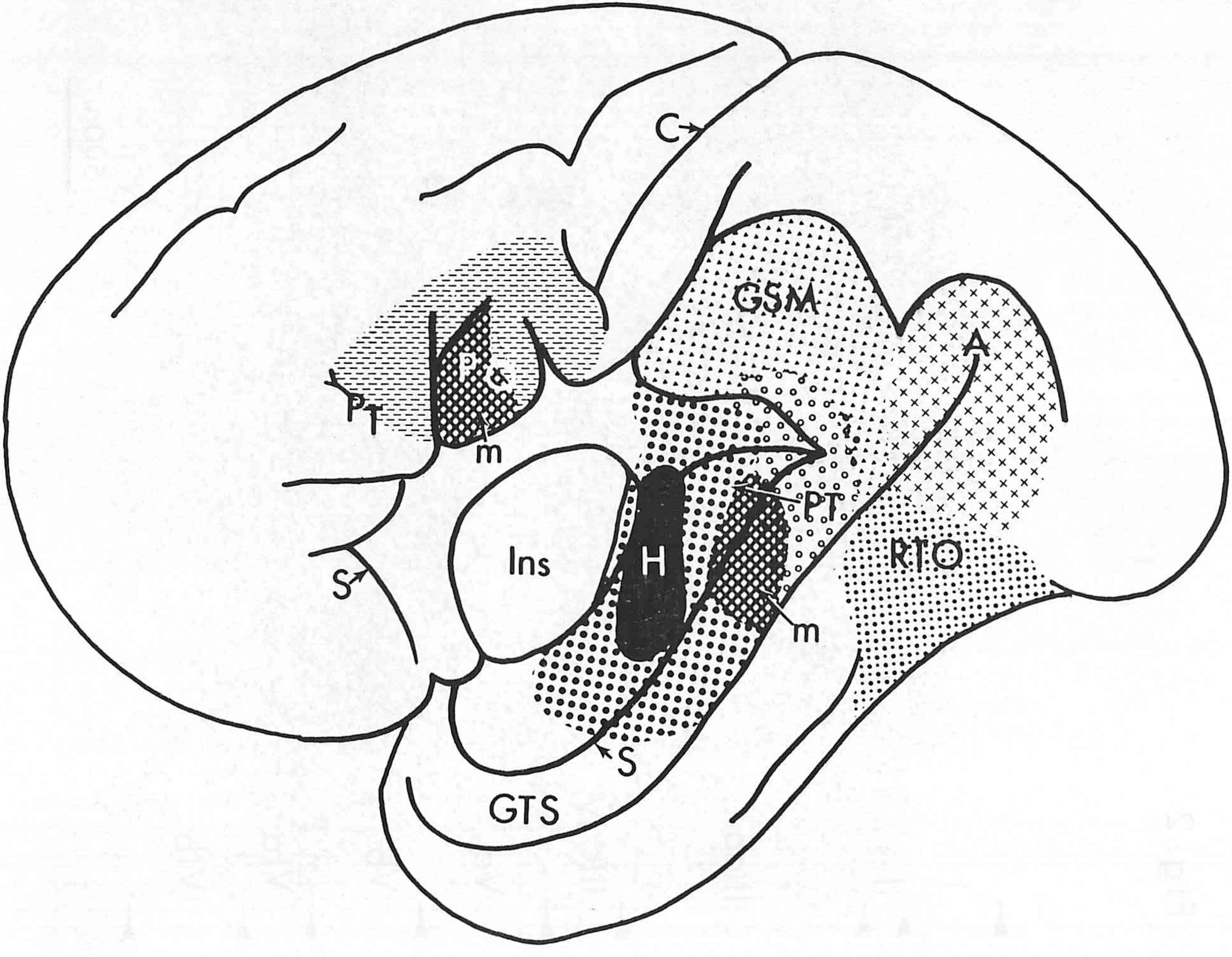


Fig. 2

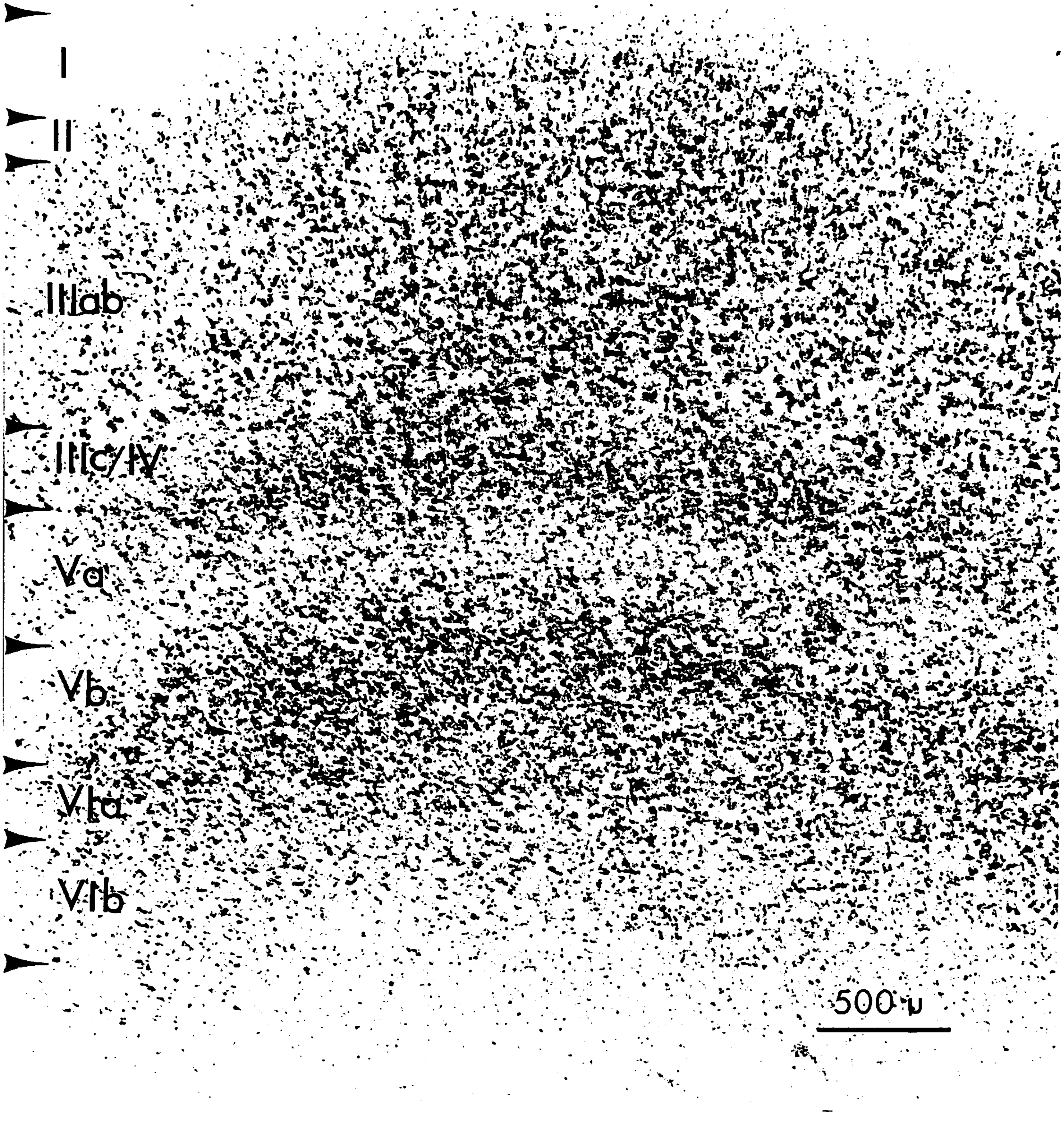
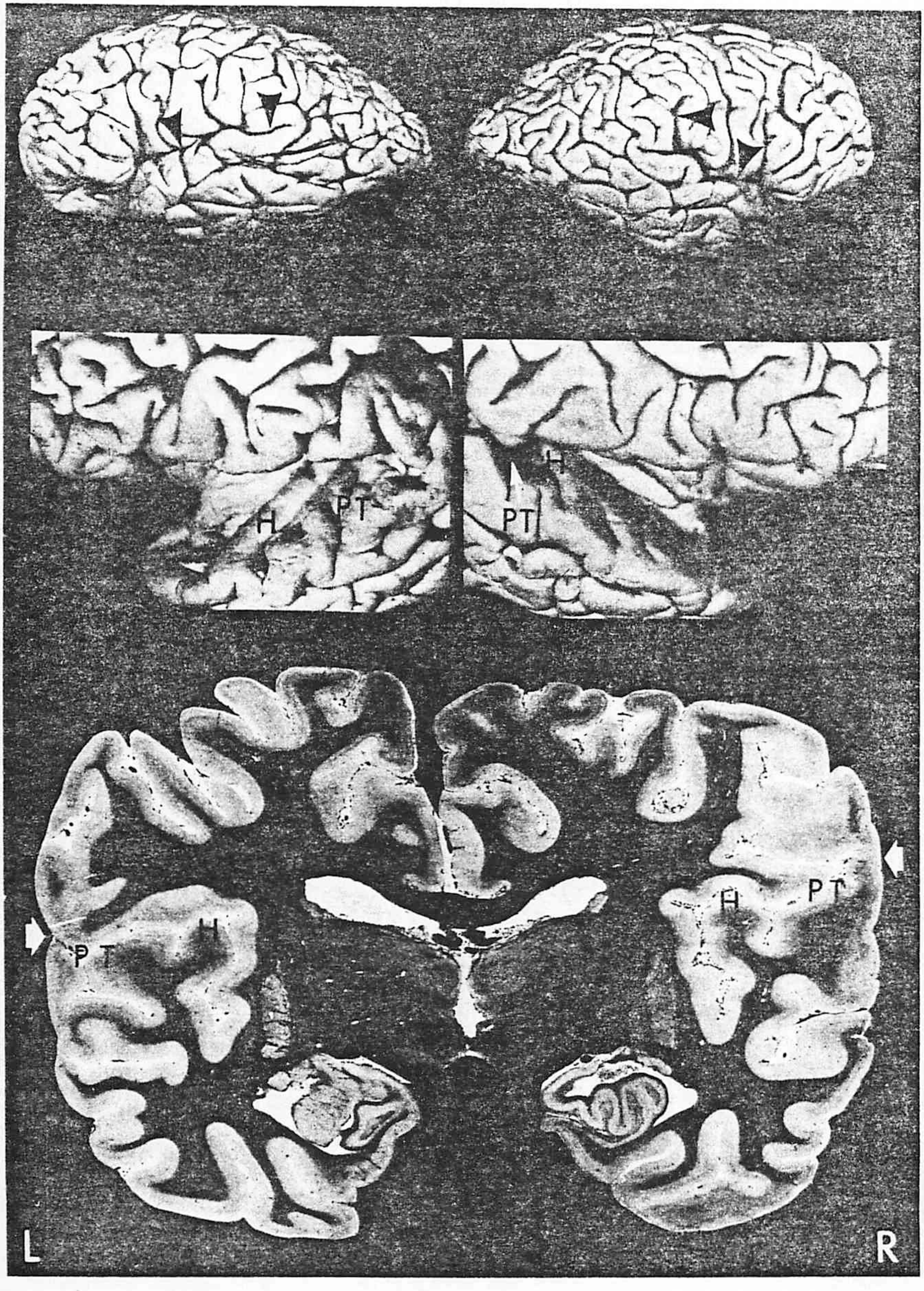


Fig. 3



H. WHITAKER: ELECTRICAL LOCALIZATION AND NAMING

The classic data of Penfield and Roberts have their limitations. The data were pooled with an average of 1-1/2 stimulations per patient. Techniques changed over the 20 or 30 years that the observations were accumulated. And Penfield omitted a number of sites he felt were not central.

I persuaded Ojemann to make a wide range of stimulations of brains exposed for excision of epileptic foci. We stimulated "healthy areas" of the epileptic brains, and found over 50% of left cortex implicated in language function.

About 25% of epileptics do not respond to chemical treatment, and a tenth of these come to surgery, which is remarkably successful. The patient is alert during the operation. During the first two hours, the skin is folded back from the skull, and a bone flap is removed. The dura mater is turned back to expose the brain. The capillaries become engorged with blood, which seems to impair brain function. At this stage, it is not clear which brain region is which -- there are no labels! -- or where the epileptic focus is. The surgeon places silver ball electrodes on the brain, and looks for spikes in the EEG to determine the epileptic area, which is marked with lettered tickets. Next, he must map the language area -- using numbered tickets -- to avoid damage to it when resecting the epileptiform cortex. For this test, the surgeon uses bipolar electrodes, setting the current to be very small. (The threshold

for disruption of naming seems to be below the threshold for sensory experience, e.g., tingling of a tooth.)

80 slides are made of objects that the subject can name perfectly -- both as tested before the operation and on the day after the operation (before edema-caused mild disphasia sets in). The surgeon stimulates a point of cortex to check for impairment of naming. He waits for recovery before stimulating again -- at a different site. On this basis we mapped sites as follows:

Solid black circles: failure to name but ability to speak every time. The patient can read "this is a" from the slide, but cannot produce a name for the object pictured on the slide. This condition is different from misnaming, paraphasia, or perseveration.

Striped circles: intermittent failure to name

Stippling: motor cortex (usually, but not always, the classic homunculus in the Rolandic area).

White circles: no errors

Asterisk: naming errors, at about the normal error rate.

The surgeon can safely excise the areas which don't abut black circles without causing aphasia (in the one exception, the aphasia was only transient).

Different patients exhibit quite different patterns. One patient gave us permission to map the insular cortex and we got naming errors there. A woman with a large cortical scar had the rolandic cortex distributed across the parietal lobe, bilateral language function and, apparently, no "black circles". In another patient, the homunculus was upside down. This shows how far the brain can reorganize (or depart from the conventionally held organization). No wonder there are controversies about localization.

We had two bilingual patients. One spoke English and Spanish. The sites for blocking naming were not the same. She had learnt both languages as a child. She had the right hemisphere dominant for language. Another patient spoke English and Dutch, having learnt English as a teenager. He had the same sites for blocking of naming for both languages. Unfortunately, the sample is too small for us to draw general conclusions.

The one area implicated in naming for all subjects was Broca's area "a", the pars opercularis. There were effects in some but not all subjects in Broca's area "b", pars orbitalis and pars triangularis.

DAVID CAPLAN: COMMENTS ON THE GALABURDA AND WHITAKER PAPERS

There are three stages in our understanding of the neural basis of a function: The "where" of gross localization, the detailed microscopic anatomy, and the physiology. Whitaker uses physiological techniques, but addresses the first question: not *how* naming is accomplished, but *where* there is involvement. This provides a fine grain accompaniment to such cruder but non-invasive techniques as recording regional blood flow,

studying evoked potentials attendant on speech, or using deoxyglucose as a marker of metabolic activity of neurons. All these will extend our notion of where language "is" in the brain.

In his written paper, Whitaker emphasizes the spatial arrangement within the naming area. He sees widely distributed "islands" for naming, with *graded* effects, and asks if these are a macroscopic extension of columnar structure. There is a pleasing lesion-stimulation agreement -- the one area common to all patients seems to coincide with Galaburda's area of magnocellular pyramids in layer IIIc of cortex.

There are some questions and reservations about the Whitaker-Ojemann study. What is the effect of fronto-temporal epilepsy upon the areas involved in naming? Epileptic foci can wander from lobe to lobe and hemisphere to hemisphere, presumably altering neuronal function in many areas of brain. We know that for at least some early lesions, major re-organizations of language in brain result, even to the extent of change in cerebral dominance for language. Thus, the implications of these studies for normal cerebral representation of naming are weakened. Perhaps the most interesting result, and one which may be immune tothese reservations, is thefinding that naming is a graded function locally.

Similar qualifications apply to the implications for normal second language representation in brain. The Dutch bilingual had a frontal lobectomy before learning English, and the Spanish subject was right-hemisphere dominant — both quite atypical situations.

Galaburda may well be offering us the first proposal of a cell-type marker for the language areas of the brain. But has he identified the right areas? (cf. Levine's paper and the controversy it generated.) An area which is always lesioned when there is a Broca's aphasia is not necessarily an area whose lesion yields a Broca's aphasia. If a lesion localized to pars opercularis yields only transient aphasia, what is the histology of the area that takes over? And if a much greater lesion seems required for persistent aphasia (cf. J. Mohr's CT-scans) what of the histology of the areas implicated in the Rolandic strip and the parietal operculum?

Neuroanatomy has been our basic source of knowledge of where a lesion produces a disorder. But we know very little about *how* the lesion achieves that effect. It seems to me that we cannot jump immediately from the gross functional level to the detailed anatomical level of the histology. We need physiology to bridge the gap. As Arbib and Caplan (1979) argue, the analysis of neural nets seems to be the fruitful path to the interpretation of histology.

DISCUSSION OF THE GALABURDA AND WHITAKER PAPERS

Marin: A tiny lesion in Area 17 produces a well-located scotoma, and a similarly small lesion in auditory cortex yields an audiologically measurable effect. In discussing aphasia, we too often talk as if all effects were the same. But a tiny opercular lesion yields devastating effects in articulation, while I would not expect a small lesion to yield agrammatism or a specific anomia.

Levine: Lesions of left pars opercularis have occurred in right-handed people with no speech deficit that was clinically noted. This cannot be attributed to the take-over of speech by the right hemisphere, since there exist bilateral lesions of the entire pars opercularis which yielded mild dysnomia but no signs of Broca's aphasia. As I argue in my paper, the left precentral gyrus seems to be the one area always involved in Broca's aphasia. I don't believe there are distinct areas for determining the articulatory program and for executing it. I think these functions are congruent and both require the precentral gyrus.

Whitaker found pars opercularis stimulation most consistently blocked naming without speech arrest. He discarded precentral gyrus data, where stimulation blocked *all* verbal behavior — reading of the test phrase and naming of the object. Now, isn't reading a test phrase also a visual naming task, as is naming an object? If so, the precentral gyrus may be more, not less, implicated in visual naming. One cannot just throw out the data because the effect of stimulation may be "purely motor". In fact, our studies of patients with lesions of the precentral gyrus indicate that reading comprehension is impaired even when no overt speech output is required.

Hudson: Whitaker's pictures show us the great variability in localization between individuals. I think we need a sort of 'topological transformation' to bring the different maps into correspondence.

What can the study of granular/pyramidal cell ratios in the brain tell us about function? Harking back to Wood's neural net model, we could change excitability, rather than lesioning, particular subregions. But how can we go from this lesion of analysis to an *interpretation* at the level of psycholinguistics, e.g., that STM limitations affect the quality of parsing?

Woods: Could someone please lay out the basic anatomy for the non-neurologists. How does information get from the cochlea to Broca's area? Where does it go from there? What can we say about the speech/hearing loop?

Levine: The detailed anatomy can only be inferred from studies of non-human primates such as, for example, Jones, E.G. and Powell, T.P.S. "An anatomical study of converging sensory pathways within the cerebral cortex of the monkey." Brain 93: 793-820, 1970. Auditory input from both ears arrives at each primary auditory area in Heschl's gyrus. From here, activity projects mainly locally to the surrounding auditory association cortex. Connections from there reach the frontal lobe, including the third frontal gyrus, but not the precentral gyrus.

There is thus a real problem for those of us who believe the precentral gyrus to be an important part of the language area. If so, it should receive abundant auditory and visual input. But most studies do not find such input, at least directly.

Lavorel: Many explanations are based in terms of where programs are stored. But what of "compiling", assembling sequences as needed? Can we get a language of "hyper-concepts" and "hyper-notions" avoiding undue use of predefined structures?

Arbib: The "planning" in the discussion of syntax and translation in my paper is a sophisticated form of compilation.

Galaburda: What makes one think that a particular area will perform differently from another? Different stains give different information. Maybe the biochemistry reflects physiological differences in the cell. It worries me that my lypofucsin stain may connect too well with the data on aphasia.

Whitaker: Most of the patients we studied as adults had epilepsy by age 3, so that we can expect some brain reorganization. Perhaps some of the areas we study are potential recruits for language in the normal brain.

Our bilingual patients were indeed atypical. Penfield had many bilingual patients, but made no usable observations on their bilingualism.

I like to look at stimulation as a small reversible lesion which gives the brain no time to reorganize. [Editor's query: How can a local "lesion" block naming ability in toto?]

The 1979 *Science* article by Ojemann and Mettier suggests a central core with two concentric areas around it. This may offer a convergence with Jason Brown's conceptual framework as well as with Galaburda's histological data.

The Neuropathologic Basis of Broca's Aphasia and Its Implications for the Cerebral Control of Speech

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Sudden loss of speech, associated with right hemiplegia, is a very common syndrome, and there are few neurologists who have not seen many such cases. The speech loss, which may be total at first, undergoes a variable degree of improvement over the subsequent days and weeks. Some patients regain no speech, but may moan or cry out to attract attention. Others regain the use of a limited repertoire of neologisms, words, and short phrases that are used in a stereotyped and perseverative manner to respond to questions. These phrases may be enunciated clearly with no significant articulatory difficulty. When speech becomes somewhat richer, repetition of single words is often far more successful than uttering these words in naming or in spontaneous speech. But repetition too breaks down beyond the single word or phrase. Still other patients regain even more speech, but it is uttered slowly and effortfully, often in a monotonous measured pace, with frequent stumbling over words and misarticulation. In patients with this degree of recovery, verbal paraphasias are common. Syntax may also be abnormal, as words carrying little semantic and/or speech emphasis may be omitted, leaving a bare bones or telegram-like output. Finally, some patients recover normal, or nearly normal speech, only occasionally misarticulating slightly or pausing to find a word.

Early workers emphasized that despite lack of speech, use of the oro-lingual musculature for non-speech acts was entirely normal. It is now clear, however, that the overwhelming majority of these patients also suffer from inability to utilize lips, tongue, and pharynx in a variety of voluntary acts other than speech. In the first few days of the illness many such patients have difficulty swallowing their food and saliva, but this dysphagia usually recovers very promptly. Severely affected, speechless patients may be unable to protrude their tongue, either to verbal request or in imitation of the examiner. Less affected patients, such as those uttering some single words or phrases, may protrude the tongue well, but are slow in moving it from side to side. Even when this can be

done at reasonable speed, acts such as whistling or clucking are poorly done. In a population of these patients the degree of speech loss is highly correlated with the degree of such oral apraxia (De Renzi et al., 1966).

Writing with the unparalyzed left hand is almost always impaired. Spontaneous writing or writing to dictation is usually more impaired than copying, but severely affected patients may have trouble with all of these tasks. The correct letters may be poorly formed, incorrect letters appear with frequent perseverations, and letters may be written atop one another or at uneven heights.

Language comprehension is also affected, but to a variable degree. In general, responses to spoken requests are poorer than imitation of the examiner's movements, whether these be oral or limb gestures. Thus, the patient may not open his mouth or touch his nose to spoken request but may do so in imitation of the examiner. Not all visual input, however, enjoys such favorable status. Comprehension of written language is usually as impaired as, or more impaired than, comprehension of speech. The same patient who sticks out his tongue to spoken request may be unable to obey the printed command of "STICK OUT YOUR TONGUE". This impairment of comprehension is manifest not only in tests requiring the patient to obey commands, but also in tests requiring matching of spoken or written phrases to appropriate pictures and in tests requiring only binary (yes - no) decisions about spoken or written questions. Such comprehension deficits involve not only ordinary language but also other semantic systems such as that of numbers and arithmetic operations.

The designation of this common aphasic syndrome has had a long history. Broca (1861) called it "aphemia", but Trousseau (1864) argued for "aphasia". When Wernicke (1874) contrasted these aphasics with others who spoke copiously, this aphasia became "cortical motor aphasia" or Broca's aphasia. "Expressive" (as opposed to receptive) and "non-fluent" (as opposed to fluent) were terms introduced by later authors.

The neuropathological basis of the syndrome has also had a long history, punctuated by polemical attacks and counterattacks. Broca localized aphemia to the third frontal convolution of the left hemisphere. (Fig. 1) He felt that this region contained the engrams (or programs) for the learned movements that constitute speech. During Broca's era, the prevailing view was that movements could not be elicited by mechanical or electrical stimulation of the cortex, and so the notion of "motor cortex" did not exist. In 1870 Fritsch and Hitzig overturned that view by demonstrating that electrical stimulation of the cortex of the dog could produce various movements. Subsequently, Wernicke (1874) identified Broca's area with that portion of the cortex from which electrical stimulation yielded movements of the speech musculature.

As the anatomy of motor cortex in man became established, however, it soon became clear that the third frontal gyrus was <u>not</u> the motor cortex for the muscles utilized in speech. Instead the motor cortex straddled the Rolandic sulcus, with the lowest thresholds in the precentral gyrus and slightly higher thresholds in the postcentral gyrus. Faced with this situation, Dejerine (1914) and Liepmann (1915) developed a model that has had a tenacious hold on the minds of most students of aphasia ever since. According to this view, the third frontal gyrus (pars opercularis plus varying amounts of pars triangularis depending on whom you read) contains the motor programs for speech. Neurons in this area drive neurons in the precentral gyrus, which is a motor "executor" area, from which axons reach the lower cranial nerve nuclei that innervate the speech musculature. Accordingly, lesions of the motor cortex were thought to produce contralateral facial and lingual weakness and to impair speech output but to leave "inner speech" intact. The patient could thus comprehend speech and print and might write normally if not hemiplegic from extension of the lesion higher or deeper into the precentral gyrus. In contrast, lesion of Broca's area was thought to result in loss of the motor programs for speech, but not necessarily in paralysis of oro-lingual musculature for non-speech movements. However, the loss of motor speech programs could have repercussions on other language activities, producing the clinical picture of Broca's aphasia described at the outset of this paper: speechlessness with recurrent utterances or agrammatism, agraphia, variable impairment in speech comprehension, and often severe alexia.

The purpose of this paper is to examine the evidence for this model. The primary source of this evidence will be neuropathologic studies of patients with Broca's aphasia and of patients with focal lesions of Broca's area. We will also, however, examine some evidence from electrical stimulation of cortex in waking man and some evidence from studies of comparative anatomy. We shall conclude that there is little evidence to support the classical model, and shall tentatively propose a different formulation.

I. <u>Evidence from Neuropathological Studies</u>.

Pierre Marie, at the turn of the century, posed the first serious challenge to the classical model. His evidence was assembled and presented in great detail by his student, Moutier (1908), and comprised largely cases of cerebral infarction documented by post-mortem examination. Later, Marie and Foix (1917) studied the aphasias resulting from missile wounds incurred by soldiers during World War I. In this study, post-mortem examination was not usually done, but the large number of cases and the apparent consistency of the wound sites within a given category of aphasia allowed strong conclusions about localization. Later, Niessl von Mayendorf (1926) again summarized the evidence from early cases of cerebral infarction and added new cases, reported after Moutier's publication. The evidence from traumatic aphasia during war was again vigorously pursued during and after World War II by several investigators (Nathan, 1947; Schiller, 1947; Conrad, 1954; Luria, 1970). Finally, the development of scanning techniques, first radioisotope scanning and later computerized tomography, has allowed the large scale study of cases with cerebral infarction, even when post-morten examination cannot be obtained (Benson and Patten, 1967; Kertesz et al., 1977;

Mohr et al., 1978).

Evidence against the classical model, derived from these studies, comes from both "negative" and "positive" cases. A negative case is one in which a lesion occurred in Broca's area but either no aphasia developed or the aphasia was unlike Broca's aphasia. A positive case is one in which Broca's aphasia was present but the lesion completely spared Broca's area. Marie appreciated the need for both types of evidence and collected each kind of case.

A) "Negative" cases. The literature contains several examples of right-handed patients with infarctions of the left third frontal gyrus who were either never aphasic or who developed transient mild aphasia. Niessl von Mayendorf (1926) presented summaries of 12 such cases, all with infarctions involving the pars opercularis and varying amounts of pars triangularis and pars orbitalis of the left third frontal gyrus. The topography of the lesions in four patients who were never aphasic is shown in Fig. 2.

Recent evidence from computerized tomography shows that lesions limited to the third frontal gyrus, may show only transient, mild aphasia (Mohr et al., 1978) or no aphasia at all. An example of such a negative case from our own material will illustrate this clinico-pathological relationship.

A 50 year old right-handed hypertensive man (PM 230-90-86) had a convulsion with loss of consciousness. He awoke within an hour or two and was confused but not aphasic. After several further seizures that evening, an endotracheal tube was inserted. C.T. scan on the following day is shown in Fig. 3. On his third hospital day he was extubated. Examination showed confusion but no aphasia. Asked where he was, he said, "I am at the airport...in a hospital." He subtracted serial 2's from 10 without error. Detailed psychological testing during the following week revealed deficits in intellect and memory affecting both verbal and non-verbal tasks. Concreteness of thought, difficulty in shifting mental set with perseverations and impaired concentration were evident. There was no aphasia.

The occurrence of such negative cases was at least in part realized by advocates of the classical model, who attempted to reconcile this evidence with their view that the third frontal gyrus contained the motor programs for speech. The most common such explanation was that in such patients the inferior frontal gyrus of the <u>right</u> hemisphere took over the programming of speech, resulting

in either no aphasia at all, or only transient aphasia.

Recently, we had the opportunity to study a patient who allowed us to test this hypothesis directly (Levine and Mohr, 1979 - Case 1). In this patient only mild dysphasia was present even though the third frontal gyrus was destroyed bilaterally.

A 65 year old man developed sudden mutism and right hemiplegia. The limb weakness improved in 3 days, but speech did not return for several weeks. For a while, he spoke with single words and numerous verbal paraphasias. After several months, his speech was "nearly normal". One year later he again became mute and was unable to swallow. Within hours, however, his speech returned to its previous state. However, severe personality changes developed. He developed bizarre delusions of persecution and became pathologically compulsive. He displayed little affect except for brief outbursts of anger, and he seemed emotionally distant. Examination revealed moderate dementia with marked concreteness and slowing of thought, poor reasoning and memory, and difficulty with visuospatial tasks. Yet his speech was fairly well preserved. He rarely spoke spontaneously except for an occasional complaint. But he answered readily when addressed, speaking in short phrases. His voice was a husky, breathy monotone, and occasionally sounds were deleted or blurred. But his speech was intelligible and appropriate, as in this conversation:

"What kind of work were you in?"	"Oil burner work."
"What did you do?"	"Install the burners."
"Anything else?"	"Made em run."

His repetition of speech and reading aloud were nearly normal except for mild dysarthria and dysprosody. Naming of visual objects and of objects described verbally was slightly impaired. Speech comprehension was moderately deficient; he could perform two-step commands but not three-step commands. Reading comprehension was impaired only for difficult material. His profile of scores on the Boston Diagnostic Aphasia Examination is shown in Fig. 4. Thus, language production, although not normal, was only mildly to moderately impaired. There certainly was no evidence of the syndrome of Broca's aphasia. His C.T. scan (Fig. 5) showed bilateral low density lesions, consistent with infarction, involving primarily the frontal lobe on the left and frontal and temporal lobes on the right. Broca's area was completely destroyed or undercut bilaterally.

In searching the world's literature for other cases with bilateral lesions of Broca's area, we found six reported cases, all of whom spoke or wrote sufficiently well to exclude them from the category of severe Broca's aphasia (Levine and Mohr, 1979). Recently we encountered another case, reported by Gianulli in 1908, and reviewed by Niessl von Mayendorf (1926).

The patient was a 74 year old man who had two strokes. Examination showed dementia. Speech was paraphasic ("pristo" for "tristo") and fluent. Repetition was also paraphasic. Speech and print comprehension were preserved for simple requests but were not normal. Post-mortem examination showed atrophic, cavitated lesions in the posterior portions of the third frontal gyri bilaterally (Fig. 6).

Cases such as these demonstrate that even bilateral lesions of the third frontal gyrus may result in only mild aphasia. Yet the classical model - even the variation that allows for substitution by the right hemisphere - would predict that such patients should be unable to speak at all because the "motor programs" for speech are no longer present.

This evidence poses a serious challenge to any theory which assigns to the third frontal gyrus a dominant role in speech. We must now ask what cerebral structures are crucial to speech, if not the third frontal gyri? To answer this question we are required to examine the evidence from positive cases.

B) "Positive" cases. Marie and Moutier (1908) presented neuropathologic findings in a series of patients with Broca's aphasia. They concluded that patients with Broca's aphasia invariably had lesions in a zone bounded anteriorly by the anterior margin of the insula and posteriorly by the posterior margin of the insula (Marie's lenticular zone - Fig. 7). The superior-inferior borders and medial-lateral borders of this zone were never clearly established. This zone is situated posterior to the pars orbitalis and pars triangularis of the third frontal gyrus, which were often entirely intact. The "lenticular zone" contained the claustrum, lenticular and caudate nuclei, the thalamus, and the internal, external and extreme capsules. Cortical components included the insula, fronto-parietal operculum, and lower portions of the precentral and postcentral gyri.

The crucial structures within the lenticular zone were never fully elucidated by Marie and Moutier. Many patients had infarction or hemorrhage in the putamen and in the adjacent internal or external capsules. Although Marie often seemed to emphasize the importance of the putamen, this conclusion must be suspect because involvement of white matter lateral, medial and superior to the putamen must have interrupted projection fibers to and from the inferior and

opercular fronto-parietal cortex as well as commissural and intrahemispheric association fibers. Indeed, in some cases the putamen was spared, but the lesion involved the inferior portions of the precentral and postcentral gyri and varying portions of the posterior parietal lobe. In his later studies of traumatic aphasia with Foix, Marie concluded that extensive cortical lesions, centered on the inferior pre- and postcentral gyri, produced global, or Broca's aphasia. Niessl von Mayendorf (1926), reviewing his own experience and the literature of Broca's aphasia resulting from stroke, concluded that the true Broca's area "coincides with the central projection of those muscles active in speech, an area which can be determined by electrical stimulation of the brain." Broca's area and motor cortex were not distinct, but identical.

The identity of motor cortex and Broca's area, first postulated by Wernicke, but later denied by Dejerine and by Liepmann, has been further supported by studies of traumatic aphasia in soldiers of World War II. Nathan (1947) concluded that the most common localization of facial-lingual apraxia, affecting both speech and non-verbal voluntary movement, was the lower pre-central gyrus. His patients had deficits of language comprehension as well, although the speech difficulty was most conspicuous. Schiller (1947) found that lesions resulting in articulatory difficulty centered on the lower precentral gyrus. All of his cases showed other aphasic disturbances such as impaired naming. The extensiveness of the aphasia was strongly related to the degree of tissue loss within the perisylvian region. Conrad (1954) showed that wounds resulting in motor aphasia - whether Broca's aphasia or pure word-muteness (subcortical motor aphasia)-centered around the middle and inferior portions of the central sulcus, not the third frontal gyrus. "...Practically all foci are situated within the area of the enlarged motor fields of the cortex". The severity of the motor aphasia was related to the size of the lesion.

The results of radioisotope and tomographic X-ray scanning of lesions are consistent with the pathoanatomical evidence supporting the location of Broca's area in the motor cortex itself. Kertesz (1977) found large radioactive uptake over the precentral gyrus in all of his fourteen cases of Broca's aphasia. Using C.T. scans, Mohr et al. (1978) have stressed that only large perisylvian lesions produce severe, permanent speechlessness. Levine and Mohr (1979) have stressed that, within this perisylvian region, the most critical region is the precentral gyrus rather than the third frontal gyrus.

These points can be illustrated by two of our most recent cases. The first case (Trojanowski et al, 1980) was a patient with crossed aphasia, i.e. he developed a typical severe Broca's aphasia with a left hemiplegia. He recovered no speech except for one or two recurrent utterances over a nine month period. On post-mortem examination, the only major lesion was in the right hemisphere, verifying that this right-handed man had indeed developed speech, for an unknown reason, in the right hemisphere. Of interest to us, however, is that his lesion (Fig. 8) was nearly entirely confined to the cortex and immediately subjacent white matter of the precentral gyrus. It involved nearly the whole extent of this gyrus, but hardly encroached on the frontal gyri anteriorly or the postcentral gyrus posteriorly. Thus, it appears that (at least in this patient) extensive lesion of the dominant precentral gyrus is sufficient to result in Broca's aphasia.

The second case is a 70 year old man who developed sudden speechlessness with right hemiplegia. Six months after his stroke he has recurrent perseverative utterances but is unable to communicate. He repeats only single words or a short phrase. Moderate deficits in speech comprehension and severe alexia are present. There is some oral dyspraxia. His C.T. scan (Fig. 9) shows a focal lesion involving the lower third of the pre-and post central gyri. Only slight encroachment on the third frontal gyrus and possibly on the anterior portion of the superior temporal gyrus is present.

In summary, the overwhelming bulk of the neuropathologic evidence seems to indicate that:

1) The third frontal gyrus - including the pars orbitalis, pars triangularis, and even the pars opercularis can be destroyed in the dominant hemisphere and even bilaterally with only mild dysphasia. Bilateral lesions may result in diminished spontaneity, disorders of affect, and dementia, but do not produce Broca's aphasia.
2) Broca's aphasia is characterized by a deficit in speech output ranging from speechlessness through recurrent, stereotyped utterances, to agrammatic and/or poorly articulated speech; by variable deficits in speech comprehension; and often by severe alexia. It usually results from extensive lesions of the inferior fronto-parietal cortex, including the third frontal gyrus, precentral and postcentral gyri, and inferior parietal lobule.
3) In this extensive area, it appears that the lower half of the precentral gyrus is the most critical cortical region. In at least one of our patients, an extensive anatomically verified lesion of the precentral gyrus alone resulted in severe, permanent Broca's aphasia. Destruction of this region or of its callosal and subcortical projections was the one finding invariably encountered in all of our own cases and in those reported in the literature.

II. Anatomic and Physiologic Studies.

The neuropathologic evidence that we have reviewed strongly supports a crucial role for lesions of the left precentral gyrus in the production of Broca's aphasia. In this section we shall review some of the neuroanatomic and neurophysiologic knowledge bearing on the function of this cortex, particularly its relationship to speech.

The cortex of the precentral gyrus in man and in monkey, consists of two major cytoarchitectural and myeloarchitectural types, both of which are agranular. Area 4, myeloarchitecturally astriate and containing Betz cells in layer V, occupies the anterior bank of the Rolandic sulcus and extends forward onto the convexity of the precentral gyrus, less so in man than in monkey. Immediately anteriorly, occupying the remainder of the precentral gyrus, is area 6, which is unistriate and contains no Betz cells.

Pandya and Kuypers (1969) and Jones and Powell (1970) have studied the cortico-cortical connections of these regions in the macaque. Areas 4 and 6 are reciprocally connected. Area 4 is also reciprocally connected to the cortex of the postcentral gyrus, while area 6 is reciprocally connected to area 5 of the superior parietal lobule, itself a recipient of heavy projections from the postcentral gyrus. Thus, areas 4 and 6 of the frontal lobe can be considered parts of a more widespread, heavily interconnected system, situated in the fronto-parietal region. The system also includes at least Brodmann's areas 3, 1, 2, and 5.

This system appears to function primarily in relation to movement and somatic sensation. The somatotopic motor mapping of the contralateral body-half on the precentral gyrus (MI) is well known. Similar motor maps are present in the postcentral gyrus (SI), in the medial surface of the superior frontal gyrus (MII - supplementary motor area) and in the parietal operculum (SII). (Fig. 10) The threshold for movement by electrical stimulation is lowest in the precentral gyrus. It is of interest that the loci for tongue and mouth movements in three of the four motor representations (MI, SI, SII) are nearly contiguous, occupying the inferior portions of the central gyri. It must be emphasized, however, that such maps are potentially misleading. In any given map, there is much overlap of adjacent body parts - i.e. movement of a given body part can be elicited by stimulation of an area much wider than its representation on a homunculus or simiusculus (Phillips, 1966). The area depicted on the map is only the most sensitive region in a more widely distributed representation.

Early investigators considered area 6 and area 4 to be related hierarchically, Area 6 was a motor association cortex, integrating diverse inputs from all sensory modalities and programming an "executor" area 4 to carry out complex motor responses. There is, however, little evidence for this view. Woolsey (1952) showed that the somatotopic map MI extended forward, beyond area 4, into much of area 6. The map was such that area 6 mediated movement of the axial musculature and proximal limbs, while area 4 contained the representations of the distal limbs. Thus area 4 and the immediately anterior area 6 appeared to be related, not hierarchically but in a complementary manner - i.e. as two parts of a single map. Kuypers (1973) and his colleagues, studying the brainstem and spinal cord projections of areas 4 and 6, have provided evidence consistent with Woolsey's findings. Area 6 projects primarily to the ventromedial portion of the brainstem reticular formation and spinal cord intermediate gray matter. These regions are known to project onto motoneurons controlling the axial muscles and proximal limbs. In contrast, area 4 projects to the dorsolateral portion of the reticular formation and spinal intermediate zone. These areas project onto motoneurons controlling limb and tongue muscles.

However, the relationship of area 6 to area 4 is not completely known. Completion of the motor map MI takes up only some of area 6. The supplementary motor area is also located in the dorsomedial portion of area 6 (far from Broca's area), and its functional relationship to MI is unknown. There is still more of area 6 in the superior frontal gyrus, parts of which yield pupillary dilatation on electrical stimulation (Woolsey, 1952).

Whatever the functional relationship of areas 6 and 4 actually is, it is clear that the inferior portions of these areas on the precentral gyrus are involved in speech movements. Indeed Penfield and Roberts (1959) have shown that stimulation of this region (in either hemisphere) in the unanesthetized human can produce rudimentary vocalization. This response cannot be produced from any other cerebral region.

Although the precentral gyrus thus appears to mediate speech, some of the necessary inputs to this area seem to be indirect. One would ordinarily expect that the cerebral region mediating speech should receive strong input from auditory and visual areas of the brain. Yet, if we can extrapolate from the macaque, this does not appear to be the case, at least directly. According to Jones and Powell (1970) and Pandya and Kuypers (1969), the inferior portions of the precentral gyrus (areas 4 and 6) receive no extensive auditory and visual input. Such input first reaches the frontal lobe via the granular, or pre-frontal cortex as well as the cortex of area 8 that is transitional between agranular cortex (areas 4 and 6) posteriorly and granular cortex anteriorly. The inferior portion of the transitional and granular cortex, lying in and anterior to the inferior limb of the arcuate sulcus of the macaque, is probably homologous to the third frontal gyrus in man. It has numerous connections to auditory and visual association cortex as well as to polymodal association cortex in the angular gyrus and frontal and temporal poles.

The third frontal gyrus, receiving such a multimodal input, could thus be involved in acts requiring cross-modal integration, including many aspects of language-related behavior. There is little doubt that such is the case. Stimulation of the left third frontal gyrus in man may produce speech arrest or paraphasias, just as does stimulation of the left parieto-temporal cortex. But it is also very clear that the third frontal gyrus is not a "motor-memory" store or a motor "association cortex". If it were, stimulation should result in motor phenomena not found with stimulation of other cortical areas. (For example stimulation of visual and auditory association cortex result in visual and auditory experiences). But it does not. Penfield and Roberts (1959) could find no difference between the paraphasias produced by stimulating the third frontal gyrus and those obtained from the parieto-temporal region. Foerster (1935) was unable to produce any movement by stimulating the third frontal gyrus. It may well be true that activity in the third frontal gyrus indirectly affects areas 4 and 6. But the connections are unlikely to be powerful and direct.

Thus, while it appears that the third frontal gyrus has a role in language, it is not the specific role of motor learning envisioned by Broca. The lesions producing Broca's aphasia do so by involving the inferior portions of sensorimotor cortex, particularly areas 6 and 4 of the precentral gyrus. Even these areas may not be related hierarchically: there is no evidence for distinct "memory" and "executor" areas for speech movements.

III. The Cortical Mediation of Speech - an Alternative Model.

We have outlined the evidence that the true "Broca's area" and motor cortex are not separate cerebral areas but are in fact identical. The classical model, elaborated by Dejerine and by Liepmann, is incorrect. One cannot distinguish a cortex containing "motor engrams" for speech (the third frontal gyrus) from an "executor" cortex (the precentral gyrus), with the former driving the latter. Rather, the sensorimotor cortex for speech musculature, occupying the inferior central gyri, particularly the precentral gyrus, is a single complex whose integrity is necessary for the voluntary movements that we call speech.

We shall now attempt to discuss the pathogenesis of many of the clinical signs of Broca's aphasia in light of this view. This discussion will also serve to refine our understanding of the cerebral control of speech.

A. Speech and Other Oro-Laryngeal Movements: Differential Hemispheric Specialization.

Broca first observed that although his patient was unable to speak, he had little or no difficulty in utilizing his mouth, tongue, and pharyngeal muscles for other acts, such as swallowing food. This observation led Broca to postulate that the deficit was a loss of motor-speech memories rather than paralysis. The patient no longer could recall "the procedure for articulating words". Later, this distinction between paralysis and loss of "motor-memories" was reflected in models of cerebral localization. A center for motor-speech memories (third frontal gyrus) was distinct from a general, "executor" motor cortex (precentral gyrus) in the left cerebral hemisphere.

We have already pointed out that one rarely, if ever, observes a patient with Broca's aphasia in whom *all* non-speech movements of mouth, lips, and tongue occur normally (DeRenzi et al., 1966). But *some* acts, such as the ability to move, crush, and ultimately swallow food, are ultimately achieved adequately by nearly all patients with Broca's aphasia. Since we claim that separate areas for speech and non-speech movements do not exist, how do we explain why patients who cannot speak can eat? If the lesion in sensorimotor cortex is sufficient to eliminate speech, why do some other oro-lingual movements still occur?

We suggest that these movements are retained because of preservation of a) remaining areas of the dominant sensorimotor cortex and b) the entire non-dominant sensorimotor cortex. We shall discuss the role of the preserved ipsilateral cortex in the next section. Here we shall concentrate on preservation of the entire right hemisphere. In this regard, the oro-lingual manipulation of food, although admittedly a complex set of movements, can be satisfactorily effected with a single intact hemisphere, which projects bilaterally to bulbar nuclei innervating the oro-pharyngeal muscles. There is no known difference in the effectiveness of left and right sensorimotor cortex in mediating this behavior (although further study of this matter would be welcome). Speech, however, depends upon the integrity of the left sensorimotor cortex far more than the right. The dominance of left over right hemisphere for speech has been known, of course, since it was postulated by Broca (1861) and by Dax (1865).

Thus, we suggest that a lesion of the left sensorimotor cortex will affect speech more than eating. The following case (Levine and Mohr, 1979 - case 3) illustrates this point, and also illustrates the limitations of the right sensorimotor cortex in mediating speech:

> A 20 year-old right-handed woman suddenly became mute with right hemiplegia. After several weeks of speechlessness she began to use the word "here" in a stereotyped way and to curse occasionally. Finally, she began to use a variety of single words and an occasional two-word phrase. She retained a good singing voice and was able to sing entire songs with lyrics. She could also recite the Lord's Prayer, the Apostle's Creed, and the preamble to the U.S. Constitution. There was no dysphagia. Nine years later she suddenly became mute and unable to swallow. One year after this second stroke she swallows with difficulty and remains mute. Her face and tongue are largely immobile, but she often breaks into a broad grin on eye contact with the examiner.
>
> The C.T. scan of this patient is shown in Fig. 11. The first stroke was a massive infarction destroying the entire perisylvian region of the left hemisphere. Speech was drastically impaired, and the residual speech was highly similar to that described by Smith (1966) after dominant hemispherectomy for glioma. Nevertheless, swallowing quickly recovered. The second stroke was much smaller and involved the inferior fronto-parietal region of the right hemisphere - including motor cortex. This stroke deprived her of her residual speech and significantly impaired swallowing and other oro-pharyngeal movements.

B. Hemispheric Specialization is for Tasks - not for "Responses" or for "Comprehension".

It is thus tempting to conclude that some motor acts, such as speech, are left-lateralized while other acts, such as moving food with the tongue, are not. While this hypothesis would explain why speechless patients can eat after lesions of the left sensorimotor cortex, it proves to be misleading and oversimplified. It is incorrect to say that "speech" or any other "motor-behavior" is lateralized to the left hemisphere. To specify what is lateralized one must include not only a description of the motor behavior, but also of the *context* in which the behavior occurs - especially the stimulus, if there is one, that provokes the motor act.

Let us take tongue protrusion as an example. A patient with very severe Broca's aphasia (in the early stages following an acute, large left perisylvian lesion) may be unable to protrude his tongue to any form of request, even though he can use the required musculature in swallowing. Somewhat later, he may protrude his tongue in imitation of the examiner but not to spoken or written request. Still later, he may protrude it upon spoken request but not upon written request.

How shall we describe such behavior? We cannot simply say that the deficit is one of output or production. At one stage, the patient can deliver the output under some circumstances - such as imitation - but not others, such as spoken or written request. We also cannot say that the deficit is one of input or comprehension of speech or print. Some patients, although unable to perform the movement to spoken request, can indicate which of a series of movements performed by the examiner corresponds to the spoken request. Or, although unable to protrude the tongue when asked, the patient may effortfully move the tongue inside his mouth as though "understanding" the spoken request but unable to comply. So either analysis of this behavior as an "output" deficit or as an "input" deficit oversimplifies the picture.

Instead of trying to label the deficit as "output" or "input", it appears better not to subdivide the behavior into such components, but to consider the act as a whole, including the initiating stimulus and the context in which it occurs. In this manner one can argue that protruding the tongue on imitation, protrusion to spoken request, and protrusion to written request are different acts, each with a distinct neural substrate. These acts became progressively more difficult for the patient with Broca's aphasia because they demand progressively more participation of the left hemisphere.

This hypothesis provides an opportunity to understand the selective deficits of tongue protrusion on an anatomic basis. Ordinarily, activity from the left sensorimotor cortex, projected to the bulbar motor nuclei, is sufficient to mediate tongue protrusion under all of the above circumstances. This conclusion is warranted because damage to the right sensorimotor cortex, even complete right hemispherectomy, does not usually interfere significantly with such behavior. If the bulbar output of the left sensorimotor cortex is cut off - for example, by infarction in the internal capsule - the various deficits of tongue protrusion occur only transiently or not at all. The explanation for this lack of deficit, we presume, is that output to the brainstem from the right sensorimotor cortex is also sufficient, *as long as the right sensorimotor cortex is callosally connected to its preserved counterpart in the left hemisphere*. If one adds to the capsular lesion a callosal lesion, disconnecting the left and right sensorimotor cortex the tongue protrusion deficits will appear. The right sensorimotor cortex will be able to move the tongue in circumstances such as moving food in the mouth, where activity in the right hemisphere alone is sufficient to mediate the behavior. But where left hemisphere participation is required, as it apparently is to an increasing degree in imitation, response to spoken input, and response to written input, the act becomes increasingly more difficult. The left sensorimotor cortex cannot interact effectively with the right because the main routes for such interaction - the homotopic callosal connections - have been interrupted.

Such double lesions, involving the left internal capsule and the corpus callosum are very rare, although they have been observed (Bonhoeffer, 1914). We have also recently seen such a case. The very same effect can be produced by a single lesion, situated deep in the left hemisphere where the projection fibers and the callosal fibers intersect, or it can result from lesion of the left sensorimotor cortex itself. These lesions will interrupt both commissural and subcortical projections, duplicating the effects of the separate lesions described above.

The above argument is highly similar to that of Liepmann, later reintroduced by Lange (1936) and by Geschwind (1965), to elucidate the neurological substrate of "oral apraxia" associated with Broca's aphasia. We suggest that the same reasoning can be applied to explain the speech deficit as well.

Like the tongue protrusion deficit, the speech difficulty in Broca's aphasia defies description in such simple terms as "output" or "input" disorder. It is not a simple output disorder, akin to the dysarthrias of myasthenia gravis, progressive bulbar palsy, or even pseudobulbar palsy, which involve (bilaterally) the neuromuscular junctions, lower motor neurons, and cortico-bulbar projections respectively. In Broca's aphasia, syntactically complex utterances, emitted with little articulatory difficulty, occassionally occur in the form of stereotypes, recitation, or song. Yet, the very same words may fail to occur on request, even in repetition. In those patients whose spontaneous speech amounts to only isolated words, these words may be uttered in some circumstances better than others. For example, such patients usually repeat a noun better than they produce it in naming the corresponding object. This selective failure to utter a word or phrase also cannot be understood simply as a "comprehension" deficit. Some patients may be able to match a spoken or written word or phrase to a picture even when unable to repeat the phrase or read it aloud. Even where this is not the case, the struggling inarticulate output which characterizes Broca's aphasia is hard to reconcile with the notion of a "comprehension" deficit alone.

Accordingly, we propose that, like the "oral apraxia", the speech difficulty in Broca's aphasia can be understood as the effect of a) depriving the bulbar nuclei of direct output from the left precentral gyrus and b) isolating the preserved output of the right precentral gyrus from transcallosal input of the left precentral gyrus. In this way the bulbar motor nuclei receive input only from an intact right hemisphere that has not learned certain speech acts as well as the left. This effect is most commonly achieved by a single lesion affecting the left precentral gyrus and immediately subjacent white matter.

It might be expected that injury to the sensorimotor cortex itself would have more widespread effects than a combination of interhemispheric disconnection and interruption of the brainstem projections. Lesion of the cortex itself also interrupts intrahemispheric connections between sensorimotor cortex and other areas of the left hemisphere. The combined callosal and capsular lesions may leave these connections intact. We have seen, however, that such intrahemispheric connections are largely within the sensorimotor cortex itself. Non-capsular projections of sensorimotor cortex (such as to the basal ganglia) would also be affected by the cortical lesion but not by the combined capsular-callosal lesions. The significance of these different lesions with regard to the patients' behavior is still unknown.

C. <u>The Relationship of Speech and Writing</u>.

The large majority of patients with Broca's aphasia are agraphic. The right arm is hemiplegic and cannot, of course, write. The left hand usually writes with a coarse, slow, wavering stroke and produces either distorted, unrecognizable letters, or letters which are recognizable but wrong, or an occasional correct word or two. Copying is often better than writing spontaneously or to dictation - but copying too, may be defective.

To Wernicke and to Dejerine, lesion of the Broca's center of motor-speech memories resulted in <u>both</u> the speechlessness and the agraphia. Writing required "inner speech", and the latter required integrity of Broca's area, Wernicke's area and their interconnections. Neural activity corresponding to "inner speech" activated the arm area of the left sensorimotor cortex to produce writing.

However, although most non-fluent aphasics are agraphic, not <u>all</u> of them are. Conversely, there are rare patients with marked agraphia, who have little or no speech deficit. To the classical authors such cases were easily explained. Patients with either pure word mutism or pure agraphia did not have a lesion of Broca's area, but instead had a lesion of "executor" motor cortex. Those who could not speak had a lesion of the portion of executor cortex projecting via brainstem nuclei to the oro-pharyngeal muscles, while those who could not write had unilateral lesions of executor cortex innervating the upper extremity.

But we have done away with separate "motor memory" and "motor executor" areas except insofar as someday sensorimotor cortex itself may become functionally divisible. How then, do we explain the rare cases of "pure word mutism" or "pure agraphia"?

The answer appears to lie in lesion size. The sensorimotor cortex runs in a broad strip along the lateral convexity, increasing in width as one ascends dorsomedially from the sylvian fissure toward the interhemispheric fissure. In the homunculus of MI the representation of the upper extremity, although it actually overlaps that of the mouth, has its most sensitive region more dorsally. This anatomical subdivision allows for different effects of lesions depending on their precise location within the sensorimtor cortex.

We propose that "pure word mutism" may result from a discrete lesion of the precentral gyrus, affecting only the inferior portion and sparing the more dorsal representation of arm and hand to a large degree. We also suggest that a more dorsal lesion, affecting the arm and hand area but sparing the inferior face region, may result in right mono- or hemiplegia and agraphia of the left hand without aphasia. A combined lesion of both areas will produce Broca's aphasia.

Existing evidence seems to support this point of view. Three cases of (approximately) "pure word mutism", studied at post-mortem examination, proved to have lesions restricted to the inferior precentral gyrus (Dejerine, 1906; Lecours, 1976). We have seen two such patients, both of whom are still living. Although each clearly had a stroke preceeding their aphasia, neither has evidence of an extensive lesion on C.T. scan. There is only widening of the left sylvian fissure and of the frontal horn of the left lateral ventricle to suggest a small lesion in the appropriate place.

Numerous, but scattered reports in the literature also suggest that more dorsal circumscribed lesions of sensorimotor cortex may result in agraphia without aphasia. This was probably the stimulus for Exner's (1881) postulating a writing center in the second frontal gyrus. Such cases probably always involve the motor cortex representing the upper extremity in the precentral gyrus.

In contrast to these more circumscribed lesions, patients with Broca's aphasia have larger and/or deeper lesions, compromising <u>both</u> face and upper extremity regions (Mohr et al., 1978). We have reported a case of severe, permanent Broca's aphasia, resulting from a superficial lesion of the entire precentral gyrus that did not significantly invade either the frontal gyri anteriorly or the postcentral gyrus posteriorly (Trojanowski et al., 1980). This lesion is shown in Fig. 8. More frequently, the lesion is smaller but deep, interrupting both callosal and capsular fibers from a wide extent of the precentral gyrus.

We have suggested parallel models for the speech deficit and the writing deficit in Broca's aphasia. Both involve lesions of the sensorimotor cortex - especially the precentral gyrus - of the dominant hemisphere. The mouth and hand areas overlap but are not congruent; so dissociations - occasionally dramatic ones - may occur.

To this point we have discussed the relationship of deficits of speech and writing with regard to the location of possible lesions within motor cortex of the dominant hemisphere. But is the dominant hemisphere for speech acts and for writing acts necessarily the same? In the neurologic literature, surprisingly little attention has been paid to this aspect of the relationship between speech and writing deficits.

The oro-pharyngeal movements called speech lateralize to the left hemisphere in the majority of people, and most people are also left hemisphere dominant for the performance of many motor acts with the limbs. But speech dominance and limb motor dominance in <u>some</u> cases can occur in opposite hemispheres. This is especially the case in left-handed people, presumably right hemisphere dominant for limb-motor skills, of whom 60 - 80% are left hemisphere dominant for speech (Roberts, 1969). In rare instances, right-handed people - presumably left dominant for limb-motor skills - may develop speech in the right hemisphere (Trojanowski et al., 1980).

The lateralization of writing is a problem in such cases. Writing is a limb-motor skill, and so might be expected to lateralize with other such skills. However, unlike many other limb movements, writing is learned in intimate association with the hearing, reading, and/or utterance of speech. Acquisition of this skill by the speech dominant hemisphere will be more efficient than by the non-speech hemisphere, because the latter requires cross-hemispheric integration.* In most people who are left hemisphere dominant for both speech and limb praxis, this dual nature of writing creates no conflicts; and this skill happily lateralizes to the left hemisphere. However, what happens to writing in those subjects where speech and limb motor skills lateralize to opposite hemispheres?

Even less is known about the rare right-handers with speech in the right hemisphere. Such patients may become agraphic with lesions of the speaking right hemisphere, as in our case (Trojanowski et al., 1980). We know of no case of a right-handed patient, right dominant for speech, in whom a left hemisphere lesion produced pure agraphia (without aphasia) of the left hand.

D. Deficits of "Comprehension" in Broca's Aphasia.

We have already mentioned that patients with Broca's aphasia usually show some difficulty comprehending speech, marked alexia, and difficulty comprehending numbers and arithmetic operations. Of course, every test of comprehension requires some response from the patient. When the response is correct, we confidently assert that the patient comprehended. However, when the patient fails we invariably start to wonder whether the failure is one of "comprehension" or of "response generation"(praxis) ; i.e. a "comprehension defect" is not something that most people will allow as directly observable. One approach to this problem is to keep the input constant but to simplify the motor reponse that is required. If this simplification allows the patient to pass the test, one might argue that "comprehension" is preserved and the deficit is one of "response generation".

In this sense, there is no avoiding the fact that most patients with well developed Broca's aphasia have deficits of comprehension. Even if one simplifies the motor response to pointing to the correct element in a set of a few choices, there are difficulties. The patient with Broca's aphasia cannot point with normal facility to the printed word that completes an incomplete sentence. He may even show deficits in matching single dictated words or printed words to pictures - whether the words be names of arithmetic operations, numbers, objects or colors.

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* Myers (1965) has shown that, in the monkey, discriminations that require activity within a single hemisphere only (e.g. input from right visual field, output from right hand) are more efficiently learned than discriminations which require cross-hemispheric integration via the corpus callosum (e.g. input from right visual field, output from left hand). We have suggested that this efficiency difference may be the basis for the normal right ear superiority in dichotic listening and the right visual field superiority in identifying letters. (Levine and Calvanio, 1980).

It is often suggested that such comprehension deficits result from extension of the lesion into the posterior parietal or posterior temporal lobes. This suggestion, unsupported by evidence, is largely based on difficulty understanding how lesions in "motor" areas of the brain can impair comprehension, especially since "centers" for speech comprehension and for print comprehension supposedly exist in the left posterior temporal and posterior parietal lobes. These posterior centers should be able to activate the right hemisphere transcallosally. Intrahemispheric paths in the right hemisphere should then suffice to activate the right motor cortex, allowing successful performance of the required task. But such is not the case. Severe alexia and considerable speech comprehension difficulty may result from lesions of the left precentral gyrus that leave the posterior temporo-parietal language area largely intact. Such was the case in the patients whose lesions we have previously demonstrated (Fig. 8, 9). Activation of the right motor cortex from the left posterior region by a combination of posterior transcallosal and right hemispheric activation cannot alone mediate normal comprehension (Levine et al., 1980).

It is thus clear that the cortex of the left precentral gyrus participates actively not only in behavior requiring overt speech or complex limb movement, but also in acts of language comprehension as well. Perhaps this results from the fact that the child employs speech and thus activates the left precentral gyrus as he masters comprehension skills during childhood. The cortical substrate of an acquired skill may be no more than the network of cortical neurons activated during its acquisition. If so it is not surprising that deficits of comprehension as well as speech occur with lesions of the left precentral gyrus.

Regardless of the validity of this speculation, the fact remains that damage confined to the precentral gyrus may impair not only speech but comprehension of language as well. It is incorrect to omit the precentral gyrus in maps of the language area (Dejerine, 1914). More importantly, it becomes clear that identifying abstract psychological functions ("comprehension" or "semantic access"; "production" or "syntactic and/or phonologic encoding") with highly circumscribed areas of cortex is probably quite unwise. Although we might build a brain by interconnecting a set of independent modules, each with a common sense psychological subfunction in the genesis of language acts, nature has evidently chosen another way.

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FIGURE LEGENDS

Fig. 1 Lateral view of a cerebral hemisphere. Markings occupy various portions of the third frontal gyrus, defined by gross anatomical landmarks. Circles occupy the pars opercularis, situated between precentral sulcus posteriorly and ascending limb of Sylvian fissure anteriorly. Crosses occupy pars triangularis, situated between the ascending and horizontal limbs of the Sylvian fissure. Horizontal lines occupy the pars orbitalis. P.C.G. denotes the precentral gyrus situated immediately posterior to the third frontal gyrus.

Fig. 2 Four cases from the literature of lesions of the left third frontal gyrus in right-handed subjects with no history of aphasia:
Top-left: Moutier's (1908) case "Proudhomme"; softening of the pars opercularis.
Top-right: Archambault's (1913) case; softening of the pars opercularis.
Bottom-left: Henschen's (1920) case; softening of the left pars opercularis.
Bottom-right: Foulis' (1879) case; nearly complete destruction of the third frontal gyrus; the precentral gyrus intact.

Fig. 3 Location of lesion in PM. Three levels of C.T. scan are shown (top-right, bottom-left, bottom-right in ascending order), each showing hemorrhage (densely white area) in left third frontal gyrus. At top left the levels of the section are shown and the projection of the hemorrhage on the surface of the hemisphere is outlined.

Fig. 4 Profile of scores for case 1 of Levine and Mohr (1979) on the Boston Diagnostic Aphasia Examination (Goodglass and Kaplan, 1972). Scores are expressed in number of standard deviations above or below the average score of a standardization group of aphasics. Note the presence of mild to moderate aphasia, very unlike the profile expected for a patient with significant Broca's aphasia.

Fig. 5 Location of lesions in case 1 of Levine and Mohr by computerized tomographic (CT) scan. The C.T. scan slices from inferior upward are shown in the middle row left, middle row right, bottom row left, and bottom row right. The top row shows diagrams of the lateral surfaces of the left and right hemispheres. The straight lines depict the centers of the C.T. sections shown below. The heavy black outline represents the surface projection of the lesion in that hemisphere.

Fig. 6 Location of the lesions in Gianulli's case. The left hemisphere (top) and the right hemisphere (bottom) both show softenings of the pars opercularis of the third frontal gyrus.

Fig. 7 Marie's schematic horizontal section of a cerebral hemisphere, illustrating the "lenticular zone" with its anterior (A) and posterior (B) boundaries.

Fig. 8 Photograph of the lateral surface of the right hemisphere in the case of Trojanowski et al illustrating the large lesion confined to the precentral gyrus.

Fig. 9 C.T. scan of an unpublished case of Broca's aphasia with a focal lesion of the inferior precentral and postcentral gyrus.

Fig. 10 Wooley's (1958) diagram illustrating the location of the four major topographically organized areas from which electrical stimulation of the macaque cortex yields movements. MI - (motor) precentral gyrus MII - (supplementary motor) medial superior frontal gyrus and cingulate sulcus SI - (somatosensory) post central gyrus SII - (second sensory) parietal operculum.

Fig. 11 Location of lesions in case 3 of Levine and Mohr (1979) by computerized tomographic scan.

Fig. 1

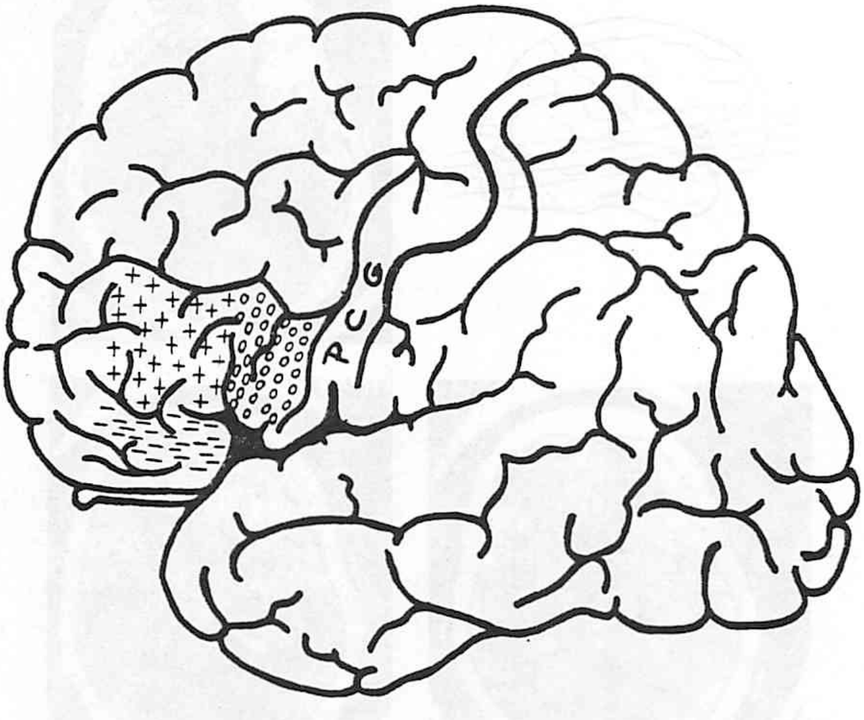


Fig. 2

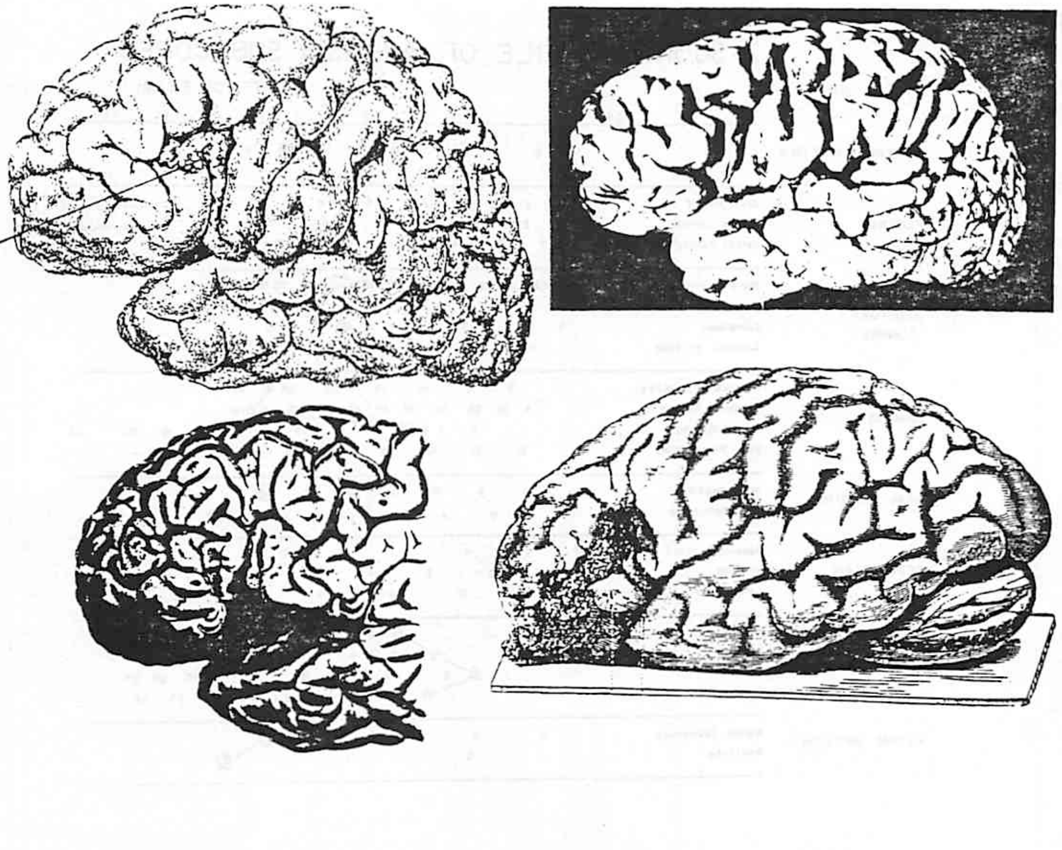


Fig. 3

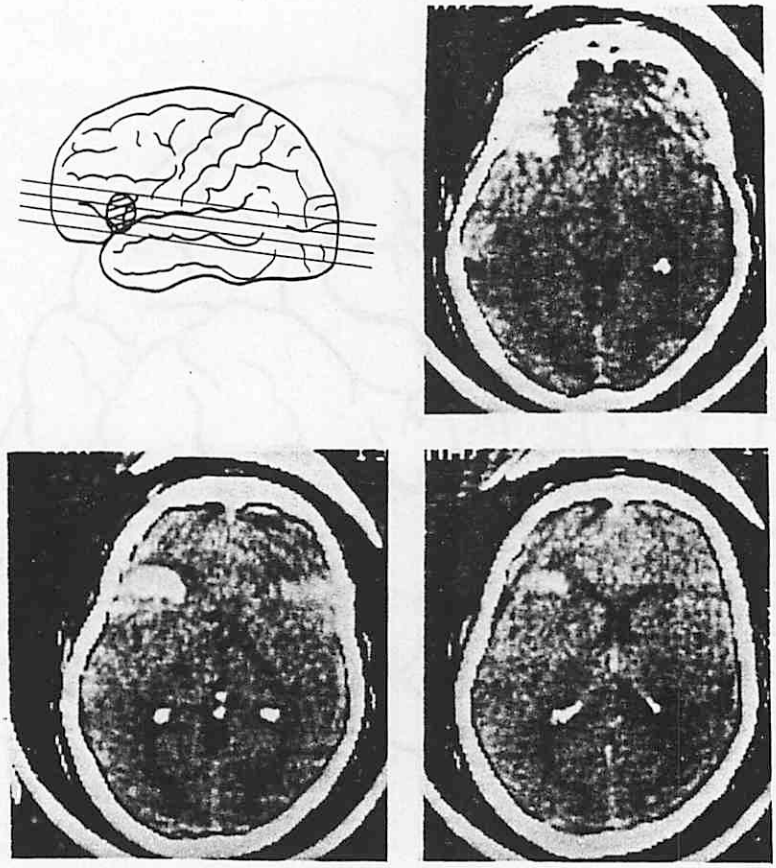


Fig. 4

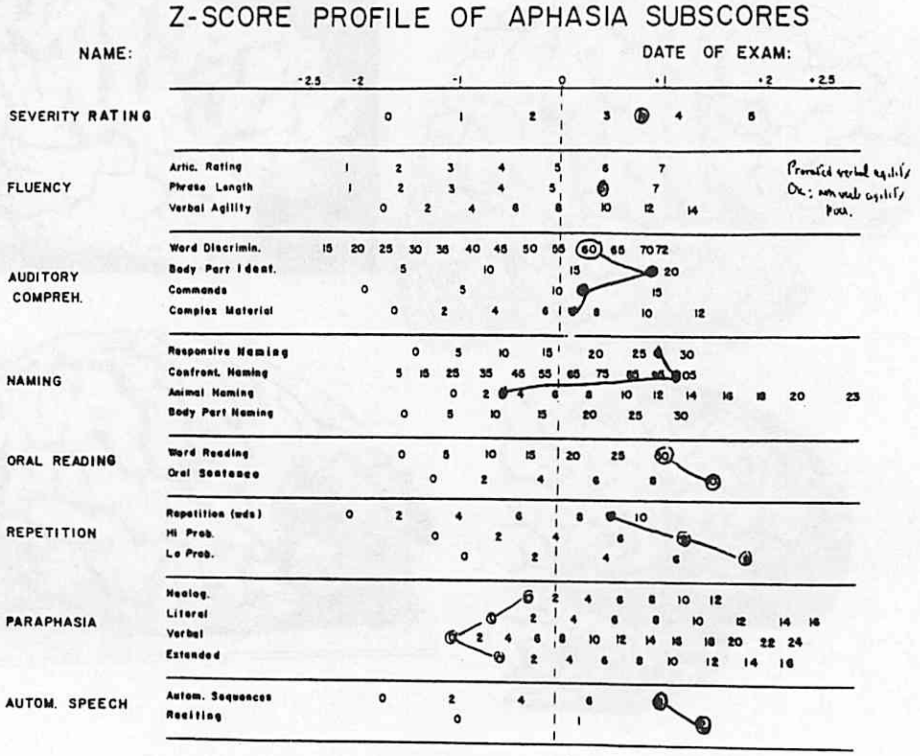

Z-SCORE PROFILE OF APHASIA SUBSCORES
NAME:
DATE OF EXAM:
-2.5 -2 -1 0 +1 +2 +2.5
SEVERITY RATING 0 1 2 3 4 5
FLUENCY
Artic. Rating 1 2 3 4 5 6 7
Phrase Length 1 2 3 4 5 6 7
Verbal Agility 0 2 4 6 8 10 12 14
Provoked verbal agility/ Oc: non verbal agility/ poor.
AUDITORY COMPREH.
Word Discrimin. 15 20 25 30 35 40 45 50 55 60 65 70 72
Body Part Ident. 5 10 15 20
Commands 0 5 10 15
Complex Material 0 2 4 6 8 10 12
NAMING
Responsive Naming 0 5 10 15 20 25 30
Confront. Naming 5 15 25 35 45 55 65 75 85 95 105
Animal Naming 0 2 4 6 8 10 12 14 16 18 20 23
Body Part Naming 0 5 10 15 20 25 30
ORAL READING
Word Reading 0 5 10 15 20 25 30
Oral Sentence 0 2 4 6 8 10
REPETITION
Repetition (wds) 0 2 4 6 8 10
Hi Prob. 0 2 4 6 8
Lo Prob. 0 2 4 6 8
PARAPHASIA
Neolog. 0 2 4 6 8 10 12
Literal 0 2 4 6 8 10 12 14 16
Verbal 0 2 4 6 8 10 12 14 16 18 20 22 24
Extended 0 2 4 6 8 10 12 14 16
AUTOM. SPEECH
Autom. Sequences 0 2 4 6 8
Reciting 0 1 2

Fig. 5

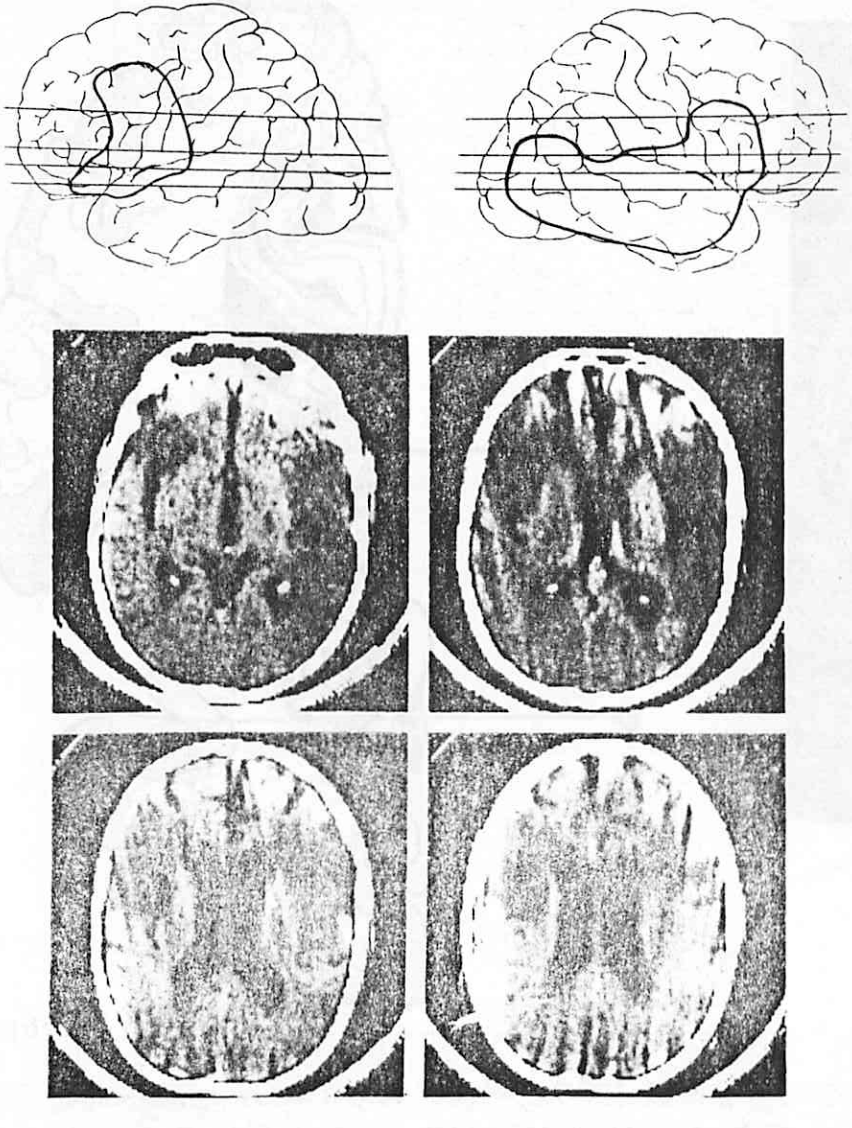


Fig. 6

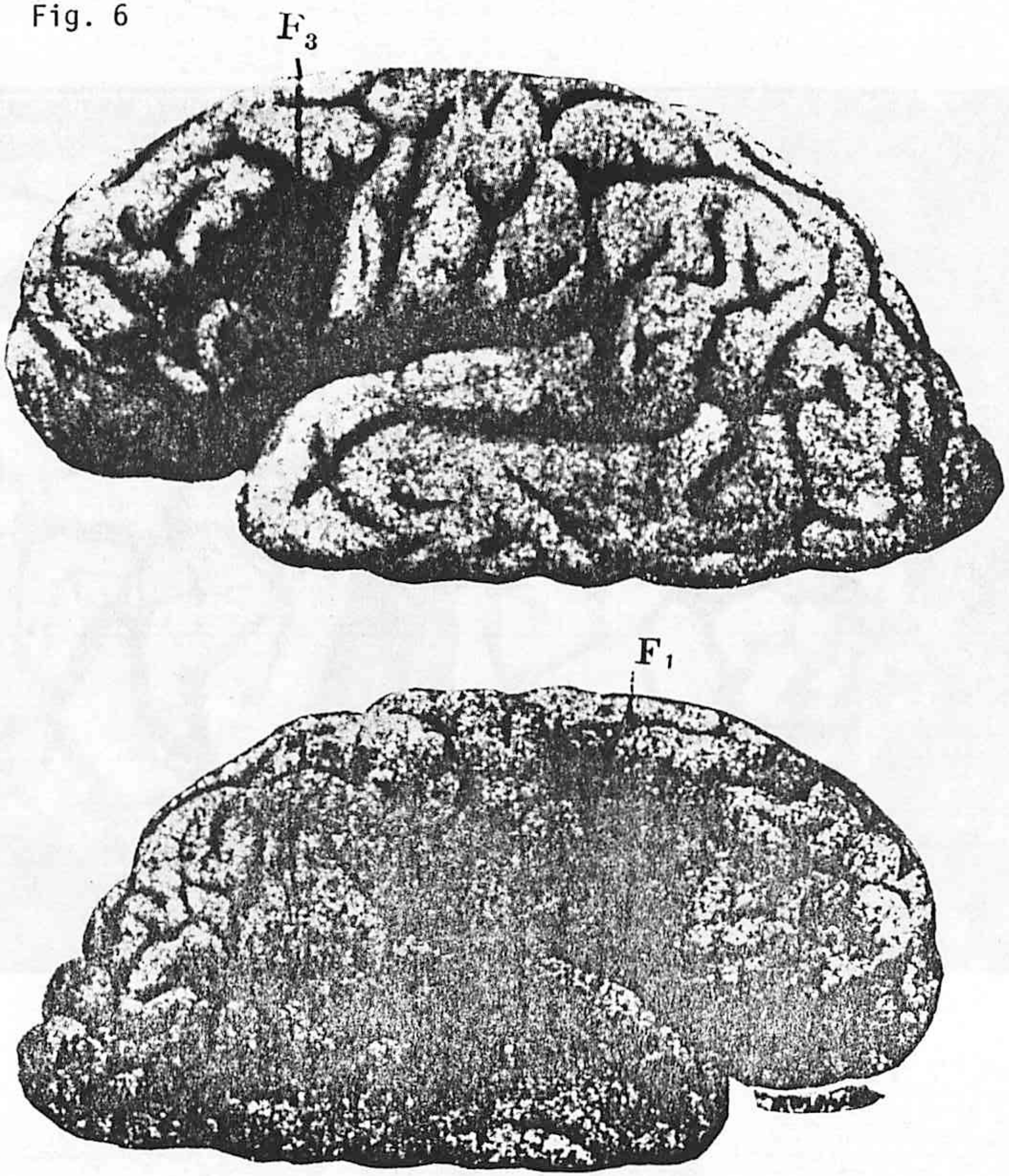


Fig. 7

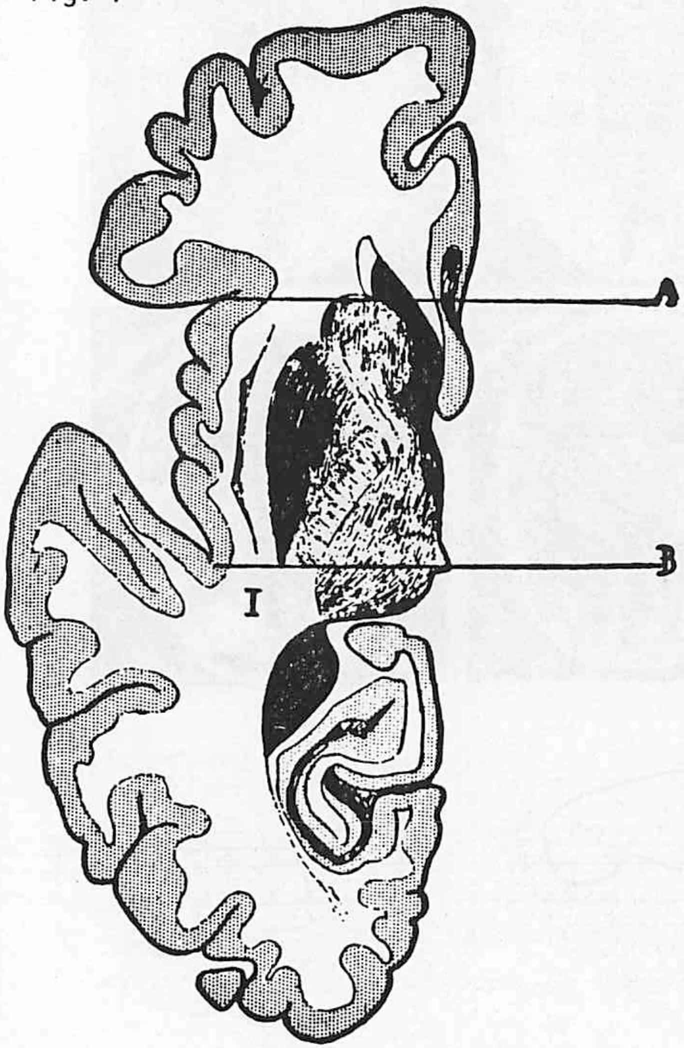
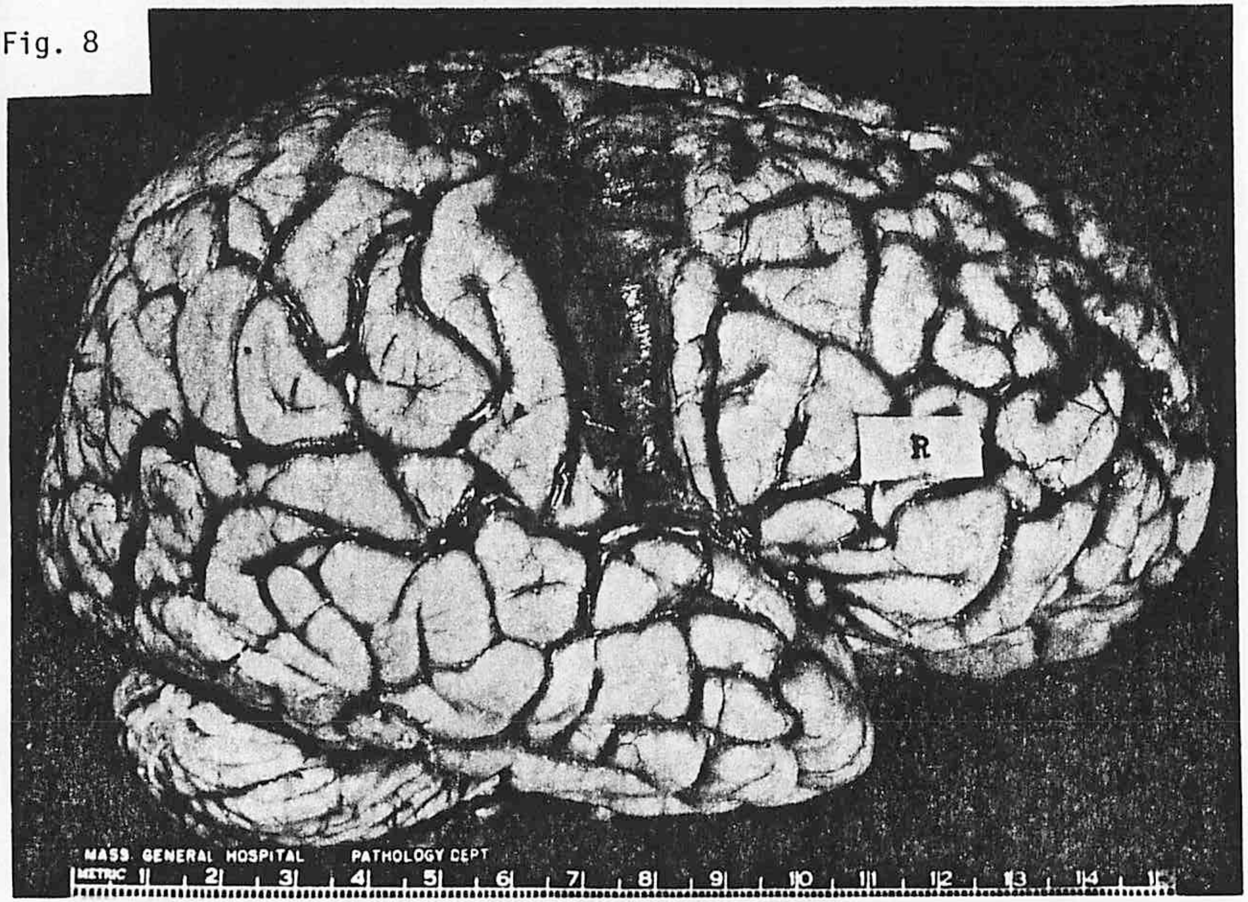

A
B
I

Fig. 8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MASS. GENERAL HOSPITAL PATHOLOGY DEPT
METRIC 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15

Fig. 9

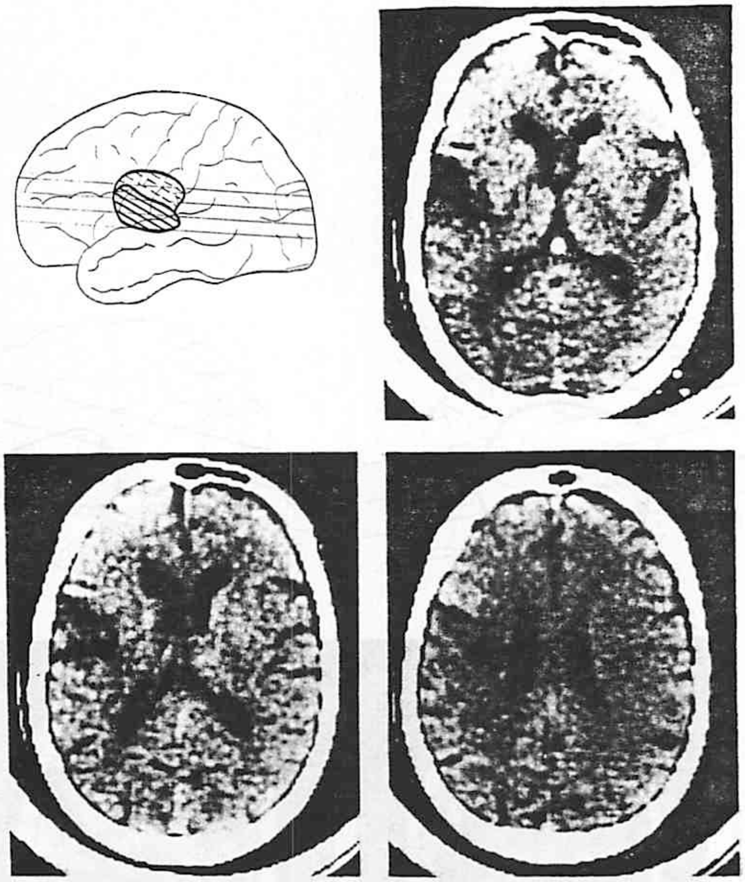


Fig. 10

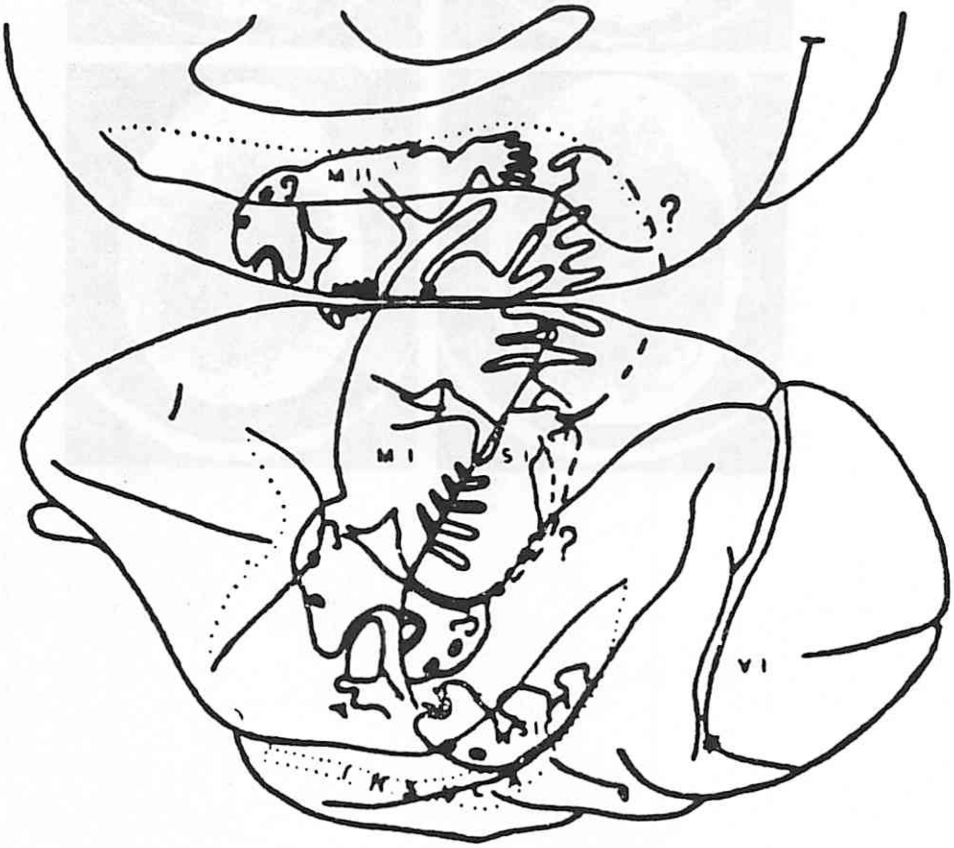
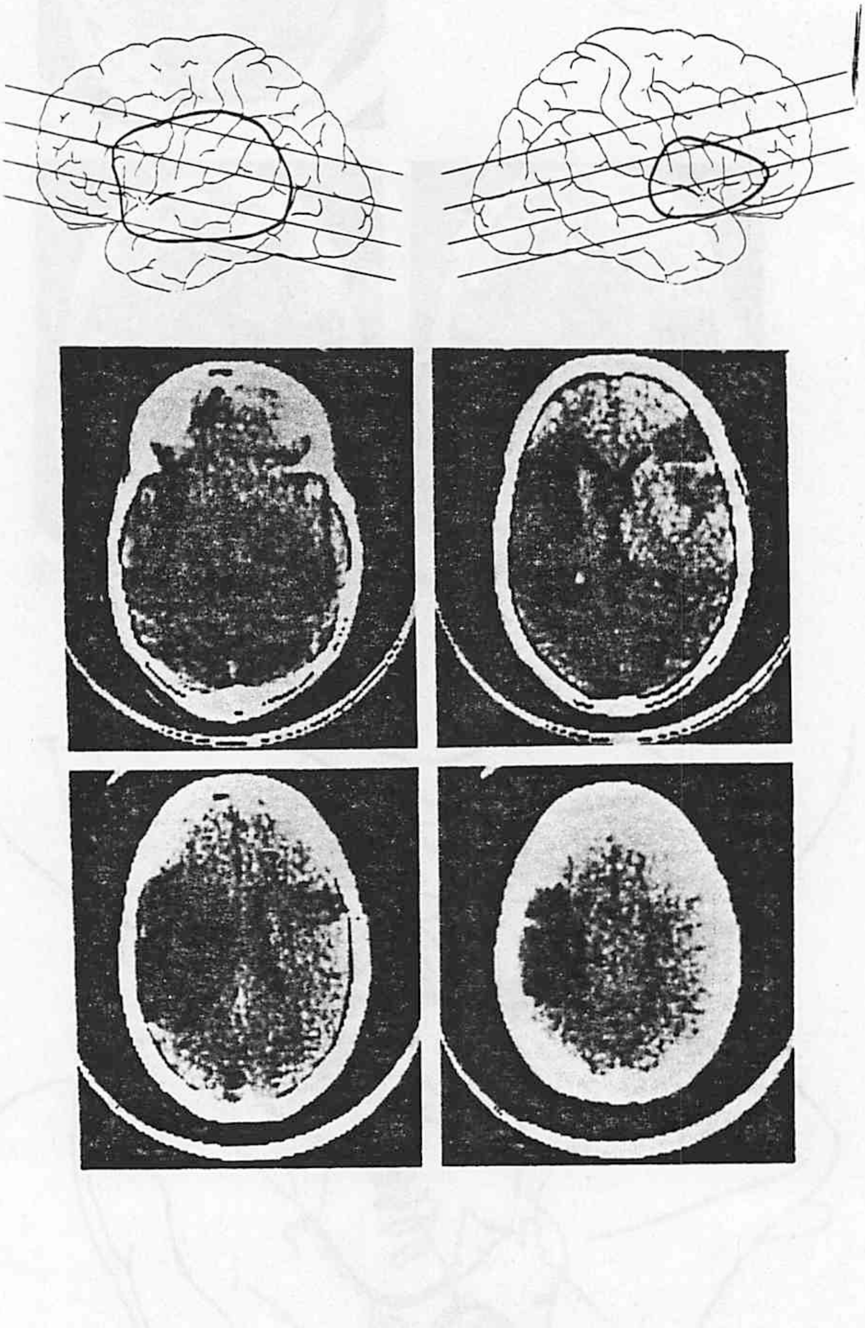


Fig. 11



DISCUSSION OF LEVINE PAPER

<u>Halwes</u>: The split brain studies showed that there was a lot of language processing in the right hemisphere. Zaidel, at CalTech, has developed a technique for presenting a sustained visual stimulus to a single hemisphere -- the "stoped image mask" controls for eye movements. He found that the right hemisphere had almost an adult vocabulary but perhaps 6-year-old syntax, and could not handle auditorily presented nonsense (suggesting that it lacks a purely phonetic representation).
[Reference: Zaidel, E. Auditory vocabulary of the right hemisphere following brain bisection or hemidecortication. <u>Cortex</u> 12: 191-211, 1976.]

<u>Kertesz</u>: Wernicke ascribed recovery of aphasics to learning to use the right hemisphere to talk. Language is, in fact, elaborated through the collaboration of many regions of the brain.

Some would consider aphasic those Levine would say are not aphasic -- he seems to limit himself to "severe persistent Broca's aphasia". Many Broca's aphasics can return home because their ability to communicate has returned to a reasonable functional level. We should differentiate patients on the basis of their test scores. I think Levine's CT-scan case did involve Broca's area -- it is hard to localize a slice in a CT-scan.

<u>Wood</u>: Whether Broca's aphasia is associated primarily with lesions of the third frontal gyrus or with lesions of the precentral gyrus is an important issue. However, in discussions such as this, one often comes away with the impression that the "where" question (i.e., specification of lesions necessary and sufficient to rpoduce symptom pattern x) is <u>the</u> question. But from the perspective of a comprehensive computational theory, the "where" question is far less important than "what" and "how".

<u>Whitaker</u>: Good localization makes for better linguistic theory, correlating specific neural entities with types of neurons. Unfortunately, descriptions of symptoms vary from author to author (did Marie just describe an anarthria?), and CT-scans cannot make the fine localizations that Galaburda can. And 19th century drawings may be unreliable guides to localization. [Editor's Query: How good are the data linking cell-types with specific gyri?] We need sophisticated neurolinguistic analysis to separate direct control of the vocal tract, sequential control of articulators, superimposition of an intonation contour, and word selection.

<u>Levine</u>: The studies demonstrating high vocabulary level in the right hemisphere of split brain patients have all involved non-speech responses. In other words, subjects were required to match a printed or heard word to a picture. There has been no evidence of a significant motor-speech capacity from the right hemisphere and therefore no discrepancy between our findings and tose at Caltech.

With regard to Andy Kertesz' comments, what I call Broca's aphasia is what Broca (and Wernicke, and Isserlin) called Broca's aphasia. However, if my patients as a group are more severely impaired than those of Kertesz, my arguments for the wide ranging involvement of the precentralgyrus in many language activities are only made stronger.

Finally, localization does make a difference, because it affects theorizing. If, as I claim, a lesion in the "motor cortex" can also impair speech comprehension and print comprehension, the defect cannot just be in the motor control oflips and tongue. Ithink that the significance of an area of cortex in any skill may be clarified by analyzing how that skill is learned. We learn to read by saying words when confronted with visual patterns. Thus, motor cortex is activated repeatedly during the acquisition of reading, and for this reason may form part of the cortical substrate of this skill.

Caplan: There seems to be a real convergence between the work on linguistic representations and that of the psycholinguists; and between parsing studies by both psycholinguists and AI people. However, one of the biggest dangers I see is that too many people still seem to believe that a structural analysis of language implies a structural analysis of brain, or vice versa. While avoiding this conflation, two gaps still remain -- in the clinical description of aphasic syndromes, and in neurophysiological studies relevant to language processing. We may well need mutual adjustment of terminology to make neurology and neurophysiology compatible with AI and psycholinguistics. The insistence that neurological data be modellable, at least in principle, can make a real methodological difference in the clinic. We cannot then affort to talk just of "amount of comprehension" in global terms, but must instead lay out the symptoms in terms that can make contact with processes (cf. Gordon and Marin's "Neuropsychologic Aspects of Aphasia"). With this we can begin to build cooperative computation models (cf. Arbib and Caplan, 1979) which should bridge the gap from neurology and gross anatomy to the modelling of detailed neural circuitry.

IMPLICATIONS OF SIMULATED LESION EXPERIMENTS FOR THE INTERPRETATION OF LESIONS IN REAL NERVOUS SYSTEMS

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1. Introduction

Behavioral deficits following brain damage have long been used to make inferences about the functional organization of the nervous system in general, and about the organization of human language in particular. The work of Flourens (1824, 1825) is generally cited as the first attempt to explore the functional organization of the brain by studying the behavioral consequences of systematic brain lesions in animals (e.g., Boring, 1950; pp. 61-67). Subsequent arguments for and against localization of function in the brain were based almost exclusively upon the results of lesion experiments (e.g., Ferrier, 1876; Munk, 1881; Goltz, 1881; Franz, 1902; Lashley, 1929, 1950). Nowhere has the influence of lesion data been more strongly felt than in the continuing debate between localizationist and anti-localizationist theories of brain organization for language (e.g., Hughlings Jackson, 1874; Head, 1963; Luria, 1973; Geschwind, 1974).

In view of the inferential importance of lesion experiments, it is surprising that the logical basis for their interpretation has been discussed (in public, at least) so infrequently. With the notable exception of Gregory (1961), the problem of interpreting lesion data has most often been discussed in the context of related questions, not as an important issue in its own right. For example, most of the relevant discussions have occurred in the context of localization of function (e.g., Klein, 1978; Uttal, 1978) or recovery of function (e.g., Rosner, 1970, 1974; LeVere, 1975; Eidelberg and Stein, 1974). The question "What inferences about the functional organization of the nervous system can be justifiably made from lesion experiments?" has received surprisingly little direct attention.

In this paper I describe a novel way of investigating what can be learned from lesion experiments, based on simulated lesions in a simple neural model of associative memory. There are both advantages and disadvantages in using a neural model for this purpose. On the positive side, three different advantages may be noted: (1) Unlike lesion experiments in real nervous systems, simulated lesions in a neural model permit lesion size and location to be precisely controlled and systematically manipulated. (2) Arbitrarily large numbers of lesions, subjects, and behavioral tasks can be studied in simulations, all of which are limited resources in real lesion experiments. (3) Perhaps most important, the mechanisms of information representation and processing in a neural model can be completely and quantitatively specified. Therefore, the problem of inferring functional organization from lesion data can be addressed directly using a neural model in a manner that is impossible for real nervous systems. On the negative side, using simulated lesions for this purpose is subject to the following limiations: (1) The specific model used to study simulated lesion effects may be inadequate or incorrect in a number of ways; to the extent that its assumptions are invalid, certain conclusions drawn from the simulated lesion experiments may also be invalid. (2) The range of "behavioral tasks" that can be studied in simulated lesion experiments is constrained by the particular model employed. For example, the capabilities of the model used in this paper are limited to association of a given pattern or input activity with a given pattern of output activity. Therefore, tasks other than "association", "recognition", and "discrimination" cannot be investigated using this model. (3) As discussed in greater detail below, the manner in which the simulated lesions are implemented has important consequences upon the conclusions drawn.

Thus, the principal aim of this exercise may be stated as follows:

Given that we know the mechanisms of information representation and processing that are built into the model by definition, to what extent can we unambiguously infer them from behavioral deficits following damage to the model? The model I shall use for this purpose is formulated at a lower level than many that will be discussed at this conference, in the sense that it is formulated in terms of the connections and activities of individual neurons instead of abstract processes having no neural realization. Nevertheless, the model has been shown to be capable of approximating a number of interesting "cognitive" phenomena. Regardless of the validity or generality of the specific model employed, the general approach can be applied to any model in which aspects of neural structure are directly represented.

A secondary objective of this paper is to consider the interpretation of lesion effects in the context of a model in which information processing and storage are distributed across large populations of neurons. In recent years, it has become increasingly clear on both empirical and theoretical grounds that progress in understanding nervous system function requires consideration of the cooperative activity of relatively large groups of neurons (e.g., Arbib, 1972, 1980; Edelman, 1978; Mountcastle, 1978; Szentagothai and Arbib, 1974; Freeman, 1975; Szentagothai, 1978; Erikson, 1974). Therefore, neural models that involve one or another form of distributed processing have received increasing attention (e.g., Grossberg, 1976a,b; Anderson et al., 1977; Pribram, Nuwer, and Baron, 1974; Willshaw, Buneman, and Longuet-Higgans, 1969; Little and Shaw, 1975; Kohonen, 1977; Marr and Poggio, 1977)[1].

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[1] It is worth noting that explicitly psychological (as opposed to physiological) models have begun to incorporate distributed processing assumptions (e.g., McClelland, 1979).

The remainder of the paper is organized in the following way: Section 2 reviews the assumptions and mathematical representation of the model, presents a simple numerical example to illustrate the model's properties, and discusses measures for assessing the model's performance. Section 3 describes the implementation of the simulated lesions, the effects of systematic variations in lesion size and location on the model's performance, and the effects of specific lesions on specific associations. Finally, Section 4 considers the implications of these results for the interpretation of real lesion experiments.

2. Anderson's Model of Associative Memory

The mathematical model used for the simulated lesion experiments is the one recently applied to the problems of feature detection, categorical perception, and probability learning by Jim Anderson and his colleagues of Brown University (Anderson, 1972, 1977; Anderson, Silverstein, Ritz, and Jones, 1977). Closely related models have been applied to the development of "feature detector" cells in visual cortex (Nass and Cooper, 1975; Cooper, Liberman, and Oja, 1979), item recognition (Anderson, 1973, 1977), vowel perception (Anderson, Silverstein and Ritz, in press), and to general associative memories (Kohonen, 1977; Kohonen et al., 1977; also see Little and Shaw, 1975; Shaw, 1978; Roney and Shaw, 1978). In one form or another, all of these models incorporate what have been called "correlation matrix memories" (Kohonen, 1972) and all share two fundamental assumptions: (a) that memory is distributed over a large population of neurons; and (b) that memory storage involves synaptic modification that depends upon activity in pre- and

postsynaptic neurons (Hebb, 1949).

2.1 Assumptions and Mathematical Representation[2]

Anderson et al. (1977) based their model on the following general assumptions: (a) "Nervous system activity can be most usefully represented as the set of simultaneous individual neuron activities in a group of neurons"; and (b) "Different memory traces (sometimes called 'engrams'), corresponding to these large patterns of individual neuron activity, interact strongly at the synaptic level so that different traces are not separate in storage" (p. 415).

The model assumes the existence of two sets of N neurons, α and β, in which every neuron in α is synaptically connected to every neuron in β (Figure 1). Set α is assumed to receive input from some unspecified source (either other neurons if α consists of interneurons, sensory receptors if α consists of first-order sensory afferents, or the environment if α consists of sensory receptors). Set β is assumed to send output to some other unspecified location (either other neurons if β consists of interneurons, muscle cells if β consists of motorneurons, or the environment if β consists of muscle cells). For convenience, I shall refer to sets α and β as "input neurons" and "output neurons", respectively.

The activity of each neuron in the model is represented as a continuous variable, assumed to correspond to the neuron's firing frequency relative to

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[2] The description of the model in Section 2.1 and much of the data presented in Section 3 were reported by Wood (1978).

some baseline level; thus, activity consists of both positive and negative values relative to baseline. Each input neuron is assumed to be connected to every output neuron with a given synaptic strength, and all neurons are assumed to be simple linear integrators of their inputs. Thus,

$$g(i) = \sum_{j=1}^{N} x_{ij} f(j) \qquad [1]$$

where $g(i)$ is the activity of neuron i in β, $f(j)$ is the activity of neuron j in α, and x_{ij} is the synaptic strength connecting neuron j to neuron i. Given these assumptions, the patterns of activity in α and β can be represented as the N-element vectors $\underline{f}$ and $\underline{g}$, which reflect the activity at a given time over all N neurons in each set. For convenience, the input vectors are assumed to be normalized:

$$\left[\sum_{i=1}^{N} f(i)^2 \right]^{1/2} = 1 \qquad [2]$$

The fundamental association assumption of the model is that a given pattern of activity in α, input vector $\underline{f}_1$, is associated with a given pattern of activity in β, output vector $\underline{g}_1$, in such a way that the synaptic strengths x_{ij} are equal to the product of the input and output vectors:

$$\underline{A}_1 = \underline{g}_1 \underline{f}_1' \qquad [3]$$

where $\underline{A}_1$ is an N X N matrix of synaptic strengths, $\underline{g}_1$ and $\underline{f}_1$ are N-element column vectors, and $\underline{f}_1'$ is the transpose of $\underline{f}_1$. Anderson et al. make no explicit assumptions about the learning process by which the synaptic strengths become altered in this way; they simply assume that the result of the learning process can be represented as the product of the input and output vectors. For more detailed discussion of this and related synaptic modifiability assumptions, see Anderson (1977), Kohonen et al. (1977), Kohonen (1977), and Stent (1973).

Once input vector $\underline{f}_1$ has been associated with output vector $\underline{g}_1$ according to Equation 3, the occurrence of pattern $\underline{f}_1$ in α produces pattern $\underline{g}_1$ in β. Any number of input and output patterns may be associated by first computing the association matrix according to Equation 3 for each pair and then summing the individual association matrices:

$$\underline{A} = \sum_{i=1}^{M} A_i \qquad [4]$$

If the input vectors are orthogonal, the occurrence of any input vector previously associated with an output vector produces the appropriate ouput vector in response, that is,

$$\underline{A}\underline{f}_i = \underline{g}_i \qquad [5]$$

for any number of input and output vectors (on the order of N).

Anderson et al. (1977) actually presented two distinct models. The first is the two-population model just described (Figure 1), in which every neuron in one population is connected to every neuron in the other. Their second model incorporates feedback from each neuron onto itself and adds saturation of the activity of individual elements. The latter model was applied to the specific empirical problems addressed in their paper. For simplicity, the model without feedback was used for the simulated lesion experiments discussed here. However, simulated lesions like those employed here could be easily generalized to the feedback model or to any of the other correlation matrix models mentioned above.

2.2 A Numerical Example

Anderson et al. (1977) presented a numerical example of the model's performance based on eight input and eight output neurons and the four sets of arbitrary input and output vectors shown in Table 1. This example will be used here to illustrate some basic properties of the model. The model is first "taught" to associate each input vector with the corresponding output vector in Table 1 (i.e., $\underline{f}_1 \rightarrow \underline{g}_1$, $\underline{f}_2 \rightarrow \underline{g}_2$, etc.) according to Equations 3 and 4. The resulting association matrix for this example, $\underline{A}$ in Equation 4, is also shown in Table 1.

In order to test the model's ability to associate, each of the four input vectors is presented as a stimulus to the model, and the resulting output vectors are calculated according to Equation 5. These computed output vectors are presented in Table 2. As indicated by comparing the computed output vectors with the original output vectors in Table 1, association is perfect for the orthogonal input vectors used in this example. That is, the computed output vectors are identical to the output vectors the model was originally taught to associate.

2.3 Measures of the Model's Performance

In order to assess the model's ability to associate different types of inputs and outputs and to assess its performance after simulated lesions, we need a quantitative measure of association performance. What we wish to know is how closely the output vectors computed by the model correspond to the original output vectors the model was taught to associate. Anderson et al. (1977) illustrated what they termed a crude way of measuring similarity between computed and original output vectors based on total vector length,

defined as:

$$L = \left[\sum_{i=1}^{N} g(i)^2 \right]^{1/2}$$

Using the eight-dimensional numerical example shown in Table 1, Anderson et al. created 1000 random input vectors, used them as stimuli for the model, and recorded the lengths of the resulting output vectors. Even this crude measure of similarity yielded at least some degree of discrimination among the four output vectors. However, many of the random input vectors generated output lengths identical or highly similar to those of the original output vectors. A better measure of performance is obviously needed.

More sensitive indices of performance could be based on any of the many available parametric and nonparametric measures of similarity (e.g., Mosteller and Rourke, 1973). The most natural measure in the context of the matrix algebra representation of the Anderson et al. model is the cosine of the angle between two vectors in N-dimensional space. When expressed as deviation scores around the means of the vectors, this measure is identical to the product-moment correlation coefficient used by Wood (1978) to assess the model's performance[3]. Note that perfect performance as defined by this meas-

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[3] The randomly generated input vectors used by Wood (1978) to test the model had zero mean and unit variance. Therefore, there was little difference between the product-moment correlation coefficient and the cosine of the angle between vectors for these data. However, with input vectors having means that differ systematically from zero the two measures would differ significantly.

ure is a correlation coefficient of 1.0. This value cannot, of course, be duplicated by any other output vector.

Because of its sensitivity and natural interpretation in the context of the model, the correlation coefficient was used as a performance measure by Wood (1978) and will form the basis of most of the data reported in this paper. It should be understood, however, that choice of a performance measure implies certain commitments about the model's interpretation. For example, the correlation coefficient is sensitive to the pattern of activity across the output neurons, not to the absolute amount of activity. This property reflects the fact that the correlation coefficient is, in effect, normalized over the variances of the two vectors. Thus, a computed output vector with greatly attenuated overall activity can still yield a good correlation to the original output vector, as long as the pattern of activity across the neurons in question is similar. If one wished to include absolute amount of activity as well as its pattern in the measure of similarity, covariances or cross-products could be used instead. Like the vector length measure discussed above, however, a given covariance can arise from a variety of different input vectors. For this reason, the correlation coefficient was used as the primary performance measure for assessing lesion effects.

The preceding discussion has emphasized measuring the similarity between the output vector generated by the model in response to a stimulus and the corresponding output vector the model was originally taught to associate. However, we also need to be concerned about the relationship between each computed output vector and other output vectors whose associations the model has learned. A combined measure of association, involving both "recognition" and "discrimination" performance can be obtained by forming the confusion matrix shown at the bottom of Table 2. This matrix consists of pairwise correlations among the computed and original output vectors. The diagonal elements in the confusion matrix may be thought of as an index of the model's "recognition" performance; that is, the degree to which the model responds to each input vector with the appropriate output vector. Thus, larger diagonal elements in the confusion matrix indicate better performance. As noted above, recognition performance is perfect ($r = 1.$) with orthogonal input vectors; with nonorthogonal inputs, however, the diagonal elements would be smaller as will be illustrated below. The off-diagonal elements in the confusion matrix may be thought of as an index of the model's "discrimination" performance, the degree to which the model responds to each input veccor with an output vector appropriate for some other input vector. In this case, lower correlations indicate better performance. A combined index of recognition and discrimination performance is given by the difference between the mean diagonal and mean off-diagonal elements of the confusion matrix. For the confusion matrix shown in Table 2, this value is $1.00 - .33 = .67$.

3. Simulated Lesion Experiments

Simulated lesions were made on elements of the model by assuming that the activity of each neuron included in the lesion would not contribute to the association process represented in Equation 5. This assumption was implemented by setting the appropriate element of the input or output vector in Equation 5 to zero.

This mathematical representation of a lesion is a gross oversimplification in a number of respects. First, lesions in real nervous systems not only eliminate the functional contribution of the specific neurons removed by the lesion, but may produce a variety of indirect effects upon remaining neurons. For example, Sprague (1966) showed that the effects of unilateral removal of superior colliculus in the cat could be offset by removal of the remaining colliculus. Similarly, Sherman (1974) reported that removal of visual cortex led to an improvment of visual deficits produced by visual deprivation. Indirect or "distant" effects of brain lesions were first emphasized in von Monokow's (1914) concept of "diaschisis" and have received increasing empirical attention in more recent investigations of functional recovery following brain damage (Eidelberg and Stein, 1974). Second, the effects of a lesion are not constant but change over time; some effects may disappear (i.e., "recover") whereas others may not (Eidelberg and Stein, 1974). Third, in addition to recovery at the behavioral level, recent anatomical studies have demonstrated clear evidence of gradual structural changes following brain damage, particularly when the lesions are sustained neonatally (Sidman and LaVail, 1974). For these reasons, the simulated lesions described here should be viewed as a static approximation to the complex, dynamic set of consequences induced by brain damage. Whether or not it will be profitable to attempt to model these dynamic effects of brain damage is an open question[4].

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[4] It would not be difficult to incorporate assumptions about lesion-induced structural changes into the model. At present, however, the available anatomical data are insufficient to constrain the many possible ways in which such an assumption could be implemented. For example, do all neurons undergo changes after a lesion or only those near the removed tissue? What are the rules by which new connections are established? Are they synaptically functional? In absence of relatively precise answers to these questions, it is not clear how valuable such an exercise would be.

3.1 Lesions of Neural Elements in the Model

Following a given lesion, the residual performance of the model was tested in the manner described above for the intact model: the four input vectors originally taught to the model were presented as stimuli and the four corresponding output vectors were recorded in response. The similarity between the computed and original output vectors was calculated from confusion matrices identical to that shown at the bottom of Table 2. Correlations were always based on N = 8, regardless of the number of neurons removed by the lesion.

To illustrate this process, Table 3 presents computed output vectors and confusion matrices for lesions of Input Neuron 1 and Output Neuron 8 in the numerical example presented above. Input Neuron 1 corresponds to the first element in the input vectors and the first column of the matrix shown in Table 1, whereas Output Neuron 8 corresponds to the eighth element in the output vectors and the eighth row of the matrix. Three qualitative aspects of the lesion effects shown in Table 3 should be noted: (1) Although both lesions produced a decrease in recognition performance, the model was nevertheless able to produce output vectors that closely approximated those produced by the intact model in Table 2. Whereas the mean of the diagonal elements for the intact model was 1.0, the means for the two lesioned models were .994 and .936, respectively. (2) As indicated by the off-diagonal elements in the confusion matrices, lesion effects on discrimination performance are more difficult to characterize in simple terms; they depend upon similarities of the specific input and output vectors employed. In general, a lesion which removes a neuron whose activity contributes heavily to the discrimination between two input vectors will have a greater effect on performance than a lesion which removes a neuron with less contribution. This effect is discussed in greater detail below. (3) Lesions of input and output vectors produce their effects on the model's performance in different ways. Setting the activity of an output neuron to zero directly eliminates the contribution of that neuron to the output vectors but leaves the other elements of the output vectors unaltered (compare Tables 3 and 2). This is a relatively trivial effect. In contrast, lesions of an input neuron have a more widespread but subtle effect on the pattern of output activity. In this case, all output neurons are still capable of generating activity, but the pattern of activity across the set of output neurons will generally differ somewhat from that of the intact model (again compare Tables 3 and 2). Lesions involving both input and output neurons combined these two types of effect.

3.2 Effects of Systematic Variations in Lesion Size and Location on Performance of the Model

As noted in the introduction, an important advantage provided by simulated lesion experiments is the ability to vary both lesion size and location precisely and systematically over a wide range. Equivalent anatomical precision is impossible to achieve in real lesion experiments. In terms of the Anderson et al. model, lesion size corresponds to the total number of neurons removed, whereas lesion location corresponds to the particular individual neurons removed. The simulated lesions reported by Wood (1978) and reviewed here were based on eight-element input and output vectors like those

used in the numerical example described above[5].

Lesion size and location were varied systematically by removing all possible combinations of 1-7 input neurons, all possible combinations of 1-7 output neurons, and all possible combinations of 1-7 input neurons and 1-7 output neurons combined. Thus, there were

$$\left[\sum_{i=0}^{7}\binom{8}{i}\right]^{2}-1$$

or 65,024 total lesions for a given set of input and output vectors. Because the deficit produced by a given lesion may be influenced by idiosyncratic features of the input and output vectors, the set of 65,024 lesions was repeated for each of 100 randomly selected sets of four intput and four output vectors. Each vector in a given set was selected independently from a Gaussian distribution with zero mean and unit variance, and input vectors were normalized according to Equation 2.

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[5] It is important to ask in this context whether the results of simulations using models of low dimensionality (i.e., with relatively small numbers of input and output neurons) can be generalized to those of higher dimensionality. As will be shown in greater detail below, the answer to this question is in general yes. More important than dimensionality per se in determining lesion effects is the relationship among the input and output vectors. Anderson et al. (1977) also used eight-element input and output vectors on the grounds that they were "large enough to be indicative of the behavior of a real system, yet small enough to be manageable and of reasonable cost" (p. 430).

Figure 2 illustrates the effects of lesion size, averaged over the 100 randomly selected input and output vectors and averaged over the specific neurons involved. Lesions of input neurons alone, output neurons alone, and combined lesions are shown. The upper left point indicates average performance of the intact model; the mean on-diagonal and off-diagonal values were .85 and .02, respectively. This result illustrates the point made above regarding orthogonality of the input vectors; when they are not orthogonal, recognition performance is less than perfect. Nevertheless, the .85 value shown here is a reasonable performance level for a model with small dimensionality.

Figure 2 also demonstrates that lesions of increasing size produced a corresponding increase in the size of the performance deficit for lesions of both input and output neurons. However, even with relatively large lesions performance remained well above chance on the correlation measure. Figure 3 illustrates the effects of the same lesions using cross-products instead of correlations as the similarity measure. The same general relationships are preserved, except that the decrement in performance with increasing lesion size appears somewhat larger in relative terms than that shown in Figure 2. This difference is simply a consequence of including the mean value of activity in the cross-product measure as well as the pattern of activity across neurons. Any lesion will tend to reduce overall activity, leading to larger deficits when cross-products are used instead of correlations as the performance measure.

Figure 4 summarizes the effect of lesion location independent of lesion size. The deficit produced by removal of any given neuron was, on average, equivalent to that of any other neuron. This relationship corresponds closely to Lashley's classic concept of "equipotentiality" (Lashley, 1929, 1933; Lashley and Wiley, 1933): "Equipotentiality of parts... [is] the capacity to carry out, with or without reduction in efficiency, the functions which are lost by destruction of the whole. This capacity varies from one area to another and with the character of the functions involved" (Lashley, 1929; p. 25). Similarly, the relationships between lesion size and magnitude of the performance deficit shown in Figures 2 and 3 are reminiscent of what Lashley termed "mass action": "Equipotentiality is not absolute but is subject to a law of mass action whereby the efficiency of performance of an entire complex function may be reduced in proportion to the extent of the brain injury within an area whose parts are not more specialized for one component of the function than for another" (Lashley, 1929; p. 25). I will return to these concepts in Section 4 below.

3.3 Effects of Specific Lesions on Specific Associations

The lesion effects just described were based on average results over a large number of individual lesions and many different input and output vectors. Can we expect the same pattern of results from a single set of input and output vectors? The answer is yes and no. It is yes when the input and output vectors are mutually orthogonal. In this case the deficit produced by any given input (or output) neuron closely approximates that produced by any other input (or output) neuron[6]. However, if the input or output vectors are

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6. For the case of mutually orthogonal inputs, Wood (1978) suggested that the deficit produced by a lesion of any neuron was "precisely equal" to that of any other neuron. This is incorrect, because it is possible to choose of set of input vectors that meet the global orthogonality criterion but in which the activity of one or more neurons is redundant across the set of vectors. Lesions including such neurons produce somewhat smaller deficits on average than lesions including neurons that are not redundant across the set.

highly correlated, marked departures from equal lesion effects can be obtained. Table 4 presents an example constructed by Wood (1978) to illustrate just how marked certain departures from equal effects can be.

In this example, input vectors $\underline{f}_1$ and $\underline{f}_2$ are distinguished from each other and from $\underline{f}_3$ and $\underline{f}_4$ by the activity of each of the eight input neurons. In contrast, input vectors $\underline{f}_3$ and $\underline{f}_4$ are distinguished from each other only by Input Neurons 1 and 2 and are identical for Input Neurons 2-8. Therefore, as the confusion matrices at the bottom of Table 4 illustrate, lesions of Input Neurons 1 and 2 produce a disproportionately large and selective effect on performance relative to lesions of the other input neurons. This effect is selective in that it is limited to associations between $\underline{f}_3 \rightarrow \underline{g}_3$ and $\underline{f}_4 \rightarrow \underline{g}_4$; lesions of Input Neurons 1 and 2 produce minimal deficits for vectors and . For the intact model, recognition performance for input vectors $\underline{f}_3$ and $\underline{f}_4$ was .89 and .92, respectively, whereas the corresponding values following a lesion of Input Neuron 1 were .91 and .52. In contrast, recognition performance for the same two input vectors was .53 and .96 following a lesion of Input Neuron 2. Table 4 also shows that confusion errors between input vectors $\underline{f}_3$ and $\underline{f}_4$ were significantly and selectively influenced by lesions of Input Neurons 1 and 2.

Examples such as the one in Table 4 can be generated ad infinitum and can be extended to include any number of neurons. For example, it is easy to see that a set of input vectors could be constructed so that lesions of, say, Input Neurons 3 and 4 would produce one highly selective effect, lesions of Input Neurons 5 and 6 would produce a different highly selective effect, and lesions of Input Neurons 1, 2, 7, and 8 would have roughly equivalent effects on performance. The important point, by way of summary, is that the pattern of performance deficit depends upon the mutual relationships between the input and output vectors. When the input vectors are orthogonal or nearly so, lesions of specific neurons have minimal selective effect on performance. However, when the input or output vectors can be distinguished by the activity of only a subset of the total population of neurons then highly selective deficits can be obtained. It should be emphasized that this conclusion applies regardless of the dimensionality of the model (i.e., the size of the input and output populations). Selective effects are less probable with models of larger dimensionality simply by virtue of the decreased likelihood of redundant sets of vectors of larger dimensions. However, highly selective effects can occur with a model of any dimensionality.

4. Implications for the Interpretation of Real Lesion Experiments

4.1 A Continuum of Lesion Effects from a Single Model

The lesion effects described in the preceding sections cover the entire continuum of those reported in real lesion experiments. At one end of the continuum are highly diffuse effects like those shown in Figures 6-8, in which removal of any individual neuron produces a roughly equivalent deficit to that of any other neuron and the magnitude of the deficit is roughly proportional to the total amount of tissue removed. As noted above, these results are similar to those from which Lashley derived the prinicples of "mass action" and "equipotentiality". At the other end of the continuum are highly selective effects in which removal of one neuron produced large deficits on one association and minimal effects on other associations. In fact, the results shown in Table 5 go beyond selective lesion effects to provide a clear example of the "double dissociation" result often interpreted as strong support for localization of function: "Genuine proof of specificity ('localization of function') always requires minimally some evidence of what Teuber has called 'double dissociation of symptoms' so that one lesion produces one set of symptoms and the other lesion another set" (Eidelberg and Stein, 1974, p. 208).

In light of this continuum of lesion effects produced by a single neural model, let us return now to the question with which we began: Given the mechanisms of information processing and storage that are built into the model by definition, to what extent can they be unambiguously inferred from behavioral deficits following brain damage? The mechanisms of information processing and storage of the Anderson et al. association model are stated clearly and concisely in Equations 1, 3, and 4, together with the assumption that every input neuron is synaptically connected to every output neuron. From these equations and the synaptic connectivity assumption, it is clear that the model's function, association, is <u>distributed</u> throughout the entire structure of the model and is not limited to specific subregions. That is, each input and output neuron performs exactly the same elementary operations (Equation 1) and all neurons in the input and output populations are involved in every association. Moreover, each output neuron operates on its inputs without regard to the operations of other output neurons. That is, no individual neuron has global information about the activity of the output population as a whole. Rather, the output is determined by "a population of neurons, none of which has more than local information as to which way the system should behave" (Arbib, 1980; p. 15; also see Pitts and McCulloch, 1947).

Given such a distributed processing system, we should expect that systematic variations in lesion size and location would produce effects similar to "mass action" and "equipotentiality" in Lashley's terms. Increasingly large lesions should produce increasingly large deficits in performance, and lesions of any given neuron should produce roughly equivalent deficits to lesions of any other neuron. This is exactly the pattern of results shown in Figures 2-4. For these results, then, the inferences one would be tempted to draw about functional organization would be in reasonable accord with the known mechanisms information processing and storage in the model.

Now let us consider the other end of the continuum of lesion effects exibited by the model. As noted above, the data for lesions of Input Neurons 1 and 2 in Table 4 constitute a "double dissociation of symptoms" by Teuber's definition. If interpreted in the traditional manner, one would infer from this pattern of results that association mechansims are highly localized. In the extreme, one might be tempted to conclude that the association of $\underline{f}_3$ with g_3 is mediated solely by Input Neuron 2 and association of $\underline{f}_4$ with g_4 is mediated solely by Input Neuron 1[7]. Yet the mechanisms of association embodied in Equations 1, 3, 4, and the connectivity assumption have not changed. The difference between the results in Table 5 suggesting localization of function and those in Figures 2-4 suggesting "mass action" and "equipotentiality" is

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[7] Dean (in press) and Wood (in press) have discussed the legitimacy of such an inference both in the general case and in the specific context of the simulated lesion experiments reported by Wood (1978).

not the mechansisms of association in the model, but the specific patterns of activity being associated.

An instructive exercise in this context is to view the input and output neurons in the Anderson et al. model as receptors and effectors in a simple nervous system having no interneurons. Whether the effects of a given lesion appear to indicate that assocation mechanisms are distributed or localized depends upon the specific stimuli (i.e., input vectors) and specific responses (i.e., output vectors) being associated. For example, consider a lesion experiment using the numerical example in Table 5 in which the only task investigated is discrimination between input vectors $\underline{f}_4$ and $\underline{f}_3$. As noted above, a lesion of either Input Neuron 1 or 2 produces a large deficit in discrimination between these two input vectors, whereas lesions of Input Neurons 3-8 produce little or no decrement in performance. From these results, one might be tempted to conclude that association mechanisms are highly localized. However, if the experiment had tested discrimination between $\underline{f}_4$ and $\underline{f}_1$, a very different conclusion might have been reached. In this case, discrimination performance is only slightly decreased following lesions of any of the input neurons and one might conclude that association mechanisms are more distributed. In both cases, however, the model's response to input vector $\underline{f}_4$ and the mechanisms of association of $\underline{f}_4$ with $\underline{g}_4$ are precisely identical. What differs in the two cases is the experimental context (in this case the alternative input and output vectors) in which the model's association of $\underline{f}_4$ with $\underline{g}_4$ is evaluated.

In summary, the fact that the entire continuum of empirical lesion effects can be obtained from this or any other model with no changes in structure of information processing mechanisms demonstrates a clear dissociation

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between the pattern of lesion deficits and underlying functional organization. This dissociation poses an important difficulty for attempts to infer principles of functional organization from lesion data alone. A persistent and specific behavioral deficit following removal of a given brain region is often regarded as demonstrating that the region in question plays a specific role in the functions that appear to be damaged following the lesion. Although such a conclusion may be correct, it is not the only conclusion that is consistent with the data. As the simulated lesion results demonstrate, highly selective lesion effects can also be obtained in nervous systems in which each neural element participates in a wide range of functions, depending upon task requirements (and probably a host of other variables in more realisitic models).

4.2 Distinguishing Between Deficit and "Function" in the Interpretation of Lesion Data

The conclusion that principles of functional organization cannot be directly inferred from lesion data is certainly not new, although the simulated lesion results do provide a particularly clear illustration of some aspects of the problem. Writing from the perspective of "The Brain as an Engineering Problem", Gregory (1961) presented a particularly clear logical analysis of the interpretation of lesion data. In this section I attempt to relate the simulated lesion results described above to the issues raised in Gregory's analysis. He posed the question in the following way: "Suppose we ablated or stimulated various parts of a complex man-made device, say a television receiving set. And suppose we had no prior knowledge of the manner of function of the type of device or machine involved. Could we by these means discover its manner of working?" (p. 320). Gregory's analysis of this question led him to the following conclusion: "Stimulation and ablation experiments may give direct information about pathways and projection areas, but their interpretation would seem to be extremely difficult, on logical grounds, where a mechanism is one of many inter-related systems, for then changes in the output will not in general be simply the loss of the contribution normally made by the extirpated area. The system may now show quite different properties" (p. 325).

Failure to distinguish between the pattern of behavioral deficits produced by removal of a given region and the functions of that region in the normal operation of the brain as a whole can lead to serious interpretive problems. This distinction is also not a new one. In the context of human language function, Hughlings Jackson (1874) wrote: "to locate damage which destroys speech and to locate speech are two different things". Gregory puts the distinction this way: "Although the effect of particular type of ablation may be specific and repeatable, it does not follow that the causal connection is simple, or even that the region of the brain affected would, if we knew more, be regarded as functionally important for the output... which is observed to be upset. It could be the case that some important part of the mechanisms subserving the behavior is upset by the damage although it is most indirectly related, and it is just this which makes the discovery of a fault in a complex machine so difficult" (Gregory, 1961; p. 323).

We have little difficulty making the distinction between deficit and function for peripheral parts of the nervous system whose functional roles are, relatively speaking, reasonably well understood. In this case failure to distinguish between deficit and function leads to obvious absurdities.

For example, complete bilateral section of the optic nerves produces total loss of vision, yet few would conclude that the function of the optic nerves is "vision". The optic nerves obviously play an important role in vision, but that role is not identical to "vision" as a whole nor can that role be deduced from the deficits caused by optic nerve section.

For regions of the nervous system not located near the periphery, we seem much more willing to make direct inferences from the pattern of deficit to hypothesized functional organization. At least three reasons can be cited for such willingness: (a) we know less a priori about the fuctions performed by central regions of the nervous system; (b) for central regions there are fewer obvious anatomical constraints of the type available for peripheral regions (e.g., all information from the retina to the brain travels via the optic nerves); and (c) the pattern of deficit following central lesions is usually more subtle and complex than for lesions in the periphery. Broca's area provides a particularly clear example in the context of this conference. Based on language production deficits following damage to the postero-lateral portion of the left frontal lobe, Broca (1861) and subsequent workers proposed a model "that has had a tenacious hold on the minds of most students of aphasia ever since. According to this view, the third frontal gyrus... *contains the motor programs for speech*" (Levine and Sweet, this conference; my italics). It is not difficult to find contemporary examples of such a model. For example, Geschwind (1979) writes: "Much new information has been added in the past 100 years, but the general principles Wernicke elaborated still seem valid. In this model the underlying structure of an utterance arises in Wernicke's area. It is then transferred through the arcuate fasciculus to Broca's area, where it evokes *a detailed and coordinated program for* vocalization" (p. 187, my italics). Similarly, Schnitzer (this conference) discussed "evidence for considering Wernicke's Area to be the site of... the semantic or linguistic predication function and for considering Broca's area to be the site of what I have been calling the 'housekeeping' function... traditionally called (surface) syntactic, morphological, and morphophonemic rules or constraints" (p. 6 and 5, italics mine). These interpretations may very well be correct, but it should be understood that they are based upon the same direct inference from deficit to function that seems so inappropriate in the optic nerve example given above.

An extreme example of the failure to distinguish between deficit and function is to define a region's function in terms of the behavioral deficits produced by its removal. Eidelberg and Stein (1974) make a similar point in the context of "recovery of function": "A serious semantic stumbling block may lie in the lack of agreement between neurophysiologists and behavioral scientists on what is meant by 'the function' of a particular neural system. The danger of circularity in operational definitions is nowhere more obvious, for if 'the function' of a set of neurons is defined by the permanent deficit in performance that remains after its removal, the conclusion that true functional recovery may not occur is inescapable, because that was the hidden premise of the argument" (pp. 234-235). The same is true of inferences from deficits to normal function. If the function of a given region is defined in terms of the behavioral deficits following its removal, then it is difficult to avoid the conclusion that certain functions are localized in certain brain regions even if the actual organization of the system is quite different. Gregory presented the following example which illustrates the logical absurdity to which such an approach leads: "...the removal of any of several

widely spaced resistors may cause a radio set to emit howls, but it does not follow that howls are immediately associated with these resistors, or indeed that the causal relation is anything but the most indirect. In particular, we should not say that the function of the resistors in the normal circuit is to inhibit howling" (Gregory, 1961; p. 323).

Thus, the term "function" as used in the context of lesion data appears to have two distinct meanings that are often confused. The first is the function of a given brain region as it would be specifed if we knew how the entire system worked. This type of specification is consistent with an engineering level description of how a system operates (cf., Gregory, 1961) and appears to be what many investigators have in mind by the term "functional organization". The second meaning is the function of a given region defined in terms of behavioral deficits produced by its removal. To define "function" from the first perspective we need to know the global function(s) and input-output relations of the system as a whole, the elementary functions or operations performed by individual components of the system, and the structural and functional relationships among individual components of the system that are the basis of the functions of the whole. Yet what we learn from lesion experiments is limited to whether or not removal or a given brain region produces reliable deficits in the behavior or behaviors tested.

Let us consider the two general outcomes of a lesion experiment in which the effects of one lesion on one behavior are assessed. If the lesion produces a reliable behavioral deficit, then we are justified in concluding only that the region is in some unspecified way involved in the production of that behavior. This involvement could be direct, in the sense that if we knew how the brain worked as a whole we would conclude that the region plays a causal role in the production of the behavior in question. However, as in Gregory's "howl inhibitor" example the involvement could be very indirect and the observed deficits could be produced by a very circuitous route indeed. A lesion that produces a reliable behavioral deficit is sometimes said to demonstrate that the damaged tissue is *necessary* for production of the behavior. This conclusion is correct as far as it goes, but it should be viewed as tentative because very different conclusions would be drawn if additional lesions are found to alter or eliminate the deficit (e.g., Sprague, 1966; Sherman, 1974).

Alternatively, if the lesion produces no detectable deficit, we cannot conclude that the region plays no functional role in the behavior tested. The issue here is more than concluding in favor of the null hypothesis, although that problem is involved as well. Knowing that a given lesion produces no detectable effect on a given behavior is evidence only that the region is *not necessary* for production of that behavior and that remaining intact regions are *sufficient*. Such a result does not demonstrate that the region is uninvolved. The simulated lesion results described above provide a clear example of this situation. Some lesions produced little effect on the association between a given input vector and a given output vector (see Table 4), even though at the level of "how the system works" (i.e., Equations 1, 3, 4, and the connectivity assumption) every neuron participates in every memory.

4.3 Relationship Between Deficit and Function in Distributed Systems

Figure 5 is an illustration used by Gregory to show the different ways in which an engineer might represent the structure and function of a given

system. The top section is a "blue print" consisting of pictoral representation of components and their connections. This representation roughly corresponds to a schematic description an anatomist might provide of the brain. The middle section is a "circuit diagram" in which the functional properties of the components are given instead of their anatomical structure. This representation roughly corresponds to a description a physiologist might give in which excitatory and inhibitory connections are substituted for pictoral representations of synapses, etc. The bottom section is a "block diagram" in which individual components of the system and their connections have been replaced by functional units of organization and their logical relationships. Gregory suggests that the latter may be compared with cybernetic descriptions of the brain, although somewhat more loosely we might say it corresponds to the level of functional specification that is the goal of much psychological experimentation.

These three ways of representing the structural and functional relationships among components of a system provide a basis for illustrating the added interpretive problems associated with lesions in distributed systems. In both the top and middle sections of of Figure 5, the components represented in the diagram correspond closely to the observable structure of individual elements of the system in question: both the "blue print" and "circuit diagram" are schematized to some degree, but the essential structural features of the actual system are preserved. In the "block diagram" of the bottom section, however, the structural relationships are no longer evident. If the workings of the system are known, it is possible to translate between the "block diagram" and the other two types of representation and vice versa. However, if the workings of the system are not known, such a translation can be difficult even if satisfactory "blue prints" and "circuit diagrams" are available. If the system is relatively simple and each component identifiable in the "blue print" or "circuit diagrams" contributes to one and only one functional component in the "block diagram" (as is the case in Figure 5), then translation from one representation to another may be achievable without great difficulty. In this case, lesions of components defined in structural terms will tend to produce damagealong functionally meaningful lines. However, if each structural component contributes to multiple functional components, then translation from one representation to the other will be exceedingly difficult and lesions will not tend to damage the system along functionally meaningful lines.

In summary, my purpose in this section has been to view the simulated lesion results in the more general context provided by Gregory's (1961) analysis of lesion interpretation from an engineering perspective. I have attempted to distinguish between two different ways in which the term function is used in the context of lesion data, the first based on complete knowledge of how a complex system works and the second based on observations of a system's malfunctions following damage to its parts. If we attempt to use lesion data to make inferences about function defined in the first sense we run the risk of making two types of interpretive errors: (a) attributing direct involvement of a given region in a given behavior when its involvement is in fact indirect; and (b) failing to detect the involvement of a region whose contribution is clear if the functional organization of the system is known but which shows little or no detectable lesion effect. The latter error is powerfully illustrated by the simulated lesion results in which removal of one or more neurons produces little or no disruption in association.

Finally, I have attempted to show how the lack of one-to-one mapping between structural and functional representations in distributed systems can create even more difficult interpretative problems.

Systematic lesion experiments are important in my opinion, but not because they provide an unambiguous means of inferring the functional role of the brain structures removed. Rather, the results of such experiments constitute a stringent criterion by which any theory of the functional organization of the brain should be judged.

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FIGURE LEGENDS

Figure 1: Schematic representation of the structural assumptions of the Anderson et al. model. Each neuron in input population is synaptically connected to each neuron in output population . (From Anderson et al., 1977).

Figure 2: Effects of variations in lesion size on performance of the model using a correlation-based measure of performance. (From Wood, 1978).

Figure 3: Effects of variations in lesion size on performance of the model using a performance measure based on cross-products instead of correlations.

Figure 4: Effect of lesion location on performance of the model, averaged over lesion size. (From Wood, 1978).

Figure 5: Three ways of representing the structure and function of a system. (From Gregory, 1961).

Fig. 1

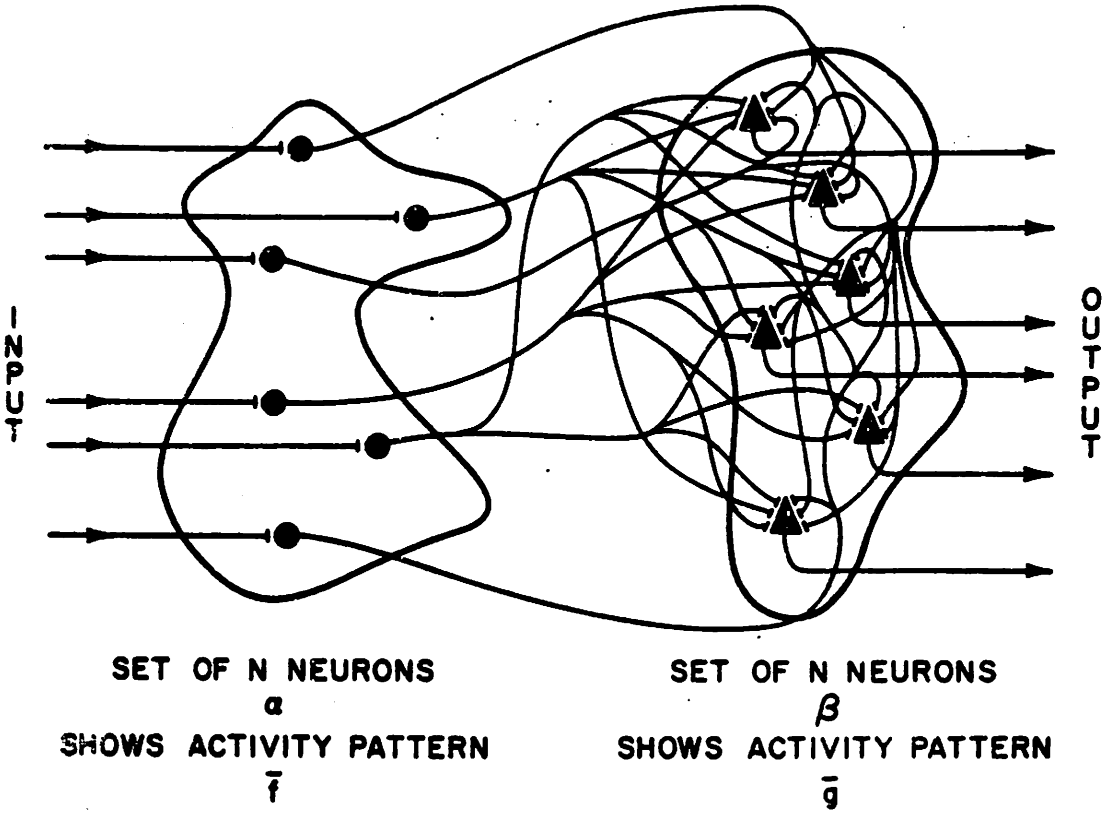
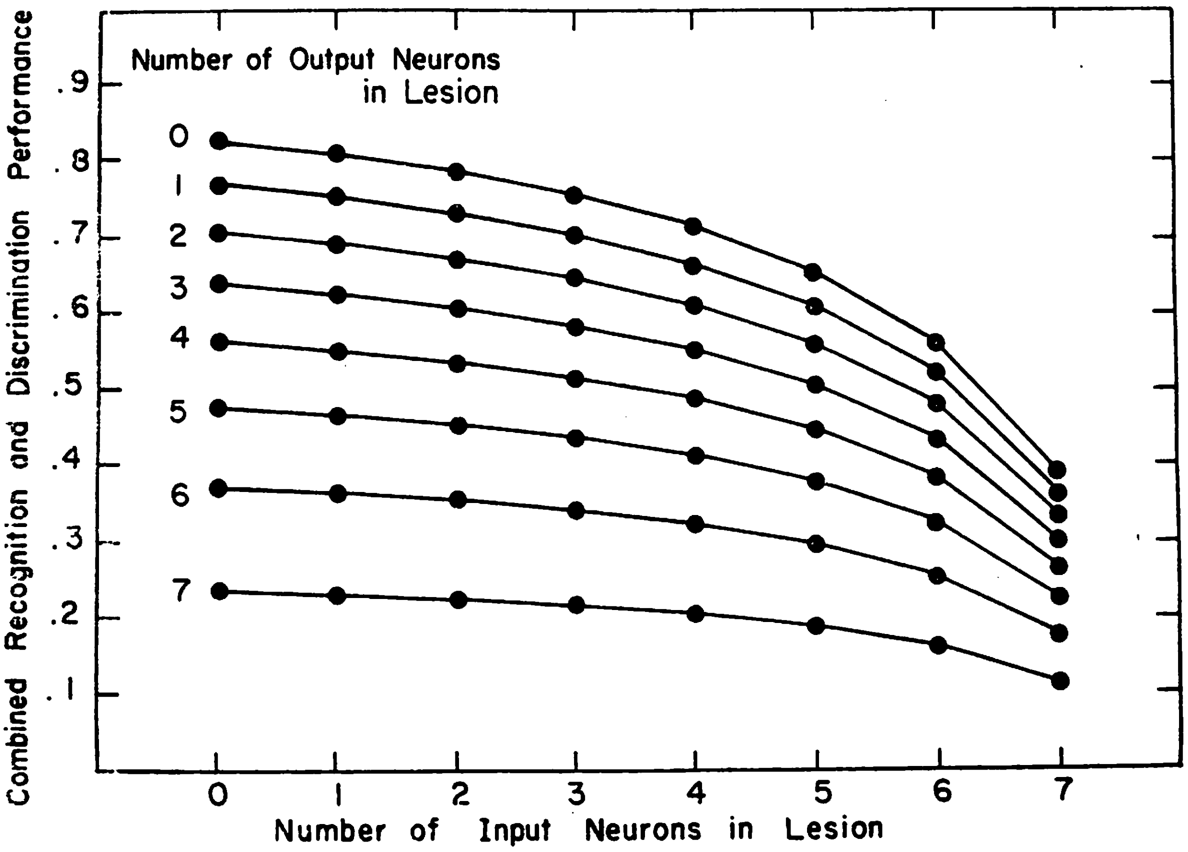

INPUT
OUTPUT
SET OF N NEURONS
α
SHOWS ACTIVITY PATTERN
f̄
SET OF N NEURONS
β
SHOWS ACTIVITY PATTERN
ḡ

Fig. 2


Combined Recognition and Discrimination Performance
.9
.8
.7
.6
.5
.4
.3
.2
.1
Number of Output Neurons in Lesion
0
1
2
3
4
5
6
7
0
1
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3
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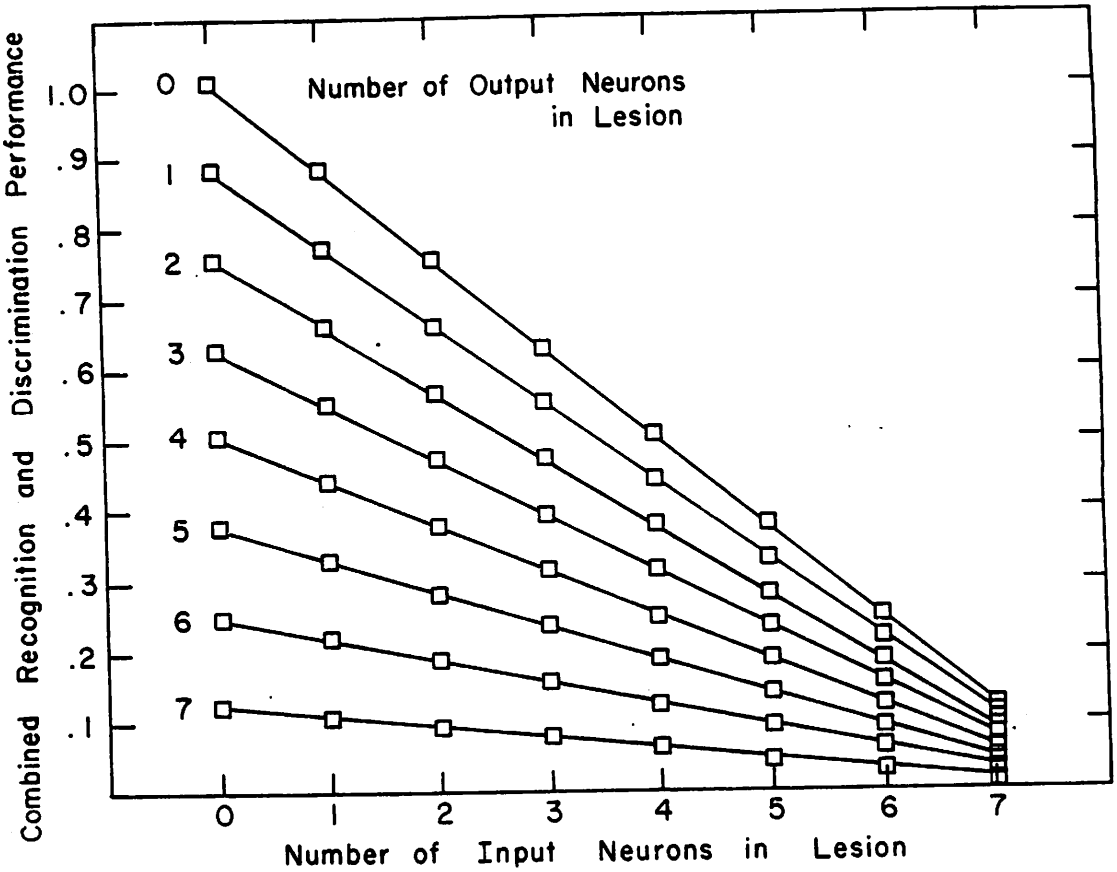
5
6
7
Number of Input Neurons in Lesion

Fig. 3


Combined Recognition and Discrimination Performance
1.0
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Number of Output Neurons in Lesion
0
1
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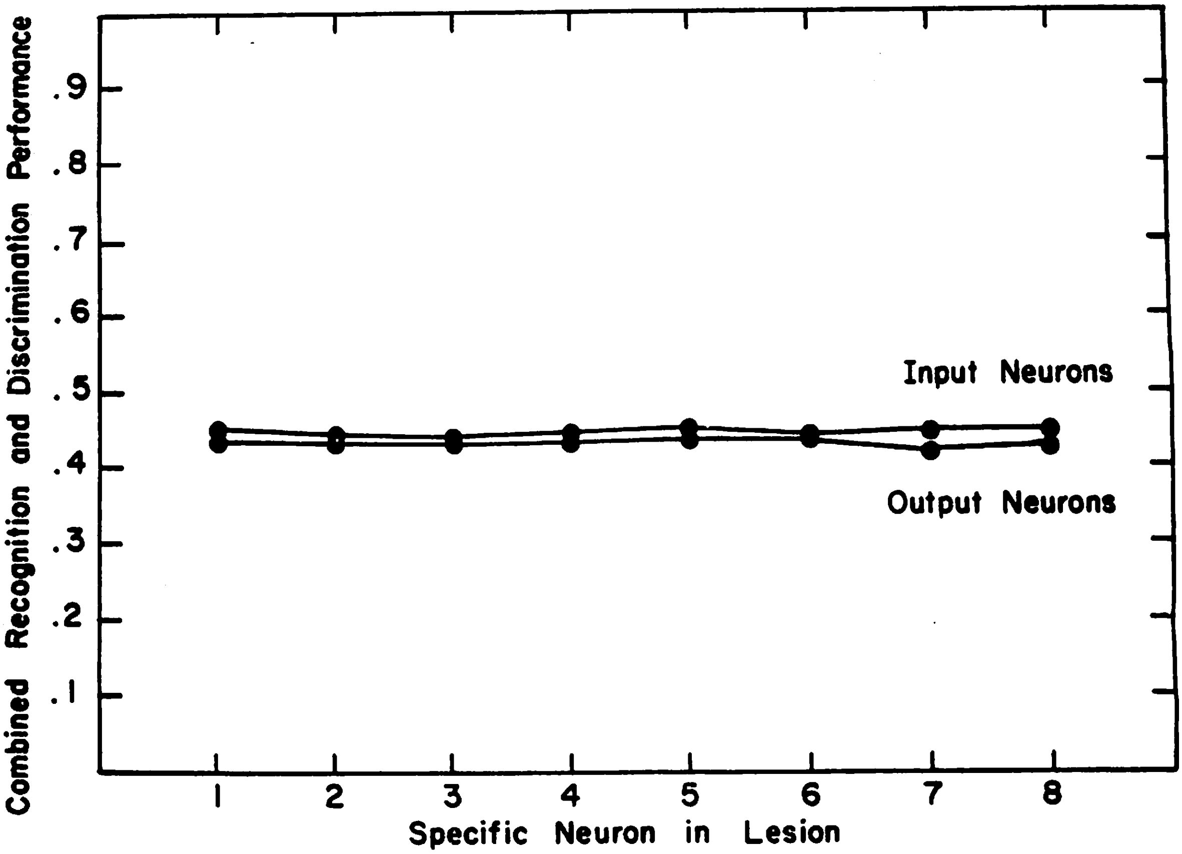
6
7
Number of Input Neurons in Lesion

Fig. 4


Combined Recognition and Discrimination Performance
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Input Neurons
Output Neurons
1
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8
Specific Neuron in Lesion

Fig. 5

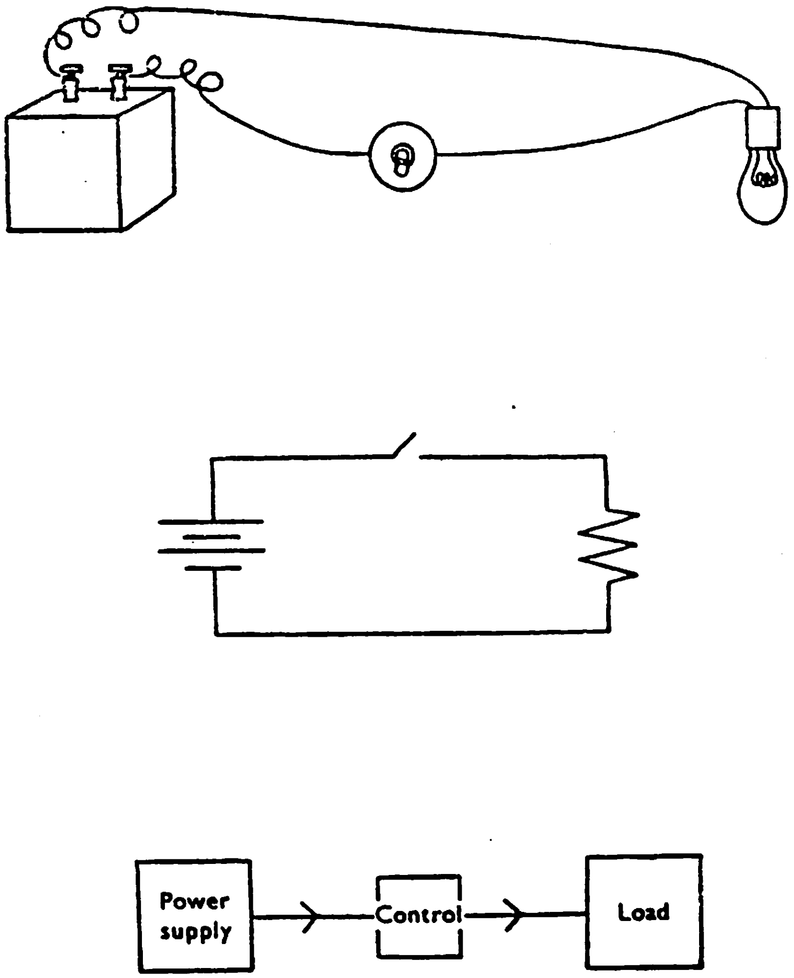

Table 1 *Input Vectors, Output Vectors, and Association Matrix for a Numerical Example*

Input vectors				Output vectors			
f_1	f_2	f_3	f_4	g_1	g_2	g_3	g_4
.354	−.354	.354	−.354	1.000	−1.000	3.000	4.000
.354	−.354	−.354	−.354	.000	2.000	.000	.000
.354	.354	.354	.354	−1.000	.000	−1.000	−1.000
.354	.354	−.354	.354	.000	−1.000	−1.000	−1.000
−.354	−.354	.354	.354	1.000	−1.000	−2.000	−1.000
−.354	−.354	−.354	.354	−1.000	−1.000	.000	.000
−.354	.354	.354	−.354	−1.000	−1.000	−1.000	.000
−.354	.354	−.354	−.354	.000	2.000	2.000	1.000
Association matrix							
.354	−1.768	2.475	.354	2.475	.354	−1.061	−3.182
−.707	−.707	.707	.707	−.707	−.707	.707	.707
−.354	.354	−1.061	−.354	−.354	.354	.354	1.061
.354	1.061	−1.061	−.354	−.354	.354	−.354	.354
.354	1.768	−1.061	.354	−1.061	.354	−1.061	.354
.000	.000	−.707	−.707	.707	.707	.000	.000
−.354	.354	−1.061	−.354	.354	1.061	−.354	.354
−.354	−1.768	1.768	.354	.354	−1.061	1.061	−.354

Table 2

Computed Output Vectors and Confusion Matrix for the Numerical Example

Computed output vectors			
$\hat{g}_1$	$\hat{g}_2$	$\hat{g}_3$	$\hat{g}_4$
1.000	−1.000	3.000	4.000
.000	2.000	.000	.000
−1.000	.000	−1.000	−1.000
.000	−1.000	−1.000	−1.000
1.000	−1.000	−2.000	−1.000
−1.000	−1.000	.000	.000
−1.000	−1.000	−1.000	.000
.000	2.000	2.000	1.000

Original output vectors	Confusion matrix Computed output vectors			
	$\hat{g}_1$	$\hat{g}_2$	$\hat{g}_3$	$\hat{g}_4$
g_1	1.000	−.016	.304	.436
g_2	−.016	1.000	.312	.016
g_3	.304	.312	1.000	.911
g_4	.436	.016	.911	1.000

Table 3

Computed Output Vectors and Confusion Matrices for Lesions of Input Neuron 1 and Output Neuron 8 in the Numerical Example

Computed output vectors for lesion of Input Neuron 1				Computed output vectors for lesion of Output Neuron 8			
$\hat{g}_1$	$\hat{g}_2$	$\hat{g}_3$	$\hat{g}_4$	$\hat{g}_1$	$\hat{g}_2$	$\hat{g}_3$	$\hat{g}_4$
.875	−.875	2.875	4.125	1.000	−1.000	3.000	4.000
.250	1.750	.250	−.250	.000	2.000	.000	.000
−.875	−.125	−.875	−1.125	−1.000	.000	−1.000	−1.000
−.125	−.875	−1.125	−.875	.000	−1.000	−1.000	−1.000
.875	−.875	−2.125	−.875	1.000	−1.000	−2.000	−1.000
−1.000	−1.000	.000	.000	−1.000	−1.000	.000	.000
−.875	−1.125	−.875	−.125	−1.000	−1.000	−1.000	.000
.125	1.875	2.125	.875	.000	.000	.000	.000

Confusion matrices

Original output vectors	Computed output vectors				Computed output vectors			
	$\hat{g}_1$	$\hat{g}_2$	$\hat{g}_3$	$\hat{g}_4$	$\hat{g}_1$	$\hat{g}_2$	$\hat{g}_3$	$\hat{g}_4$
g_1	.987	.047	.225	.475	1.000	−.061	.316	.430
g_2	.125	.997	.374	−.048	−.016	.875	.053	−.120
g_3	.332	.338	.996	.894	.304	.080	.909	.823
g_4	.441	.038	.892	.996	.436	−.101	.949	.977

Table 4

Input Vectors, Output Vectors, and Confusion Matrices Illustrating Localization of Function

Input vectors				Output vectors			
f_1	f_2	f_3	f_4	g_1	g_2	g_3	g_4
−.196	−.229	.114	.912	1.000	−1.000	1.000	−1.000
.000	−.459	.912	.114	1.000	−1.000	−1.000	−1.000
−.196	.459	−.114	−.114	1.000	1.000	1.000	1.000
.392	.229	.228	.228	1.000	1.000	−1.000	1.000
.558	−.459	.114	.114	−1.000	−1.000	1.000	1.000
.558	.000	−.114	−.114	−1.000	−1.000	−1.000	1.000
−.196	−.229	−.114	−.114	−1.000	1.000	1.000	−1.000
−.196	.459	.228	.228	−1.000	1.000	−1.000	−1.000

Confusion matrices

Original output vectors	Computed output vectors for lesion of Input Neuron 1				Computed output vectors for lesion of Input Neuron 2			
	$\hat{g}_1$	$\hat{g}_2$	$\hat{g}_3$	$\hat{g}_4$	$\hat{g}_1$	$\hat{g}_2$	$\hat{g}_3$	$\hat{g}_4$
g_1	.943	−.299	.083	.276	.960	−.319	.209	−.087
g_2	−.309	.898	−.315	.081	−.259	.933	.163	−.127
g_3	.088	−.322	.914	.803	.064	.062	.526	.251
g_4	.088	.025	.241	.522	−.086	−.154	.809	.956

ANDREW KERTESZ: COMMENTS ON WOOD PRESENTATION

I think Wood's model has an elegant simplicity and teaches us a great deal. Nonetheless, I worry that it is too simple. It lacks the diversity of the real nervous system, and does not include the nervous system's subtle compensating mechanisms. Those of us who have followed patients over a long period of time learn to think of lesions as initiating a dynamic process rather than static symptoms.

Wood has shown that a simple net can encompass a wide range of lesion effects. How much more could a brain with 15 billion neurons show? Further models should include such extra structure as columnar organization, feedback, and gating effects. How far should we go?

In the rest of this talk, I want to briefly review methods for aphasiology developed in the 70s (reviewed in my book "Aphasia and Associated Disorders: Taxonomy, Localization and Recovery," Grune and Stratton, 1979). We use standardized measurement of behavior and classify aphasics into groups on the basis of objective taxonomy rather than subjective clinical impressions. A wonderful new tool has been provided by the CT scan which allows precise description of lesions, which can be followed in the live patient while his symptoms are developing. Isotope scans are useful in acute cases, yielding good results for at most two months after a stroke (tumor cases are too messy to discuss here). By contrast, the CT (Computerized Tomography) scan improves with time, achieving sharp localization around four weeks. As the technique develops, we should be able to localize lesions to parts of gyri. Positron Emission Tomography (PET) scans are expensive to acquire, requiring access to a particle accelerator, but let one examine the functional state of nervous tissue.

We have studied the range of lesions in Broca's aphasia via numerical taxonomy. Agrammatism, paraphasia rating, word length, etc., are some of the features used in assigning a numerical rating. For each person scored as a Broca's aphasic, a lesion localization is made from an isotope scan by a radiologist (not an aphasiologist, to avoid prejudgement). A few of the lesions spare the classical Broca's area. However, the crucial point is that when we overlap all 25 projections, the total lesion density is highest in exactly Broca's area. Similarly, patients diagnosed as Wernicke's aphasics by numerical taxonomy yield a pattern of composite localizations centered on Wernicke's area. These lesions are not as localized as in the Broca's case; but many of the patients became anomics after several months. With a density cutoff of 75 to 80%, we get disjoint composites for the Broca's and Wernicke's aphasics. Finally, let me note that, in accord with Lashley's Law of Mass Action, we have found a significant correlation between the area and severity of lesions in aphasia.

DISCUSSION OF WOOD PAPER

Galaburda: Regions aren't islands. Each lesion has distant effects, and plasticity further complicates the picture.

Levine: The development of Wood's model should allow for synaptic reorganization after the lesion. Vacated synapses on output neurons, made available when some of its input neurons are destroyed, may be filled by outgrowths from other input neurons. I would also like to see the two hemispheres modelled via two, not quite equally efficient, subnets in interaction.

Whitaker: To think about equipotentiality, imagine a quarter-sized lesion anywhere in the language system. You will see similar degradation as measured by a single overall number, but we can discriminate each lesion if we describe its effects in more precise terms.

Arbib: We should not read too much into the fact that lesions to Wood's net exhibit mass action and equipotentiality. After all, the nets were designed to be equipotential -- all-to-all connections, and a homogeneous task. More interesting would be to understand with more structured nets what measures homogenize the effects of the lesions, so that we may avoid these in designing measures which can help us localize the effects of damage.

Kertesz: If you correlate the effects of lesions across an entire group of patients, you get a strong argument for equipotentiality. But in specific cases, you can see devastating effects of a small lesion.

Woods: Kertesz seems misled by the fact that there are only 16 cells in Wood's model. Wood uses a simple net for the same reason that some neurophysiologists study Aplysia -- to discover principles that can be extrapolated to larger cases. One development would generalize the all-to-all connectivity matrix to one which is sparse, with anatomy forcing blocks of zeros for regions that are not connected. We can then charge the matrix to express experimental constraints. Of course, getting language into such a net is a deeper problem.

Wood: I did not explicitly address the question of the anatomical elements to which the Andersonmodel should be applied. The most obvious relationship is between neural elements in the model and individual nerve cells in the brain. But one might also wish to identify elements in the model with neural circuits containing more than one cell, e.g. cortical columns. The key aspect of the model is the representation of information in cruclation matrix form, regardless of how this assumption is realized in structural terms. My own bias is to attempt to learn as much as possible from the abstract properties of the model without attempting toapply it as a literal process model of particular structures.

Sensorimotor Processes and the Neural Basis of Language[1]

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Abstract

We establish a perspective on neurolinguistics which seeks models of the cooperative computation between diverse brain regions, and which seeks an evolutionary relationship between "action-oriented perception" and "language use". We argue that syntax emerges as a symptom of a process of translation from a semantic goal to an utterance, and that this translation should be viewed as an ongoing planning process. An analysis of visually-guided locomotion introduces the concept of the action-perception cycle, which provides an analogy for the cycle of conversation. After an overview of the neural analysis of sensorimotor processes, we provide a comparative analysis of object naming in humans and prey-predator discrimination in amphibia. On this basis, we argue that neural models of sensorimotor processes in animals provide clues as to the neural processes involved in language representation and utilization.

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[1] Preparation of this paper was supported in part by the Sloan Foundation grant for "A Program in Language and Information Processing" at the University of Massachusetts at Amherst.

Introduction

Neurolinguistics developed in the context of clinical medicine with the goal of predicting lesion-sites from symptom-complexes and vice-versa. But, in the words of Hughlings Jackson [1874], "to locate damage which destroys speech and to locate speech are two different things." Luria [1973] -- developing the idea of 'functional system' from the work of Anokhin [1935], Bernstein [1935] and Vygotsky [1934] -- asserts that our fundamental task is to ascertain "which groups of concertedly working zones are responsible for the performance of complex mental activity [and] what contribution is made by each of these zones to the complex functional system." This transfers the emphasis from the brain-damaged patient to the normal subject. We seek a theory of how brain regions interact in some normal performance. The performance of the patient with damage to one of these regions (but note the scant respect lesions and tumours show for the boundaries of such regions) is then to be explained in terms of the interaction between the remaining brain regions, rather than in terms of the properties of the region removed. The system must still be able to perform despite deletion of some portion -- quite unlike the total breakdown that would follow removal of a subroutine from a serial computer program (cf. Arbib [1975, Sec. 5]). One should append additional mechanisms to the model to account for the effects of damage only once it has been shown that the properties of the remaining regions will not automatically account for these effects -- the only delicacy being that it may require a rather subtle theory to determine just what does follow 'automatically'. For this reason, we should be cautious about Lecours' explicit postulation of "error generators" as underlying phonemic paraphasias (Lecours et al. [1969, 1973]). Note, however, that it is the data on abnormal behavior that provide some of our best clues about the neurological validity of processes postulated in a model of the normal, so that it would be a poor strategy to build a neural model for normal function without reference to the neurological data.

One's perspective on neurolinguistics would seem to depend a great deal on one's starting point. We have seen that the classical starting point is the neurological clinic. My own starting point is the study of neural mechanisms of visuomotor coordination and an evolutionary perspective which leads me to search for common mechanisms for "action-oriented perception" (Arbib [1972]) and "language use" to provide a base from which to explore their differences. Thus, rather than asserting the existence of a separable grammar which interacts with processes for understanding or production, I would rather see a variety of grammars (e.g. in production and perception) whereby "internal Models" of meaning are related to utterances of the language via a 'translation' process. This hypothesis is based on a view of language as evolving in a context of well-developed cognitive abilities (which are then modified in turn, just as visuomotor processes in superior colliculus are modified by descending pathways from visual cortex). In total contrast are those linguists and philosophers who look for an "autonomous" theory of language processing. They reject the thesis that diverse information is used in language processing and see the core of the linguistic processor to be the generation of phonological, morphological, syntactic and logical representations, with the diverse non-linguistic information exploited in everyday language use inaccessible to this core. This thesis seems to have evolved from Chomsky's original claim [1965, Sec. I.3] for autonomy of syntax, a claim based on a divorce of linguistics from processing, let alone neurological, issues. Without wishing to discourage intensive

efforts in neurolinguistics which are primarily linguistic, I would suggest that these can only be part of the overall emphasis when the goal is to understand the interaction of multiple brain regions. Here I believe that the neurologist, neuroanatomist and brain theorist have much to offer that the linguist does not.

The integrated theory I envisage will incorporate a great deal of neuroanatomy as we try to better characterize regions within the brain and the detailed projections that link them. But what manner of "region" will enter meaningfully into neurolinguistics? Such gross anatomical regions as the parietal lobe or the cerebellum are too large, while individual "columns", let alone individual neurons, are too fine to make contact with conceptualizations at the level we diagram below. (Does a word in the lexicon have its own set of columns in the cortex? Would such a set contain one column or many thousands. It is perhaps premature to speculate but we find some clues in a recent study of changing visuomotor representations in the cortex of the cat (Spinelli and Jensen [1979]).) Classic cytoarchitectonics and modern biochemistry provide different answers for differentiating the brain into a multiplicity of separable populations. Figure 19 of Szentágothai and Arbib [1975] shows the neuron network of the cerebellar cortex schematically transformed into five two-dimensional matrices, reminding us of the insight to be gained by subdividing a region into interacting layers; while Boylls' synergy controller model (ibid., Ch. V) suggests that the cerebellar cortex in isolation cannot be viewed as a meaningful unit in motor control, and that it is only in relation to a number of adjacent nuclei that its posited role in the adjustment of motor synergies can be defined. What is interesting for our discussion of methodology is that Boylls' choice of units resulted from the confluence of a theoretical analysis refining the

Bernsteinian theory of synergies (Bernstein [1935, 1967]) and a detailed review of the constraints and mechanisms documented in the anatomical and physiological literature.

The paper by Arbib and Caplan [1979], and the attendant commentaries, suggested the following conclusions about neurolinguistics:

1. There is a body of moderately reliable information relating symptom-complexes to localized lesions, but much needs to be done to relate symptom-complexes to the interaction of remaining brain regions rather than to properties of the site of the lesion. It is an article of faith shared by most neurolinguists that such an analysis is in principle possible.
2. There is a body of psycholinguistic research which seeks to refine linguistic categories to provide clues to the "neural code" of language processing. The neural validity of many of the posited codes is still controversial.
3. A new framework is needed to develop, modify, and integrate the approaches outlined in (1) and (2). Arbib and Caplan suggested that precise models using the language of cooperative computation (based on studies in both brain theory and artificial intelligence) may provide such a framework. This proposal needs full experimental testing on the basis of detailed modelling.
4. Computational models are abstract models and must not be confused with crude comparisons of brain and computer. Much can be learned from computational models by pencil-and-paper theorizing, but computer simulation should allow more detailed study of their properties.

5. Neurolinguistics has been too isolated from general issues of neural modelling, and from an appreciation of the relevance of issues in visual perception and motor control. We argue that the cooperative computation style of modelling can integrate neurolinguistics with studies of visuomotor coordination, and that the "modules" posited in a C^2-model can provide a bridge to synapse-cell-circuit neuroscience.

Syntax as a Symptom of Translation

Many formal accounts of grammar appear to offer a "give me an S, give me an N, ..." approach to sentence production. A base component "grows" a tree from an S at the root, or describes a path from an initial node in some network, and then further processes elaborate both the semantics and the surface structure of the deep structure (be it tree or labelled path) so obtained. However, I would argue that it is more fruitful for psycholinguistics and neurolinguistics to view sentence production as a process of translation from an often ill-defined goal structure to a (relatively) well-formed sentence of the language. I shall illustrate this in the present section by the analysis of a simple statement in the programming language PASCAL (though my analysis should be intelligible to readers unfamiliar with the language). Then, in the next section, I shall relate this posited translation process to planning concepts developed in Artificial Intelligence (AI), a branch of computer science concerned with the design of programs that exhibit certain aspects of intelligence.

PASCAL syntax contains many rules, including

<statement> ::= begin <statement>; <statement> end

<statement> ::= while <expression> do <statement>

<statement> ::= <variable> := <expression>

as well as rules for <variable> and <expression> that let us "grow" the PASCAL program

```
(1)      begin r := x;
               while r ≥ y do r := r-y
         end
```

according to the syntactic tree shown in Figure 1. In fact, PASCAL syntax does not adequately constrain the production of statements, e.g. by proper placement of arithmetic versus Boolean expressions. But the point here is that the program (1) was not obtained as shown in Figure 1, but by the

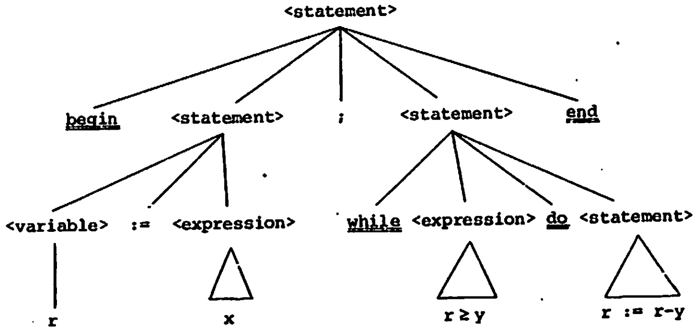


Figure 1. A PASCAL Syntactic Tree.

following process of "top-down design".

We are given the goal of designing a PASCAL statement which will take numbers $x \geq 0$ and $y > 0$ and set a variable r to the remainder x mod y when x is divided by y. We decide to do this by subtracting y from x repeatedly so long as $x \geq y$; and we decompose this into

a) Initialize r to the given value of x; and

b) So long as r has a value ≥ y, decrease that value by y.

We then call on our knowledge of PASCAL to elaborate (a) and (b) respectively, as

(2) r := x

(3) while r ≥ y do r := r-y .

We know that the PASCAL syntax for "do S_1 then do S_2" is begin S_1; S_2 end. Thus, with a certain sense of layout, we combine (2) and (3) to obtain our PASCAL program (1).

Here, in short, is the contrast:

For the formal description of PASCAL, we have a syntax whose rules let us "grow" well-formed statements, and a formal semantics which lets us assign, by induction on the height of its syntactic tree, a well-defined input-output function to each well-formed statement.

The "production theory", by contrast, is less well-defined. We start with a semantic goal G expressing what input-output function a statement is to achieve. A planning process then produces increasingly detailed "program sketches" in some planning language by a process of step-wise refinement which may well involve repeated backup (Wirth [1971], Alagić and Arbib [1978, Ch. 1, pp. 116 and 133-134]). Eventually, a level of refinement is reached which allows the plan to be translated into a PASCAL program which meets the semantic goal. The two points to note here are: First, the syntax no longer appears as production rules, but only as translation rules; and, second, the planning process invokes a great deal of knowledge (e.g., about arithmetic), only a small amount of which may be well-formalized at present. Of course, a major goal of AI is to chart an increasing body of knowledge in terms of well-formalized "knowledge structures" such as scripts, schemas and frames which are themselves amenable to computational manipulation.

I suspect that relatively little of these "knowledge structures" will be fully formalized in the near future, and that the performance of individuals will depend on idiosyncracies which will be formalizable in principle but not, a priori, in practice. After the fact, we may reconstruct that a particularly novel approach to solving a problem depended on a chance remark overheard in a hotel bar in Copenhagen seven years before, but the chances of such an item being included beforehand in a workable model of an individual's "Long Term Memory" are negligible. However, this argument against complete representation does not diminish the importance of seeking an adequate formalism for the representation of knowledge, and of developing computational models of how knowledge is used in planning. Linguists increasingly appreciate the need for an articulated model of the dictionary or lexicon for linguistic theory; the work of AI linguists on knowledge structures may be viewed as the development of an articulated model of the encyclopaedia. In neither case is a complete set of entries the criterion for progress.

Planning as an Ongoing Process

We have suggested that syntax emerges as a symptom of a translation process, and that this translation process is not from a fully elaborated deep structure to a surface structure, but rather involves a planning process that takes us from a (perhaps incompletely specified) semantic goal via a process of refinement until we achieve a plan sufficiently articulated for translation into a well-formed expression of the given language. But this account is still misleading if it suggests that a whole sentence or paragraph is "laid out" in its entirety "internally" prior to the actual production of an utterance. Rather, I would argue that we have a production process in which the next fragment of the utterance depends on the actual utterance generated so far, as well as the semantic goal (which may be redefined as the process continues), and the current state of the plan (much of which will be but partially developed until the utterance nears completion), quite apart from feedback (if any) from the intended audience. In this section, I first recall some notions from AI studies of planning, then look at the action-perception cycle of visuomotor coordination, and finally link these to a theory of conversation.

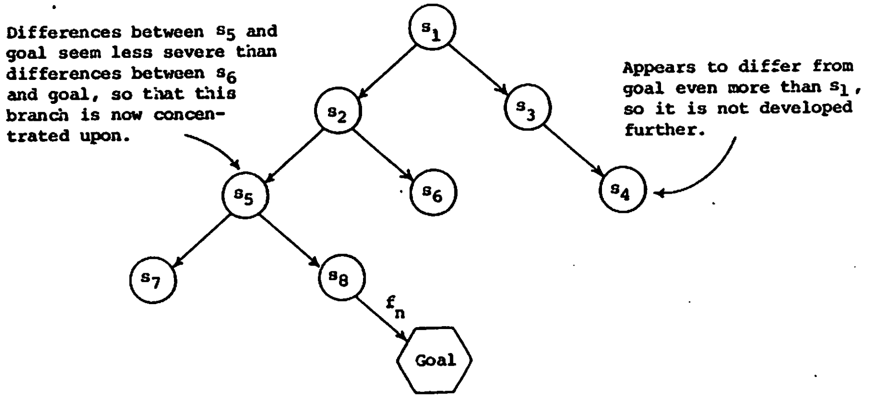
We start with an approach to planning called the General Problem Solver (GPS) due to Newell, Shaw and Simon [1959]. (For the implications of GPS for cognitive psychology, see Miller, Galanter and Pribram [1960] and Newell and Simon [1972]. For a brief exposition of GPS in relation to heuristic search and feedback, see Arbib [1972, Sec. 4.2].) GPS is a general framework for solving problems of the following kind:

(i) We are given a set of states, and a set of operators. Each operator is applicable to some (but not necessarily all) of the states; when applicable, the result of applying operator f to state s will be to transform it into a new state f(s).

(ii) We are given an initial state s_0 and a goal state s_g. Our task is to find a (reasonably short) sequence of operators $f_1,\ldots,f_n$ which will transform s_0 into s_g, i.e. f_1 is applicable to s_0 and $s_1 = f_1(s_0)$; f_2 is applicable to s_1 and $s_2 = f_2(s_1)$ ...; f_n is applicable to s_{n-1} and $s_g = f_n(s_{n-1})$.

(iii) To aid us in our quest for an appropriate sequence of operators, we are given a finite set of differences, and a means whereby, given any ordered pair of states, we can list which differences obtain between them. In addition, we are given an operator-difference table which gives us for each difference a list of those operators that are likely to reduce it.

Unfortunately, the differences give only a rough indication of what needs to be changed, and there is no guarantee that applying a recommended operator will indeed transform the latest state into one that is "closer" to the goal state. Moreover, a recommended operator may not be applicable to a given state -- leading us to generate the subgoal of transforming the given state into one to which the operator is applicable. The general control program of GPS is thus designed to develop a "decision tree" which keeps track of the application of various possible operators to various states, "growing" those branches which seem to be leading towards the goal, as in Figure 2.



Differences between s_5 and goal seem less severe than differences between s_6 and goal, so that this branch is now concentrated upon.

Appears to differ from goal even more than s_1, so it is not developed further.

Figure 2. An example of the sort of "decision tree" that might be grown by the general supervisory part of a GPS program.

To fix our thinking, the states might be the position of a robot in an environment, the initial state its position at the onset of planning, while its final state is the position to be achieved by the to-be-planned sequence of basic actions. GPS, with its finite set of differences (such as "to the left of" and "ahead of" in this case) is limited with respect to a system with access to more subtle descriptors of the differences between states (such as actual differences in coordinates modified by information on the presence of obstacles, in this case). This has led to planning techniques based on "heuristic distance" (e.g., using straight-line distance as a "heuristic" approximation to the actual distance to be traversed by the shortest path which traverses all obstacles). Two basic papers in this literature are due to Doran and Michie [1966] and Hart, Nilsson and Raphael [1968] (see Nilsson [1971] for a textbook account);

and these provided part of the basis for the studies of robot path-planning by Fikes and Nilsson [1971] and Fikes, Hart and Nilsson [1972]. But these studies provided step-by-step planning of the entire path from initial position to the goal, and the attempt to improve upon this with an AI approach to hierarchical planning led Sacerdoti [1977] to elaborate "A Structure for Plans and Behavior" (a title deliberately adapted from the "Plans and the Structure of Behavior" of Miller, Galanter and Pribram [1960]). Sacerdoti developed an AI program called NOAH (Nets Of Action Hierarchies) that solves a problem by first creating a one-step solution to the problem (essentially "Solve the given goal") and then progressively expanding the level of detail of the solution, filling in ever more detailed actions. All the individual actions, composed into plans at differing levels of detail, are stored in a data structure called the procedural network. Actions can be initiated before the entire plan is developed to the level of actions which are immediately implementable. If and when more detail is required, or if an unexpected event creates the need for replanning, the system can modify or further develop the plan at any point.

In outlining a theoretical framework for the analysis of skilled motor behavior -- such as speaking, typing, writing, drawing, dancing, or playing music -- Shaffer [1980] stresses that an extended skilled action starts with a plan that provides a set of goals and the essential structure of the performance. He cites Sacerdoti's [1974] proposal for planning in a hierarchy of abstraction spaces as analogous to his view that the plan is executed in "a continually renewing succession of higher-order units by a motor program, which may construct one or more intermediate representations leading to output, adding the details necessary to specify the movement sequence." Such programs "enable more flexible problem solving and they

produce solutions more rapidly than programs that consider all the possible domains of information in a single abstract space. Both these properties, speed and flexibility, are relevant to skilled performance." (Given the emphasis of Arbib [1979] on cooperative computation, it is worth noting Corkill's [1979] analysis of a cooperative computation approach to NOAH.)

From the Action-Perception Cycle to Conversation

Given the above perspective on planning as an ongoing process, we now turn to a brief description (following Arbib [1980]) of the relation between visual perception and the control of movement. We then turn to analogies between this process and the analysis of conversation.

We propose that the following internal structures and processes are necessitated by the visual control of locomotion: the representation of the environment, the updating of that representation on the basis of visual input, the use of that representation by programs which control the locomotion; and the cycle of integrated perception and action. We seek functional units whose cooperation in achieving visuomotor coordination can be analyzed and understood irrespective of whether they themselves are further decomposed in terms of neural nets or computer programs. Our style of analysis will seek to decompose functions into the interaction of a family of simultaneously active processes called schemas, which will serve as building blocks for both representations and programs.

The control of locomotion may be specified at varying levels of refinement: the goal of the motion; the path to be traversed in reaching the goal; the actual pattern of footfalls in the case of a legged animal; and the detailed pattern of motor or muscle activation required for each footfall.

It is well-known that the fine details of activation will be modified on the basis of sensory feedback, but we stress that even the path-plan will be continually modified as locomotion proceeds. For example, locomotion will afford new viewpoints which will reveal shortcuts or unexpected obstacles which must be taken into account in modifying the projected path. We thus speak of the action/perception cycle -- the system perceives as the basis of action; each action affords new data for perception.

In terms of "units independent of embodiment" we may seek to postulate basic motor processes for, e.g., locomoting which, given a path plan as input, will yield the first step along that path as output. Another such unit would direct a hand to grasp an object, given its position as input. We refer to such units of behavior as "motor schemas". Our analysis will descend no further than the level of motor schemas, and will leave aside details of mechanical or neuromuscular implementation. Our claim will be that crucial aspects of visuomotor coordination can be revealed at this level of aggregation.

The raw pattern of retinal stimulation cannot guide locomotion directly. Rather, it must be interpreted in terms of objects and other "domains of interaction" in the environment. We also use the term "schema" for the process whereby the system determines whether a given "domain of interaction" is present in the environment. The state of activation of the schema will then determine the credibility of the hypothesis that that which the schema represents is indeed present; while other schema parameters will represent further properties such as size, location, and motion relative to the locomoting system.

Consider a schema that represents, say, a chair; and consider an environment that has two chairs in plain view. It is clear that two copies

of the chair-schema -- or, at least, two separate sets of chair-schema-parameters -- will be required to represent the two chairs. We refer to these two copies as separate "instantiations" of the same schema, each with its own set of parameter values. We may thus view the internal representation of the environment as an assemblage of spatially-tagged, parametrized, schema instantiations.

Object-representing schemas will not be driven directly by retinal activity, but rather by the output of segmentation processes which provide an intermediate representation in terms of regions or segments (usually corresponding to the surfaces of objects) separated from one another by edges, and characterized internally by continuities in hue, texture, depth and velocity. As locomotion proceeds, and as objects move in the environment, most of these regions will change gradually, and the segmentation processes must be equipped with a dynamic memory which allows the intermediate representation to be continually updated to provide current input for the object-schemas, so that the schema-assemblage representing the environment will be kept up-to-date.

Note that a schema is both a process and a representation. The formation and updating of the internal representation is viewed as a distributed process, involving the parallel activity of all those schemas which receive appropriately patterned input. The resultant environmental representation interacts with those processes which represent the system's goal structures to generate the plan of action -- exemplified by the projected path in the case of locomotion -- which can provide the input to the various motor schemas that directly control behavior.

We may view the schema-assemblage -- the structure of perceptual schemas which relates the animal to its environment -- as a spatial structure which has temporal characteristics (e.g. representing the motion of objects relative to the animal). We shall shortly discuss the possible nature of "coordinated control programs" which can coordinate the activation of motor schemas. Such a program serves to control the temporal unfolding of movement, but has spatial characteristics since interaction with objects will usually depend on their position in the environment.

There is no simple stimulus-response relationship here. Perception of an object (activating perceptual schemas) involves gaining access to motor schemas for interaction with it, but does not necessarily involve their execution. While an animal may perceive many aspects of its environment, only a few of these can at any time become the primary locus of interaction. A process of planning is required to determine the plan of action, the appropriate program of motor schema activation, on the basis of current goals and the environmental model. Perception activates, while planning concentrates. Coming upon unexpected obstacles can alter the elaboration of higher-level structures -- the animal continually makes, executes and updates its plans as it moves.

The language of "coordinated control programs" addresses the description of the coordinated phasing in and out of the brain's manifold control systems. While certain basic programs are "hard-wired", most programs are generated as the result of an explicit planning process. We exemplify this notion by the hypothetical program of Figure 3 for a human's grasping an object.

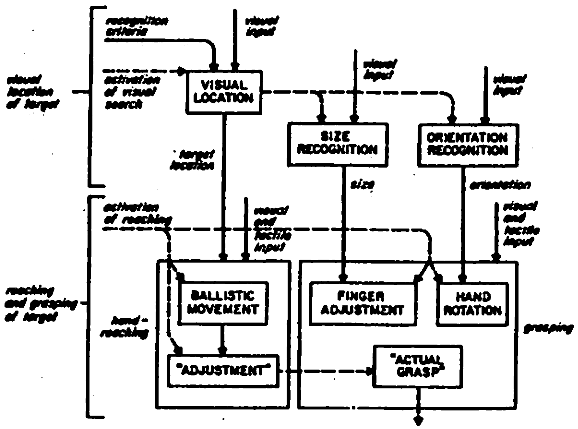


Figure 3. A hypothetical coordinated control program for visually directed grasping. The perceptual schemas atop the figure serve as identification procedures for the motor schemas in the control program of the lower half of the figure.
(---→ control path; ——→ data path)

The spoken instructions given to the subject drive the planning process that leads to the creation of the appropriate plan of action -- which we here hypothesize to take the form of the distributed control program shown in the lower half of the figure, involving the interwoven activation of motor schemas for reaching and grasping. Activation of the program (broken arrows convey "activation signals") is posited to simultaneously initiate a ballistic movement toward the target and a preshaping of the hand during which the fingers are adjusted to the size of the object and the hand is rotated to the appropriate orientation (solid arrows indicate transfer of data). When the hand is near the object, feedback adjusts the position of the hand, and completion of this adjustment activates the actual grasping of the hand about the object.

The perceptual schemas hypothesized in the upper half of the figure need not be regarded as a separate part of the program. Rather, they provide the identification algorithms required to pass parameter values to the motor schemas. This analysis of visual input locates the target object within the subject's "reaching space"; and extracts the size and orientation of the target object and feeds them to the control surface of the grasping schema. When the actual grasping movement is triggered, it shapes the hand on the basis of a subtle spatial pattern of tactile feedback. (For data on visuomotor mechanisms in reaching within extrapersonal space, and a careful review of the relevant literature, see Jeannerod and Biguer [1980].)

With this background on the action-perception cycle, we can now see that there are important parallels between visual perception and speech understanding on the one hand, and between speech production and motor control on the other. The basic notion is that speech perception, like vision, requires the segmentation of the input, the recognition that certain segments may be aggregated as portion of a single structure of known type, and the understanding of the whole in terms of the relationship between these parts. We have suggested that the animal's internal model of its visually-defined environment is an appropriate assemblage of schemas, and would offer the same for the human's internal model of the state of a discourse. Since the generation of movement requires the development of a plan on the basis of the internal model of goals and environment to yield a temporally-ordered, feedback-modulated, pattern of overlapping activation of a variety of effectors, we would argue that the word-by-word generation of speech may be seen as a natural specialization of the general problem of motor control.

If an utterance is a command, the listener must recognize it as such and translate it into a "program" for carrying out the command -- which may well involve first translating the command into an internal representation, and second calling upon planning processes to translate this into a detailed program of action tailored to the current situation. If the input is a question, the system must recognize it as such and translate it into a plan for recalling relevant information. A second translation is then required to express this information as a spoken answer. We thus suggest that the task of speech perception is to organize a string of words into pieces which map naturally into internal processes that update the listener's "internal model" -- whether or not there is an overt response to the utterance. Production serves to express some fragment of a "brain representation" as a syntactically correct string of words. We see again our stress on translation between "internal" and linguistic representations of meaning.

We may then view the action/perception cycle as corresponding to the role of one speaker in ongoing discourse. One can view the deployment and decoding of the linguistic signal as responsive to a series of constraints. The first are those inherent in the structure of the linguistic code itself, and their characterization is the goal of the theory of linguistic competence. We presently have far more information about these constraints than about the remaining levels. The ~~second~~ type of constraint arises from psychological limitations of the human language-processing systems. Recent work (Frazier and Fodor [1978]) has advanced hypotheses regarding the intrinsic nature of these psycholinguistic devices, and suggested interactions between the nature of human processing routines and the nature of language structures. A third type of constraint results from the social and pragmatic facts of conversational situations. Other levels can be suggested. We can view the utilization of language, at each of these levels, as consisting of the interaction of a stored long-term representation of the items and processes at some level and the analysis of the incoming and outgoing signal at the same level to yield a fluid and continually updated current model of the total language act. Seen this way, there are overall similarities between sensory-motor and language computations which should allow us to investigate aspects of the neural mechanisms of language by examining the neural mechanisms relevant to perceptual-motor activity.

The Neural Analysis of Sensorimotor Processes

Neuroscience has taught us how to trace the coding of information in the visual periphery (Hubel and Wiesel [1977], Lettvin, Maturana, McCulloch and Pitts [1959]); how to view the cerebral cortex as composed of columns or modules intermediate in complexity between single cells and entire brain regions (Hubel and Wiesel [1974], Mountcastle [1978], Szentágothai and Arbib [1974]); how to analyze spinal circuitry involved in motor outflow, and the later stages of its cerebral and cerebellar control (Phillips and Porter [1977], Granit [1970], and Eccles, Ito and Szentágothai [1967]). To some extent these analyses may be integrated into models of perceptual and motor processes (Figure 4).

We have good neuroscientific data on retinal response to neuronal stimulation, and of various "feature extractors" in a number of different animals. At the motor periphery, we have good neuroscientific data on basic motor patterns, their tuning by supraspinal mechanisms (the Russian school has been particularly productive here), and the spinal cord rhythm generators and feedback circuitry which control the musculature. This partial list could be extended and could be complemented by a list of major open problems at these levels: the important point here is that near the visual and motor peripheries there is a satisfying correspondence between single-cell analysis and our processing concepts.

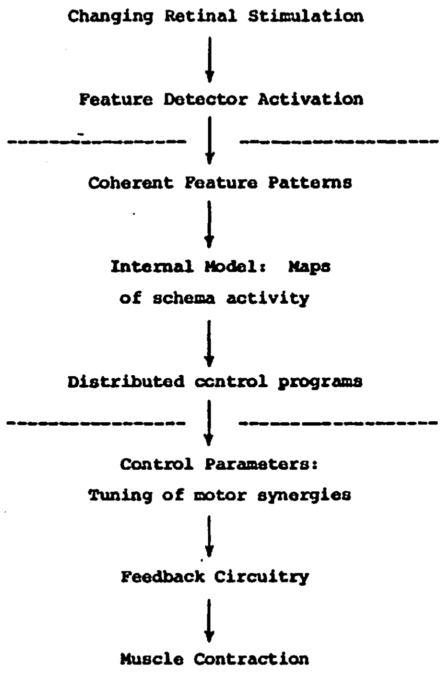


Figure 4. Stages in visual perception and control of movement. Many aspects are omitted, as are all of the important "return" pathways.

In between, the results are fewer and tend to be somewhat more speculative. By what process are the often disparate activities of feature detectors wedded into a coherent "low-level" representation of the world? How is the representation integrated into the ongoing internal model (the "schema-assemblage" as we have posited it to be)? How are the internal model and goals of the organism combined in a planning process which yields the distributed control programs which orchestrate the motor synergies? There are models for these processes (reviewed in Arbib [1980]), but many are couched in a language closer to AI than to neurophysiology, and the body of available neuroscientific data with which they can make contact is still relatively small. Nonetheless, it does seem to us that progress is well under way in the neural analysis of "perceptual structures and distributed motor control". We briefly sketch one effort of this kind.

The problem of visuomotor coordination in frog and toad has yielded to a behavioral analysis coupled with lesion and single-cell analysis (Ewert [1976], Ingle [1976]). Ingle [1968] had observed that a frog confronted with two fly-like stimuli would normally snap at one, but would sometimes snap at neither even though each stimulus alone was "snap-worthy". This suggests a process of competition between the internal representation of the "flies" (an identification of each "schema" with localized cellular activity does seem justified here) and Didday [1970, 1976] offered a model of competitive interaction in neural nets consistent with the data of 1970. The intervening ten years have seen developments in both theory and experiment. Amari and Arbib [1977] developed a general theory of competition and cooperation in neural nets, and this has proved to have much similarity with AI's relaxation and constraint satisfaction techniques for the resolution of conflicting hypotheses (Rosenfeld, Hummel and Zucker [1976], Shortliffe [1976], Waltz [1978]). Experiments have shown patterns of interaction between tectum and prethalamus, and demonstrated the interaction between prey-approach and predator-avoidance. In the next section, we develop an analogy with neurolinguistic studies of object naming.

Object Naming in Humans and Prey-Predator Discrimination in Amphibia

The title of this section would seem to betray a certain lack of seriousness on the part of the author. Can there be any useful sense in which object naming in humans can be compared to prey-predator discrimination in amphibia? I do not wish to claim too strong a relationship, but I shall try to show that certain processes posited by Luria in his analysis of clinical findings on object naming are similar to processes in amphibia which are the subject of current neural modelling. I thus hope to indicate ways in which modelling of neural circuitry (and not just of high-level interaction of regions) may come to enter neurolinguistics.

We start by discussing a diagram (Figure 5 of Arbib and Caplan [1979]) based on Luria's [1973] analysis of object naming. Each box corresponds to a brain region and to functions suggested by clinical data. In the object naming task, no acoustic model is given the subject. Instead, he is to look at an object, and code his visual perception of the object by an appropriate spoken word. Clearly, performance of object naming requires reasonably precise visual perception. Luria singles out as the anatomical site of this component the left temporo-occipital zone (Box A of Figure 5) where lesions disturb both the ability to name objects and the ability to evoke visual images in response to a given word. A patient with such a lesion cannot draw a named object, even though he can copy a drawing line-by-line. In short, lesions here seem to impair the transformation between an array of isolated visual features, and a perceptual unity into which the features are integrated.

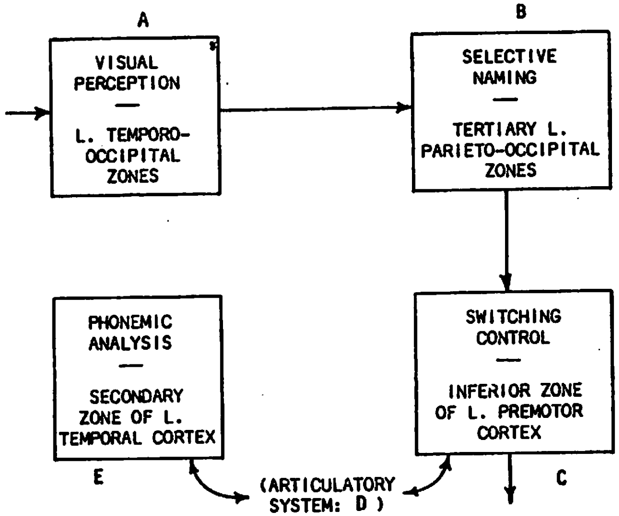


Fig. 5. Block diagram of subsystems involved in Luria's analysis of naming of objects.
(Fig. 5 from Arbib and Caplan [1979])

The next step (Box B) is to discover the appropriate name, and inhibit irrelevant alternatives. Lesions of the left tertiary parieto-occipital zones yield verbal paraphasias -- the appearance of an irrelevant word, resembling the required word either in morphology, meaning, or phonetic composition. Irrelevant sensory features of the object or of articulatory or phonetic information associated with its name can evoke a response as easily as correct features. It is as if the inhibitory constraints were removed in a competitive process. Such lesions do not disturb the phonological representation of language -- prompting with the first sound of a name does trigger its recall.

Luria notes that lesions of the inferior zone of left premotor cortex (Box C) impair shifting from the name of one object to that of another, and that lesions in the left fronto-temporal region (not represented) affect the patient's critical attitude to the developing pathological inertia and disturb his ability to correct his mistakes. It is clear that the articulatory system (Box D) must also be active in the naming of objects.

What may be unexpected is that Luria also implicates Box E -- phonemic analysis -- in the naming of objects. Lesions of the left temporal region disturb the phonemic organization of naming, yielding literal paraphasias, in which words of similar phonemic organization are substituted. In strong contrast with the verbal paraphasias induced by Box B lesions, prompting with the initial sound of the name does not help the patient with a left temporal lesion.

This provides important evidence for Luria's view of the brain as a functional system and our own stress on cooperative computation. It is now clear that Box E is not just for sensory phonemic analysis; nor is Box D purely for motor articulatory analysis. Rather, both systems participate in all brain functions which require exploitation of the network of represenations that define a word within the brain. Convergence on the proper word can be accelerated by the cooperative exploitation of both phonemic and articulatory features, and others as well.

We now turn to a brief description of prey-predator discrimination in frog and toad. Where the patient must integrate the visual input to respond with a name, the animal must respond to visual input from a number of moving objects by snapping at one, avoiding an apparent predator, or by remaining motionless. Ingle ([1976], for a review) studied frogs confronted with one or more fly-like stimuli. When confronted with two "flies", either of which was vigorous enough to elicit a snapping response when presented alone, the frog could snap at one of them, not snap at all, or snap at "the average fly". Didday [1970, 1976] designed a plausible network (consistent with data on frog tectum available in 1970) which can take a position-tagged array of "foodness" intensity and ensure that only one region of activity, usually, will persist to influence the motor control systems. In the model, the cells of the "foodness" layer feed a "relative foodness" layer whose output is to affect motor control systems. Didday also posits a population of what we shall call S-cells in topographic correspondence with the other layers. Each S-cell inhibits the activity that cells in its region of the "relative foodness layer" receive from the corresponding cells in the "foodness" layer by an amount that increases with increasing activity outside its region. This ensures that high activity in a region of the foodness layer only "gets through" if the surrounding areas do not contain sufficiently high activity to block it.

One trouble with the circuitry described so far is hysteresis: the build-up of inhibition by the S-cells precludes the system's quick response to new stimuli. Didday thus introduced what we shall call an N-cell for each S-cell. The job of an N-cell is to monitor temporal changes in the activity in its region. Should it detect a sufficiently dramatic increase in the region's activity, it then overrides the S-cell inhibition to enter the new level of activity into the relative foodness layer. With this scheme, the inertia of the old model is overcome, and the system can respond rapidly to significant new stimuli.

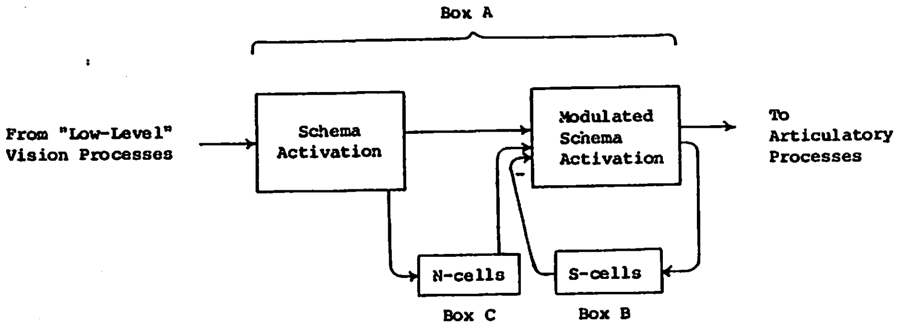


Figure 6. A re-analysis of Boxes A, B and C of Luria's analysis of naming.

With this, we may look at Figure 5 anew. We now suggest that Box A, the "visual perception box", does not produce a single percept, but rather (recall the frog's "foodness layer") produces a pattern of activity corresponding to a variety of schemas more or less consistent with the current visual input. Box B, whose designation as "selective naming" now seems less appropriate, would seem analogous to the layer of S-cells since with its removal, "irrelevant sensory features of the object or of articulatory or phonetic information associated with its name can evoke a response as easily as correct features." But if we accept this analogy, Box B does not act in the sequential fashion of Figure 5, but rather by an "inhibitory loop" as suggested by Figure 6. Since lesions of Box C "impair shifting from the name of one object to another", this suggests an analogy to the layer of N-cells, and a consequent "activating loop" as shown in Figure 6.

These analogies are at best tentative, but they do indicate how a far more comprehensive attempt to relate language mechanisms to better developed analysis of circuitry for visuomotor coordination may yield fresh hypotheses for tracing out the detailed anatomical and physiological relationships underlying language.

Conclusion

While differences between human and non-human brains will doubtless play a role in distinguishing the particular repertoire of each species (cf. Geschwind [1965]), it nonetheless seems important to provide more empirical support for particular neural models in animals, and to relate the models developed in the animal to comparable perceptual-motor systems in man, and thence to systems involved in language. It is encouraging to note that many neurologists have been concerned with this relationship of aspects of language to perceptual, motor, and other cognitive systems. Examples include Jackson's [1878-9] view of propositions, Geschwind's [1975] approach to the agnosias and apraxias, and Luria's [1973] concern with start/stop mechanisms shared between linguistic and non-linguistic motor activities. We also note the influence on Luria of Bernstein [1967], whose work laid the basis for the Moscow school which combines neurophysiological and mathematical analyses of motor control with the construction of actual robots (Fel'dman and Orlovsky [1972], Gelfand and Tsetlin [1962], Okhotimskii et al. [1979]).

Pursuit of these connections will, I have argued (Arbib [1975, 1979], Arbib and Caplan [1979]), require a framework of cooperative computation in

which, in addition to interaction between components of a language processor alone, there are important interactions between components of linguistic and non-linguistic systems. Careful analysis of the components of perceptual, motor, and linguistic tasks and of their patterns of breakdown will, it is predicted, lead to the identification of a (presumably limited) number of components which share and/or compare linguistic and non-linguistic representations. One should then be able to infer something of the neural mechanisms underlying language from an appreciation of those underlying related non-linguistic processes. As stressed by Arbib and Caplan [1979], we are confronted with the need for explicit representational systems, in this case for non-linguistic as well as linguistic entities, coupled with a cooperative computational analysis of processing which determines which representations are "translatable" and "shared" between linguistic and non-linguistic systems.

Jackson [1878-9] argued that observations of the "propositionalizing" of patients with "affections of speech from disease of the brain" would lead to a theory of language function which was not task-specific, and to a theory of brain function which did not consist of centers and connections. However, he offered no theory of the representation of propositions, nor did he distinguish between the ideational and linguistic form of propositions. In a highly similar vein, Goldstein argued that general functional principles, such as the ability to assume what he terms an "abstract attitude" (an attitude, we remark, which is probably a prerequisite for the production of a Jacksonian proposition), were lost in aphasia. A modern development of this approach is found in Locke et al. [1973] in which Jacksonian functional capacities are related to a hierarchical model of the neuraxis based on Yakovlev's work [1948].

The Jacksonian approach incorporates the claim that the functional capacities lost with respect to language functioning are also lost in other realms of behavior in the aphasic patient (cf. Pick [1913], Brown [1972, 1977]). It emphasizes the overlap of linguistic and non-linguistic functions, but uses only a rudimentary characterization of language itself. While this approach avoids the central issue in neurolingistic theory, which consists first of the study of the representation and utilization of the linguistic code in and by neural tissue, it does help us understand how this code is subject to functional factors which also regulate other cognitive and perceptual-motor capacities. A confrontation of Jacksonian ideas with the notions presented in this paper should thus help us develop fruitful comparisons of linguistic behavior with other perceptual-motor behaviors to the point where animal models can be introduced for relevant aspects of neurolinguistic processing.

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JASON BROWN: COMMENTS ON ARBIB PAPER

Arbib looks at language in the context of perceptuo-motor mechanisms. I agree with this, not only because it seems to be the correct approach, but also because it permits us to build a common language for psychology, linguistics and neuroscience. My problem is with the conceptual model on which the approach is based. Arbib has given us a componential account set in the framework of an evolutionary theory. Frankly, I don't see how you can have it both ways. The idea of interaction in a distributed system does not rest well with the idea of constraints imposed upon that system by its direction of evolutionary growth. An interactionist model is indifferent to the evolutionary concept that processing reflects the pattern of growth trends in phylo-ontogeny.

Moreover, the model implies that damage to an area of the brain, or a box in Arbib's diagram, eliminates whatever process, strategy or representation the area supports. Take for example a case of jargonaphasia such as discussed vis-a-vis Lecours' paper at this conference. With a lesion of the left Wernicke area, what does the jargon point to? What is lost in an utterance which is semantically or phonologically deviant? Certainly not the semantics or the phonology. What is the meaning of "loss" when intact performances occur alongside those which are deficient? In what sense is a function lost when it is regained a moment later? For the clinician who deals with symptoms and symptom change, the effects of pathology are more subtle than computational models suppose.

My own view is that a symptom is really a *normal* event which anticipates something, it is a state which is ordinarily traversed in a processing sequence. The effect of the lesion is to display that preliminary state, in language, in perception or in motility. I might add that this concept of the symptom gives meaning to clinical work.. It is the basis of a real clinical methodology. The idea that *symptoms reflect structure directly* allows the clinician to go beyond the usual descriptive approach, classifying behaviors and isolating syndromes without any theoretical underpinnings.

Regarding Arbib's comments on syntax, I'm not sure that the neurology of language has contributed much to an understanding of this problem. The distinction of open and closed class words (papers by Garrett and Kean) does not in my view go to the heart of the aphasic deficit. The idea that agrammatics are the way they are because of a disturbance in a mental grammar is simply another way of describing their problem rather than explaining it. Moreover, it is probably not accurate. There are several studies which have been done in our lab which go to this issue. In one study, Lucia Kellar tested aphasics with the triadic method and found that anterior and posterior cases had comparable deficits in their grammatical judgments. This indicates that deficient grammatical knowledge, as assayed by these tests, is not necessarily the basis of the agrammatism. In another study, Claudia Leslie found (after Zurif and Carramazza) that aphasics have difficulty with reversible sentences. However, when both interpretations are equally improbable (e.g. the fish chased the cow), performance improved dramatically. This indicates that anteriors can decode the syntax of the sentence when they are not required to also attend to the plausibility of the events described. In still another study, Phyllis Ross gave aphasics a letter cancellation task with "silent" and "pronounced" letters. She found that both anterior and posterior aphasics, like normals, make more errors on functors than content words, and that the content-function word difference was the same in the two groups. This suggests that anteriors do not treat functors like content words.

This is not to say that anteriors are not deficient in syntax; rather, that this is not the explanation of their agrammatism. Moreover, there are good clinical arguments against the syntax account. For example, the fact that agrammatics tend to read aloud and repeat agrammatically suggests that the problem is bound up with motor planning and production rather than syntactic judgments. Conversely, the relative preservation of the grammatical words in the posterior aphasias reflects the fact that errors tend to be determined by meaning relations. The reduced meaning content of the functors allows them to "escape" derailment in the posterior system. Certainly, the idea that agrammatism is tied to (a disturbance in) motility fits in with Arbib's view that, in production, syntax is not explicitly used as "inverse parsing" but reflects the constraints of planning.

How can one conceptualize the motor framework underlying agrammatic production? As Arbib notes, Bernstein (1967) studied rhythmic movements like walking or hammering, and argued for a motor plan as a pre-existing structure of dynamic waves preceding the movement by a second or so. Can one think of anteriors in these terms?

In previous works (e.g. Brown, 1979) I have proposed that a set of microgenetic levels forms a substructure under the utterance. The various types of anterior aphasias correspond to levels, or moments, in the unfolding of the utterance over evolutionary and developmental levels in brain organization. The clinical series proceeds from a type of mutism and akinesia (lesions of limbic-derived neocortex, or mesocortex) through agrammatism (probably associated with lesions of generalized or homotypical isocortex), to the misarticulations of the true Broca aphasic (lesion of a focal region of neocortex which becomes specified in the course of maturation).

The most primitive level in this sequence is that of the "motor envelope", linking vocalization and body motility in the same matrix of rhythmic movement. Here the act is organized about the axial and proximal musculature. There is a relationship to respiratory rhythms. We know that at its inception an utterance is organized about respiratory patterns, e.g. the "breath group" (Lieberman, 1967; Rubin, 1975), and there are impairments in respiratory timing in motor aphasia (Schoenle, 1979). This primitive level probably develops out of rhythmic systems organized at the level of the upper brainstem. These systems are presumably linked to other vegetative or autonomic cycles, perhaps even to diurnal rhythms.

The subsequent level would represent a derivation of the base frequency at the next microgenetic stage. Conceivably, this derivation (?harmonic) is to an oscillator which controls the speech rhythm or intonational pattern. A disruption here gives agrammatism. The final derivation would be to the fine articulatory rhythm by way of focal neocortex. This stage would support the terminal programmation of sound sequences, disrupted in Broca's aphasia. This concept of a series of rhythmic levels in motility, unfolding over evolutionary stages in brain organization, and linked to aphasic syndromes arising with lesions of the respective anatomical strata, is illustrated in Figure 1.

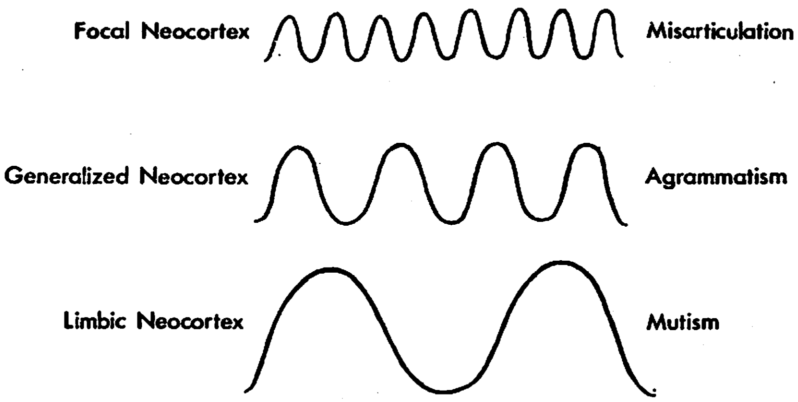

Figure 1

I should point out that the concept of a phylogenetic progression from a lower level automatic rhythmic organization to higher level mechanisms directed to the environment has been discussed by several authors (Jung, 1941; Walter, 1959). There is some evidence that the lower organization is linked to mechanisms in the rostral brainstem which synergistically relate motor, respiratory and other autonomic rhythms in a common system. Thus, Barcroft and Barron (1937) in a study of the sheep embryo, found rhythmic trunk movements timed with respiratory rhythms at an early stage of <u>in utero</u> development. Gradually, these rhythms became independent, but the original relationship could be brought out by anoxic brain damage. Lesion studies indicated that the rhythms were organized at the level of red nucleus; i.e. midbrain. Schepelmann (1979) has reviewed evidence that respiratory rhythms exert an effect on motor activity. He described human cases of brain pathology with synchronous motor and respiratory rhythms, and proposed a primary brainstem rhythm of about 3/minute regulating respiration and motility, and perhaps autonomic functions as well. There is additional pathological evidence for this idea; for example, in the rhythmic palatal and orofacial tremors which occur with brainstem lesions in man (Brown, 1967). Studies of rhythmic myoclonus in cats injected with Newcastle disease virus have demonstrated spinal and brainstem systems underlying rhythmic motor and respiratory contractions.

In addition, there have also been speculations on the relation of speech production to rhythmic activity (Lashley, 1951; Martin, 1972). Boomer and Laver (1973) have pointed to the importance of rhythmic factors, tonality and stress at the earliest stage in speech production. Others have shown similarities between language and motor systems, suggesting common organizational principles in terms of oscillatory systems (Kelso et al., 1980; Turvey, 1977).

I want to turn now briefly to Arbib's discussion of naming. In my view naming is a key to an understanding of the posterior aphasias. Arbib has given an apt description of naming as capturing a target in an object field. There are more than incidental similarities between the visual grasp of an object and the process of word selection. Consider the work on monkeys with inferotemporal lesions. The visual deficit is neither purely mnestic nor one of visual discrimination. Teuber characterized it as a defect in the selection of an object from an array. Now it is interesting that inferotemporal lesions in man do not really give rise to comparable deficits in visual recognition. Visual agnosias in which object meaning plays a part appear to be associated with medial temporo-occipital lesions. Of course, some fragments of the deficit may appear with right temporal lesions, as in the McGill studies, and mild impairments in visual recognition occur with left temporal lesions (e.g. DeRenzi, 1971). However, the most striking deficit, in the left temporal cases, is an aphasia. Moreover, the aphasia is characterized by lexical-semantic errors, and is aggravated, and qualitatively changed as well, when the lesion is bilateral. The possibility that a neural substrate which in monkey supports visual recognition is, in man, involved in lexical selection, underscores Arbib's notions about the relationships between visual search and naming, and it also emphasizes the need in aphasia study to go beyond the descriptive material to the underlying perceptual and motor events.

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In Discussion:

Perecman, E. and J.W. Brown. Phonemic jargon: a case study. In: J.W. Brown (Ed.), Jargonaphasia, Academic Press, New York, 1980 (in preparation).

Edgar Zurif: Comments on Brown's Comments

First off, although it might not have been Brown's intention to do so, he has invited the inference that language is epiphenomenal -- that its configurational properties and its reliance on the integrity of the left hemisphere are straightforwardly the consequence of patterns of constraints on the operation of motor and perceptual systems. But whatever his intention, this inference is untenable. Even though language comprehension and production are obviously dependent upon the operation of sensory-motor systems, these latter systems and the manner of their breakdown under conditions of brain damage do not predict the ways in which focal brain damage also selectively affects language capacity and the processes implementing this capacity (Goodglass, 1968; Bradley, Garrett, and Zurif, in press; Zurif and Caramazza, 1976; Zurif and Blumstein, 1978).

Further, and Brown's claim notwithstanding, attempting to account for language breakdown via a componential analysis in terms of distinctions among linguistic information types does not require that symptom localization be equated with functional localization -- no more so than for any other form of functional analysis. Yet, it may reasonably be supposed that the selective manner in which language breaks down bears some relation to the manner in which it is organized in the brain. And whatever the effect of a lesion on physiological function, however broadly based the alteration is to cerebral activity, and however much there are internal physiological readjustments within a complex hierarchy, the fact reamins that at some level of neural organization the effect of focal damage is manifestly selective in a manner that seems to be accomodated by some psycholinguistic distinctions.

Brown tries to minimize this fact by claiming that one kind of syndrome flows into another, that linguistically isolable impairments do ont have generality across languages, and that agrammatism is not tied to a problem in syntax.

The first of these two claims are, I think, wrong. I have never seen a fluent aphasic evolve into a non-fluent aphasic, nor have I ever seen "flow" in the reverse direction. Further, agrammatism is not restricted to English-speaking aphasics, and it is not rare in French-speaking aphasics.

Brown's third claim is misleading. Of course, as a general and not too surprising point, all aphasic patients fail to comprehend complex sentences more than they do simple sentences; but what is important is that they are likely to do so for different reasons (Caramazza and Zurif, 1976; Goodglass and Kaplan, 1972). In this context, Kellar's (1978) finding -- that Wernicke's aphasics are as grammatically impaired as Broca's aphasics on a task involving within-sentence word-relatedness judgments -- more likely attests to the influence of unspecified task variables (and thereby to the inadequacy of the task as a pure measure of a constituent linguistic capacity) than to the neurological inseparability of processing form and meaning. Further, it is not clear that Wernicke's aphasics offer the appropriate contrast to Broca's aphasics: recent evidence suggests that the grammatical facility shown in the spontaneous speech of Wernicke's patients is illusory -- that (in ways very different from Broca's and probably for very different reasons) they are restricted to a limited number of

automatized routines (Gleason, Goodglass, Green, Hyde, and Weintraub, in press).

Finally, this last remark is not intended as an indictment of the fluent-nonfluent distinction as a means of bracketting constituent linguistic processes. Rather, it is intended to suggest that a more reasonable analysis of the uniqueness of left-anterior agrammatism is to be sought by comparing Broca's with fluent anomic aphasics; these latter patients present with a word finding disorder, yet produce well-formed structures without the presence of semantic paraphasias and neologistic elements. In fact, anomic as well as Broca's aphasics have already been assessed on the lexical decision task referred to by Brown and mentioned in more detail by Garrett and by Kean at this conference. And unlike Broca's aphasics, anomics have shown the normal dissociation for open and closed class vocabulary items, suggesting that the failure to separately access closed class items (with the attendant, unfortumate consequence for parsing) is tied specifically to left-anterior agrammatism.

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DISCUSSION OF ARBIB AND BROWN PAPERS

[Editor's Note: The Schnitzer paper contains a discussion of levels which bears comparison with that in Brown's commentary. Since Schnitzer emphasizes the use of inter-lingual comparison in understanding aphasia, the discussion of this topic following the Brown commentary has been placed with the discussion of the Schnitzer paper.]

Galaburda: In the future, we should be able to identify tiny lesions. Meanwhile, the 19th century does have many concepts for us, especially for sensorimotor/language parallels. Note that lesions relevant to aphasia have both sensory and motor consequences.

Zurif: Language is not simply epiphenomenal to the need to order events or move articulators in time. Language has its own characteristics. Arbib's componential account is not inconsistent with Brown's account and does not require symptoms to be associated with the function of each box. Brown is mistaken if he suggests that syntax is the first thing to go -- it's just that complex sentences are harder to understand than simple sentences. But much more than syntax is involved. Broca's and Wernicke's aphasias differ with respect to parsing. Broca's can use lexical semantics, parsing with the aid of major lexical items. Zurif, Caramazza and Blumstein show syntactic limitations in Broca's aphasia that cannot be seen in Wernicke's.

Levine: Language acts *are* sensorimotor, and one needs little convincing toview them in this way. The notion that "language", as a set of human activities, is something more than a group of sensorimotor skills, probably derives in part from Broca's analysis of motor aphasia as a loss of "motor plans" for speech without loss of movement per se. But the fact that an aphasic can move his tongue, but not speak may result entirely from the fact that the right hemisphere is good at the skills of swallowing and chewing though not good at the skill of speaking.

I doubt that the brain decomposes either visuo-motor behavior or language activities in a manner that corresponds to our introspectivrly derived logical decompositions. I think that terms such as "internal representation", "plan" and "motor execution" have little to dowith thebrain, especially when suchterms are considered as "components" of a single sensorimotor act. In this sense, such "information processing" componentialmodels are no different from19th century associationistic psychology at its worst. There is little likelihood that the brain does it that way.

Arbib: It's important to realize that I do not imply that planning is a conscious process in normal speech or movement. Rather, I want to stress -- as indicated in my analysis of sentence production as a planning process -- that there are necessary processes linking action and perception which tend to be overlooked. It would be crude to label a single region of cortex as "the planner", but the distribution of the planning

function across the brain still seems to me a necessary task of neurolinguistics. The same considerations apply to our modelling of visuomotor coordination in frog and toad. Having modelled how tectal-pretectal interactions enable the frog to snap at one fly among several, we now ask how the brain enables the frog to "plan" a modified trajectory when a barrier or a chasm blocks the direct path to the fly. While this involves modelling of additional brain regions, much of the work involves "evolving" more sophisticated models of regions already studied by incorporating finer details of the circuitry to extend the functional repertoire.

Whitaker: I don't accept vision as an analog for language. Heschl's gyrus does not project to thalamus as much as visual cortex projects to lateral geniculate nucleus. Again, contrast the language level in the congenitally deaf and the congenitally blind. Language is much harder for the former, showing the difference between acoustic and visual information play a different role.

Finally, I think the use of animal models is suspect below the primate level since their evolution is quite distinct from other forms. Moreover, singe-cell analysis is irrelevant to language.

Arbib: Of course, vision and audition are different. That doesn't mean we can't learn from a systematic exploration of their commonalities as perceptual systems. And the argument against single-cell studies in non-primates has held no water in vision studies -- microelectrode work on cats has been a primary source of data on neural mechanisms in vision.

Wood: Arbib's suggestions about the role of "top-down" (e.g., cortico-geniculate) connections in visual processing are an interesting extension of traditional ideas about the role of lateral inhibition at more peripheral levels of the afferent pathway. Lateral inhibition is generally regarded as emphasizing regions of change in stimulation and de-emphasizing regions where stimulation is redundant. Arbib's top-down processes represent the same principles of emphasis and de-emphasis based on information not available at the periphery.