Linguistic Disorders and Pathologies

An International Handbook

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21. Aphasia and Models of Language Production and Perception

1. Introduction

Models, in the sense of (comparatively) explicit accounts of some set of phenomena, often embodied in a diagram, or more recently in a computer programme, have been enormously influential in the development of science. The history of psychology is littered with the corpses of models, many of which have led brief, but useful, lives – interpreting old data, or motivating the discovery of new phenomena – before being fatally wounded in a fierce exchange of rhetoric.

In the history of aphasiological studies one model – the Wernicke-Lichtheim diagram (Lichtheim 1885) – has shown remarkable resilience, despite suffering accurate sniper fire (Freud 1891) and heavy pounding (Head 1926), since its publication. In a variety of guises, it continues to loom in the background of our thinking on aphasia and still forms an important basis for our principal diagnostic categories (e.g. Goodglass/Kaplan 1972).

In this chapter I will compare Lichtheim's model with a modern variant, and discuss some of the advantages of working with models of this type, and some of the disadvantages.

2. Lichtheim's Model

Lichtheim's model was a development of Wernicke's (1874) analysis of the anatomy and functional architecture of language processing. It was revolutionary for its time in several quite different ways. It proposed an explicit information-processing account, with distinct 'centres', or what we would call nowadays, 'components' or 'isolable subsystems' (Shallice 1984), each of which was a specialised repository for a particular type of information and carried out a different information transduction.

It was moreover formulated as a directed graph in which the direction of the flow of information was not only defined but also critical to the predictions made by the model. In addition to all this, it was an attempt to synthesise information processing with anatomy in that particular bits of cortex supported the information processing components, and particular transcortical or subcortical tracts were held to be the neutral sub-

strate of the connexions between the components.

Even by modern standards, it is a sophisticated and detailed account of a wide range of known aphasic phenomena. But it was designed to go further than a redescription of available data: It defined syndromes in terms of damage to components of the model, exactly in the way that neuropsychologists nowadays are meant to, rather than simply as co-occurring symptoms. As Lichtheim (1885, 435) himself wrote,

> "The morbid types I intend to discuss ... have been determined, in as far as they are new, deductively: it was the task of clinical observations to test the validity of the inferences."

2.1. Dissociations

The model stressed the importance of dissociations rather than associations. This was a major methodological breakthrough, though not one appreciated by all workers in the area (e. g. Head 1926). Further, Lichtheim used single patient data to test and to modify the model when the data failed to conform to predictions derived from the model.

2.2. Inferences from the Standard Wernicke-Lichtheim Model

The usual form of the Lichtheim diagram is given in Figure 21.1. (corresponding to his Figure 1). The numbered lines represent points of potential damage – "interruptions" in Lichtheim's terminology – whose consequences are interpretable by reference to the model. (It is important to remember that information flow models don't do anything! They simply describe the information exchange arrangements. This is as true for modern 'box and arrow' models, as those of the past century, as will be seen below. To derive predictions about data, or to account for data in terms of this type of model, requires a rich metatheoretical interpretative apparatus, much of which will be tacit.)

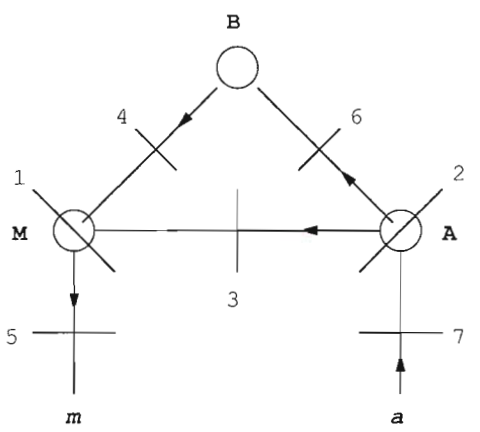

Fig. 21.1: Lichtheim's original diagram. See text for explanation (Redrawn from Lichtheim 1885, Figure 1)

Lichtheim's model distinguished in the modern manner between an input store of lexical information (auditory word images) from an output store (motor word images) (but see Allport/Funnell 1981; Butterworth 1983). A distinct expressive syndrome could thus arise from selective damage (Line 1) to the output store located in Broca's area, while a receptive syndrome could arise from selective damage (line 2) to the input store (located in what is now known as Wernicke's area). Of course, part of the predictive value of the model would be saved even if the anatomical basis of the syndromes turned out to be at fault. The differentiation between receptive and expressive difficulties was entailed by the model on the basis of information flow among component information transducers, as well as, but independently from, the anatomical locale of the components.

An important feature of the model, indeed of the whole approach, was that it was designed to predict and explain new patterns of deficit. If well-known sets of co-occurring symptoms could be explained in terms of damage to an element in the model, then the finding of new syndromes could be expected based on the selective damage of other elements in the model, in particular those held to arise from lesioning the connexions between centres. Again like the modern models that 'lesion' computer simulations (e. g. Hinton/Shallice 1991), the anatomical claims can be differentiated logically from the behavioural claims.

These 'disconnexion' syndromes required new tests to reveal quite a different set of critical symptoms, where a symptom is as importantly a spared function as a deficit. The fundamental point here is the overall pattern of deficient and spared functions.

Historically, the most important symptom was the impairment or the sparing of repetition. The model held that there existed a subcortical connexion between the auditory word images in Wernicke's area and the motor word images in Broca's area that mediated

the exact repetition of heard speech, the lesioning of which (line 3) would lead to deficits on this task without other receptive or expressive difficulties. A repetition impairment, even if it had been tested previously, would have been quite obscure without this model; a selective sparing of repetition in the absence of comprehension or volitional speech in the 'transcortical' syndromes discussed below would have been even more obscure.

2.3. Levels of Deficit

The model was able to distinguish in principled way what we might now call levels of deficit. Thus speech production can be impaired because there is an impairment at the level of concepts (Centre B in the diagram), in a manner not fully explicated by Wernicke or his followers, but presumably such a deficit would affect the comprehension as well as the production of speech.

It could also be impaired on the route from concepts to Broca's area. This would produce the syndrome known as 'transcortical motor aphasia', in which concepts and comprehension are intact, repetition will be intact, as will the ability to enunciate words in, for example, a reading aloud task, but the selection of words in spontaneous speech will be affected. Actually, Lichtheim (unlike Wernicke) believed that concepts were not found in a single centre, like the motor word images, but were a set of entities spread over a considerable region of the cortex with the connexions converging on Broca's and Wernicke's area. This allowed him to predict that this type of disconnexion aphasias will be more severe the closer the lesion is to Broca's area (or to Wernicke's area) since the same sized lesion will sever more connexions. See Figure 21.2.

At a lower level, Broca's area and hence motor word images will be affected, leading, it is claimed, to effortful speech. Here repetition problems are of a kind predictable from the speech output deficit, while comprehension will be intact. Praxic and arthric difficulties will arise through damage to the peripheral route to the vocal musculature M *m* (in Fig. 21.1.).

Similarly, levels of impairment in comprehension are predictable from the model. Thus an interruption on route *a* A (in Fig. 21.1.) should result in 'pure word deafness' – a total inability to recognise a word without hearing impairment, while damage to the auditory word images in Wernicke's area should result in poor word recognition, with concomitant difficulties in repetition and word understanding (with speech intact); while lesioning the pathways A B to concepts will leave repetition and word recognition intact, but with poor word comprehension ('transcortical sensory aphasia'), and as can be seen from Fig. 21.2., lesions close to Wernicke's area should yield a more severe version of this syndrome.

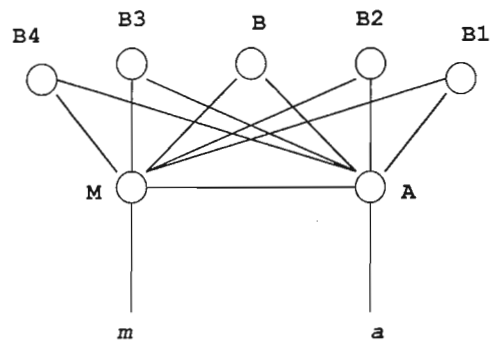

Fig. 21.2: The single concept centre, B ("Begriffe"), is replaced by concepts distributed over the cortex. (Redrawn from Lichtheim 1885, Figure 7)

2.4. Lichtheim's Account of Disorders of Reading and Writing

Less familiar perhaps to most students of aphasia are Lichtheim's attempts to integrate reading and writing disorders into his scheme. It had been well-known at least since Jackson's (1879) investigations, that if writing were perfectly preserved in the absence of speech, then some kind of core language production function must be intact and that the problem must lie at a more peripheral locus. Similar arguments can be made for the sparing of reading comprehension in the absence of spoken language understanding. Figure 21.3. (Lichtheim's Figure 2) shows his first provisional version of the relation between reading ("O"), writing ("E") and the other language functions.

Lichtheim saw very clearly the necessity for specifying functional relations between the subcomponents of his system. He postulated that the meaning of a written word depended on the prior elicitation of its 'auditory image'.

> "Reading postulates the existence of visual memories of letters and groups of letters. We may learn to understand writing through the connection between such visual representations (centre O [in Figure

21.3.]) and auditory representations: by spelling aloud we bring the auditory centre into action, and thus establish a connection, through the path O A, between O and B; in reading aloud, the tract O A M *m* is thrown into activity." (Lichtheim 1885, 437)

While modern authors would like to see some experimental evidence to support this idea, Lichtheim appeared to believe that some kind of armchair task analysis ("self-observation") or armchair developmental psychology would suffice where clinical evidence was lacking. Of course, the kinds of experimental evidence available to us, but not always employed by neuropsychologists, just were not available to Lichtheim. Nevertheless the basic idea that the meaning of a read word depends on first activating its pronunciation, still has currency in attempts to model normal skilled reading (e. g. van Orden 1987).

2.5. The Theoretical Significance of Writing Disorders

A revealing test of Lichtheim's methodological approach centres on writing. The plausible account offered in Figure 21.3. was based in part on armchair task analysis, but was supported by many observations of patients with Broca-like difficulties with speech output. Certainly modern views, as we shall see, correspond with the arrangements presented in Figure 21.3.

On the other hand, if this is the route to writing, then writing difficulties should be deducible from speech difficulties. Thus Wernicke patients, with paraphasic output, should produce comparable paragraphic writing. However, the problem arises from a famous case of Wernicke's in which the patient's speech recovered while difficulties of comprehension and agraphia persisted. Lichtheim thus modified the model so that the connection M E disappears and is replaced by the path M **A** E in Figure 21.4.

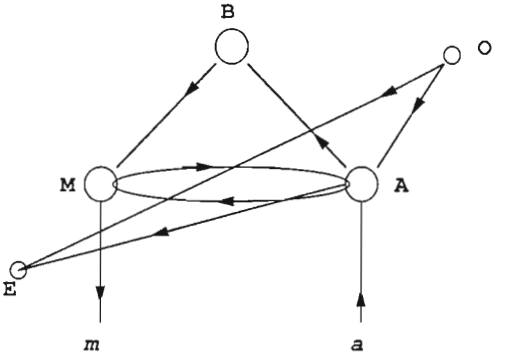

Fig. 21.4: The revised Lichtheim model incorporating reading and writing. (Redrawn from Lichtheim 1885, Figure 4)

"It is ... difficult to determine the path through which volitional or intelligent writing is executed. This tract must unite B with E, and clinical facts leave no doubt that it passes through M. There may be some doubt as to whether it leads directly to E, or passes through A on the way thither." (Lichtheim 1885, 437)

Lichtheim (1885, 444) notes that "it is not safe to draw a definite conclusion from a

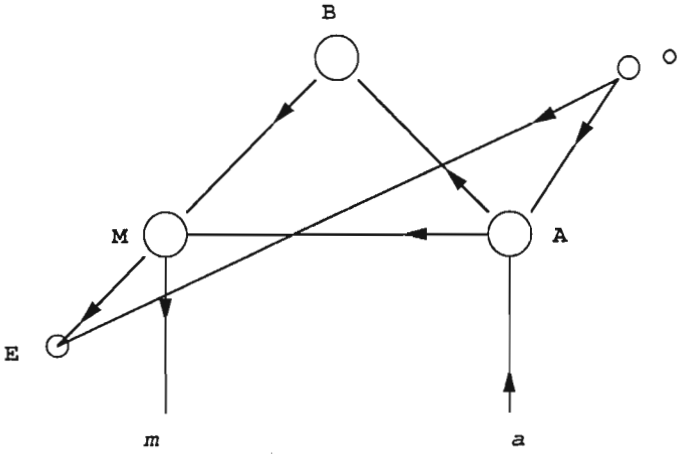

Fig. 21.3: Reading and writing incorporated into Lichtheim's scheme: First attempt. (Redrawn from Lichtheim 1885, Figure 2)

single instance", and presents two further cases (his Case I and Pitres's case) to support the route M A E. The model was thus modified on the basis of the pattern of deficit found in a single case, and supported by additional observations.

2.6. A Critical Modification: The Introduction of Feedback

A potentially critical, but universally neglected, modification concerns the path A M: this now clearly operates in two directions A M and M A. There is additional motivation for this. Lichtheim, like Wernicke, was concerned that damage to A also had an impact on speech production – speech became 'paraphasic', i.e. fluent, well-formed grammatically most of the time, but full of errors. This did not seem predicted by the model. Freud (1891) made this the fulcrum of his incisive critique of the model and, incidentally, his first published consideration of speech errors. Lichtheim seems to have wanted the auditory word image to somehow monitor or support speech production. Now this idea no longer seems quite so implausible as it did to Freud, for example. Levelt (1989) has proposed that monitoring speech production, even prior to actual utterance, is carried out by the speech comprehension process – a point to which I shall return. In Interactive Activation models (e.g. Dell 1986), the activation of whole words feeds forward to activate subword elements – phonemes and or syllables –, which in turn feed back activation to the whole word nodes, and so on. This has the effect of vastly favouring the production of strings of subword elements that make up whole, real words, while preventing the production of strings that do not.

It may be possible to formulate this kind of model in such a way as to locate the word nodes on the input pathway, but connected to lower level purely production elements in the output system. (For a critical analysis of the Interactive Activation account see Butterworth 1989; Levelt/Schriefers/Vorberg et al. 1991.)

There is a more general difficulty: if a speaker has a tendency to mispronounce words, the model would seem to allow several loci for the problem – (i) there is some impairment to motor word images in M; (ii) there is an impairment in the route M *m*, or maybe (iii) there is a deficit in the feedback monitoring of output, along the M A A M pathway. In any event the problem of paraphasias, to which we will return below, is that, whatever the pathway, simple interruptions will not tell you what kind of speech error will ensue.

Whatever the failings of this model, it allowed researchers to make specific predictions about patterns of symptoms that should and should not be found. Where they were not found, the model could, indeed had to be modified – as we have seen in the case of writing. It also encouraged researchers and clinicians to look at the whole pattern of symptoms – language use in all its forms and modalities.

3. Modern Box and Arrow Models

The nearest modern equivalent that integrates across modalities is shown in Figure 21.5. This is taken from Howard/Franklin (1988, 21: Figure 1). I have chosen this version as my stalking horse not because it is in any way crass or incompetent, but rather because the authors are the most careful of the deployers of the most elaborated of a range of essentially similar models (e.g. Morton/Patterson 1980; Ellis/Young 1988, 222: Figure 8.3). It derives, historically, not from Lichtheim at all, but rather from studies of normal word recognition and production, especially those formulated in terms of Morton's logogen models (e.g. Morton 1970; 1979) and from Marshall/Newcombe's (1973) pioneering account of the acquired dyslexic syndromes, which itself initially made no reference to Lichtheim.

Broad similarities in architecture are apparent. There are separate lexicons for speech input and speech output, which correspond to Lichtheim's auditory word images and motor word images, and a direct connexion between them (corresponding presumably to the route A M in Figure 21.1.); a further loop feeding back from M to A via some more peripheral apparatus, is carefully motivated by a beautiful series of experiments on a patient who lacks this loop. This corresponds to the path M A in Lichtheim's revised diagram (see above Figure 21.4.). For both Lichtheim and Howard and Franklin, there is direct access from semantic concepts (or the cognitive system) to motor word images, and from auditory word images to semantic concepts.

Like Lichtheim's model, there are also separate stores for input visual word forms (the 'Orthographic Input Lexicon') and writing

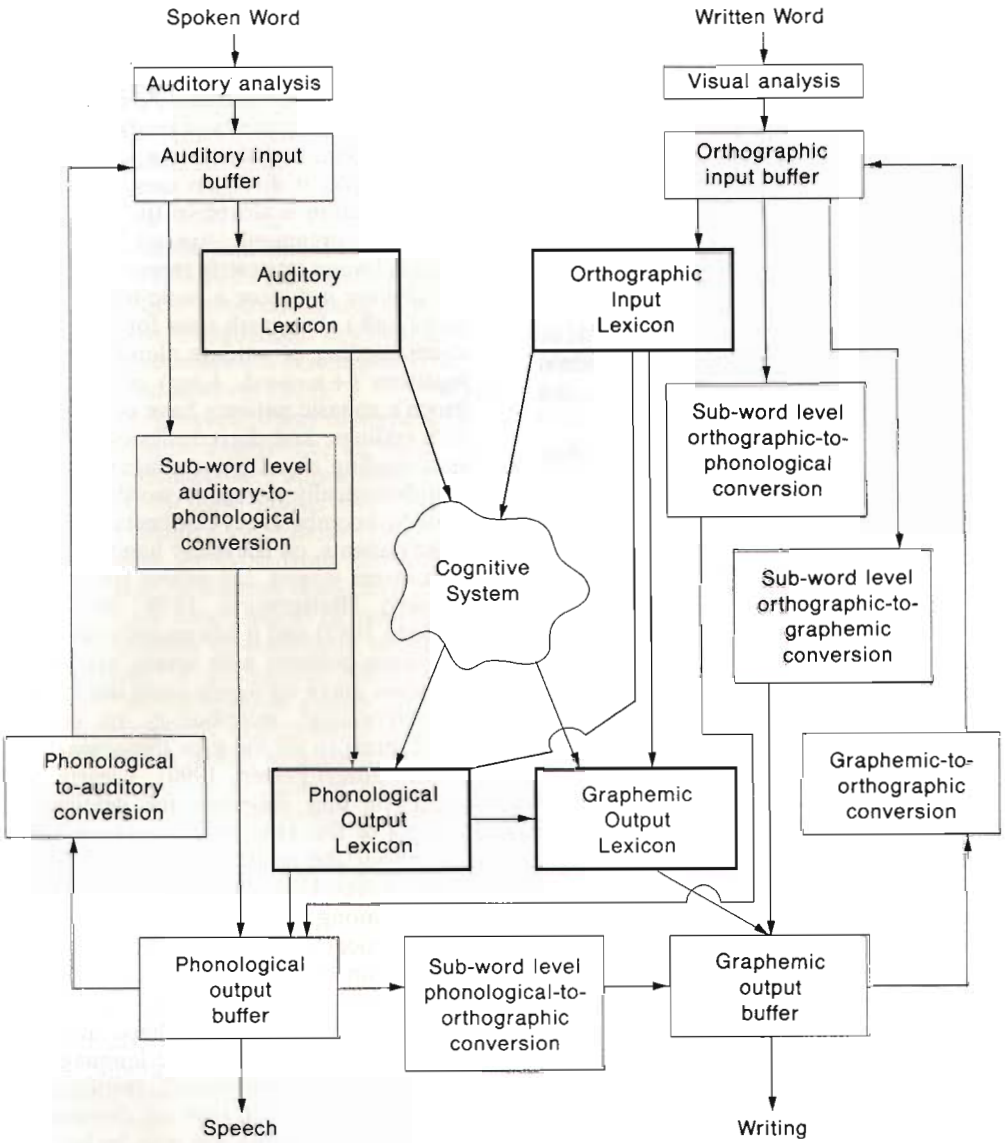

Fig. 21.5: "A simple model of single word lexical processing." (Howard/Franklin 1988, 20: Figure 1)

('Graphemic Output Lexicon'), and a direct connexion between them.

There are, however, several important differences from Lichtheim's original model, motivated in part by studies of performance by normal subjects in a wide variety of language tasks – a source of evidence unavailable to Lichtheim.

For written output, in this model but not in Lichtheim's revision (Figure 21.4.), the Phonological Output Lexicon (motor word images) maps directly onto the Graphemic Output Lexicon; and unlike Figure 21.4., there is no direct path leading from the Phonological Output Lexicon to the Auditory Input Lexicon to mediate volitional writing.

For reading, there is a direct route from the Orthographic Input Lexicon to the Phonological Output Lexicon, but no corresponding route O M in Figure 21.4.

However, the main differences are motivated by Marshall/Newcombe's (1973) idea that there could be two routes for reading a word: one which maps the whole letter string onto meaning and hence onto a pronunciation; while the second utilises the as-

sembly of subword correspondences between letters and sounds. The former will in any case be needed for irregularly-spelled words, while the latter procedure will be needed to read aloud new words or for nonwords. In Figure 21.5., in addition to the two routes from visual input to spoken output, the idea of two routes is employed to map from visual input to written output, and from auditory input to spoken output. This basic format of both whole-word and sub-word processes is repeated where possible. As with Lichtheim, a critical test of the elements of the modern model is its ability to account for patterns of spared and impaired functioning.

The well-known dissociations in reading are a case in point. Some patients are able to read only words, but are unable – or very severely impaired in reading nonwords – the classic deep and phonological dyslexias – which is normally explained in terms of this model as an impairment to the 'subword level orthographic-to-phonological conversion route', while other reading routes are intact; while other patients are unable to read irregular words, but are good at reading aloud both regular words and nonwords. This is explained as a preservation of the 'subword level orthographic-to-phonological conversion route' with deficits to whole word reading routes. (See for example Ellis/Young 1988, Chapter 8.)

Similarly, but much less well-known, is the dual route account of repeating heard speech. According to the model, subword as well as whole word procedures can be used. The subword route enables us to repeat new words or nonwords as well as real, known words. The inability to repeat nonwords, while being able to repeat real words, was first documented by Goldstein (as P.S. in Goldstein 1906 and as Case 7 in Goldstein 1948). M.K. is a modern case described in detail by Howard/Franklin (1988), who also provide a critical summary of previously reported cases.

One further obvious difference is that while short-term memory tasks ('repetition') are mediated by the line A M in Lichtheim's diagram, they are subserved by two buffers and a loop for rehearsal in Figure 21.5. The need for more complex theoretical apparatus to deal with short-term memory is motivated largely by seemingly endless experiments on the factors determining normal memory span (Baddeley 1986).

4. A Comparison and Critique

This model however suffers from the same deficiencies as Lichtheim's. At best, it represents only static aspects of the language processing system, and then only some of these. For example, it does not specify what kind of information is stored in the lexicons, nor how it is organised. Are all the words a speaker knows separately represented, or does the speaker just store a basic form of, say, a verb (*walk*) along with rules for producing or understanding or reading aloud all the conjugations (+*s*, +*ed*, +*ing*) or both? Some Broca's aphasic patients have problems with verb endings, and deep dyslexics have problems reading aloud the endings on all types morphologically complex words (see Marshall/Newcombe 1980; Coltheart 1980); Wernicke patients, on the other hand, even those with severe jargon, can inflect spoken words correctly (Butterworth 1979; Butterworth/Howard 1987) and it has recently been found that some patients with severe word-finding problems make up words using the full range of derivational morphology to construct novel forms to fill the gaps (Semenza/Butterworth/Panzeri/Ferreri 1990). Clearly, then, prediction and diagnosis for detailed phenomena of this type will depend on hypotheses about the internal organisation of the lexical stores. Unfortunately, there is no unanimity among theorists – is there ever? – about lexical storage and retrieval. Useful reviews can be found in Butterworth (1983) and Frauenfelder/Schreuder (1991).

More strikingly, and perhaps more critically, dynamic aspects of the language processing system are not treated. Insofar as they are represented at all, they are designated by lines in the diagrams, not even by boxes. No attempt is made to say how elements in the lexical stores are accessed, how for example the pronunciation – a phonetic plan – is constructed on the basis of lexical retrieval or of some sublexical assembly procedure, and there is no awareness, apparently, that words are typically produced and received not as single units but as sentences or whole discourse. There appears to be an assumption that words in construction are a simple sum or concatenation of words in isolation, which, it has to be said, goes against everything that is known, not least by the proponents of the modern model, about language production and comprehension. For example, it is known that context profoundly affects both

visual and auditory word recognition: it affects the visual exposure duration required for accurate recognition (Morton 1969), and can play a part in auditory word identification within the first 100 msecs of a heard word (Tyler 1984). In specialised models of speech recognition, like the "cohort model" (Marslen-Wilson 1987) or TRACE (McClelland/Elman 1986) contextual information is automatically invoked at the earliest stages of the recognition process. Similarly, it is known that context contributes to word-finding in spontaneous speech (Goldman-Eisler 1958; Beattie/Butterworth 1979).

These models provide no way of organising the words to form a sentence, let alone a meaningful or contextually appropriate one. There is no way for these models of the hearer to distinguish *Brutus killed Caesar* from *Caesar killed Brutus*, let alone from *Brutus was killed by Caesar*. It follows that there is no coherent way for either model to identify or explain impairments in combining words to form sentences: to distinguish agrammatic output from paragrammatic output; nor to say why a patient who can understand single words quite well is unable to understand sentences correctly. That is there is no syntax and only the sketchiest treatment of semantics.

5. A Modern Treatment of Paraphasias

Of course, modern research has made some notable advances in describing the dynamic aspects of language production since the time of Lichtheim which could be usefully incorporated into future models. One example is the process by which a stored lexical representation is realised as a string of phonemes – what is usually called, 'phonological encoding' ('PE' henceforth). The problem of paraphasias, especially phonemic paraphasias, has been critical to the assessment of the Lichtheim model. As was mentioned above, Freud made it the basis of his critique, and Lichtheim himself went through theoretical contortions to reconcile Wernicke's model with the clinical observations.

Without some idea of what actually happens inside boxes or along the arrows, it is nearly impossible to derive predictions about the fine-structure of behaviour. The idea that it has something to do with the centre for auditory word images is, at best, far too unspecific. What kind of error is likely to follow from an interruption in Auditory Word Images, even supposing these really do have something to do with mispronunciations. Similarly, interruptions in M or in the path M *m* in Figure 21.1., at most, allowed Lichtheim to distinguish speech disturbance with or without disturbance of volitional writing. In Figure 21.5., the situation is scarcely better. An interruption in the path between the Phonological Output Lexicon and the Phonological Output Buffer can be discriminated from deficits involving solely nonwords, but nothing can be said about the kinds of errors that will be made.

The model of PE that is described below will be used to attempt a diagnosis of a single paraphasic error, [semənt]. A fluent, Wernicke's aphasic patient, DJ, with largely recovered comprehension, produced mildly neologistic, but frequently paragrammatic and paraphasic speech, as in the following excerpt with the item of interest in bold:

(1) E: What do you do?
DJ: I've got a publican – publican.
E: Uh-huh. Where's that?
DJ: Old Bethnal Green Road ...
E: Are you the manager?
DJ: [dəmiəun] I'm sem- sem- What they call **[semənt]** ... Yer. Your own governor. Your own governor. I's mine.
E: Your a tenant.
DJ: I work to Truman's. But it's mine, like, you know. What I mean. Actually now I'm I'm actually the top thing you can do. You you are the – you work for the brewery as a [emnənt].
(For further case details, see Butterworth/Howard 1987)

Intuitively, the phonological encoding of a known word entails accessing a stored representation of the sound of that word, what I shall term the Phonological Lexical Representation (PLR) (a motor word image in Lichtheim's model; an entry in the phonological output lexicon in Figure 21.5.). How a PLR is itself accessed is a matter of some controversy, and will not be treated here; for, discussions see Butterworth (1989; 1992); Levelt/Schriefers/Vorberg et al. (1991).

PLR has to contain sufficient information to specify, for the word in its intended speech context, (1) the syllabic structure of the word, (2) the stress pattern of the word and (3) the segmental contents of the syllables. I leave aside entirely the question as to whether the

PLR is an 'underlying' or a 'superficial' phonological representation. (But see Caplan 1987, for arguments in favour of an underlying representation).

Following a number of authors, I will assume that these three types of information are represented separately (see Levelt 1989, chapter 9, for a review; cp. art. 1). A set of processes must then translate the information stored in the PLR into the phonetic representation appropriate to the current speech context that is ultimately passed on to the articulators. Encoding subsystems "spell out", in Levelt's (1989) useful term, information in a PLR. One can think of a PLR as containing phonological information in a condensed or abbreviated form, which requires elaborating before it can be deployed by later processes. Elaboration may involve adding information on the basis of general rules of phonology, which the (normal) speaker may be assumed to know. For example, it may involve generating allophonic variants appropriate to the current syllabic context, like lengthening a vowel before a voiced obstruent. Unfortunately, we have insufficient evidence to be precise about how this spelling out might work. Allophonic variation may already be explicitly encoded in the PLR. PLRs may also be underspecified in a more technical sense: that is, some phonetic features will systematically not be represented in the PLR, so that for example, the consonant following /s/ in *start* will be marked for place but voicing is left unspecified, and is represented instead by an archiphoneme /T/ that could be realised as either /t/ or /d/. Other types of underspecification in underlying lexical representations have been suggested in phonological theory (e. g. Kiparsky 1982; Archangeli 1985), and some of the ideas have been recruited to explain normal speech errors (Stemberger/Treiman 1986; cp. art. 5) and aphasic paraphasias (Béland 1990; cp. art. 17).

I shall follow several authors (Levelt 1989, Chapters 8 and 9; cp. art. 1; Shattuck-Hufnagel 1987) in assuming that translation processes have the form of a slot-and-filler device. The slots are defined jointly by spelling out the syllabic structure (how many syllables, and their form) and spelling out the prosodic structure – the stress (and pitch, where relevant) of each syllable. Information about the segmental content is spelled out and inserted into the appropriate slots. (This account is a considerable simplification, as well as a slight modification, of Levelt's in several ways, and the reader is urged to consult his book for a fuller description of the processes that might be at issue).

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

This outline account presented diagrammatically in Figure 21.6., leaves open many details, some of which will be discussed below.

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

In (1) above, our jargon aphasic patient, DJ, produced ['semənt] instead of the target, *tenant*. Single phoneme substitution paraphasias are not uncommon in aphasic speech (e. g. Fry 1959), nor indeed in normal speech (e. g. Shattuck-Hufnagel/Klatt 1979), yet a definitive locus in the production system needs to be determined. DJ may have a corrupted lexical representation for *tenant* in which the initial /t/ has been replaced by an initial /s/; or the /t/ may have lost elements of featural specification, in the PLR, so that only something like [−sonorant, +coronal] remains to specify the initial segment and additional features will need to be generated, in this case incorrectly; or the initial phoneme /t/ has just been lost in its entirety, yet syllabic structure clearly indicates that an initial consonant is required; or, like normal errors, where loss of information at the level of PLR, is not usually an option, something has gone wrong translating the PLR /t/ into the phonetic plan; or something arthric or praxic difficulty results in /t/'s, or stops more generally, being properly articulated.

To eliminate some of the candidate interpretations, one needs to see what other types of error the patient makes, in particular, one needs to see whether the patient makes the

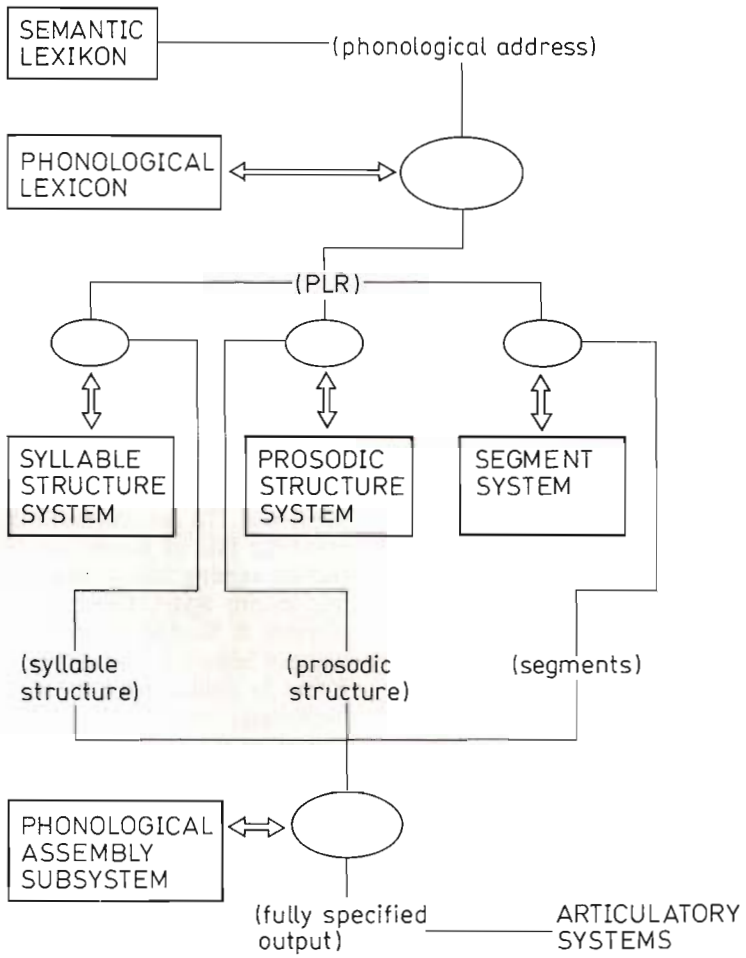

Fig. 21.6: *Outline model of phonological encoding processes.* The operation of this model can be best illustrated by stepping through the encoding of the word *tenant*, and the paraphasia ['semənt].
1. A phonological address is retrieved from the SEMANTIC LEXICON and can be thought of as an n-tuple defining a location in the PHONOLOGICAL LEXICON – e. g. ⟨2, 3, 6 ...⟩. It will have as neighbour, say, *tennis* at ⟨2, 3, 7 ...⟩. The generation of both target and paraphasia are held to start with the retrieval of the correct address.
2. The PHONOLOGICAL LEXICON associates the address with a PLR (Phonological Lexical Representation) that contains information as to how *tenant* should be pronounced – the segments it contains, and its syllable and prosodic structures. For reasons that are explained in Section 1.1, the PLR for *tenant* has not been corrupted in store.
3. This information is spelled out by dedicated, independent systems for SYLLABLE STRUCTURE, PROSODIC STRUCTURE and SEGMENTS. For the correct output, *tenant*, all the information in the PLR is correctly spelled out. For ['semənt], syllable and stress information may be fully available, though defaults could yield similar outcomes, but it is assumed that some or all of the information about the first consonant has been wholly or partly lost in transmission; the segment system generates a default segment, /s/, either from scratch, or from residual information about place of articulation, with manner information lost.
4. These are then assembled by the PHONOLOGICAL ASSEMBLY SUBSYSTEM, which fits the segments into slots in a prosodically specified syllable-structure. Thus the initial /t/, or the default /s/, is fitted into the onset position of the strong first syllable. The fully-specified output needs to include all relevant information for the ARTICULATORY SYSTEM.

The control processes, indicated by ovals, enable the generation of default information from the associated systems, or elsewhere, in the event that relevant information is missing from the PLR. For further explanation, see text. (Butterworth 1992, Figure 1)

same error every time he or she tries to produce the target.

In the case of DJ and *tenant* we do have some relevant data. As we saw in (1), DJ was a publican, working as the tenant of a brewery, a fact he was at pains to convey, since a tenant, who holds a lease on his pub, has a different, and, in DJ's view, a higher, status than a mere manager who simply operates the pub for a salary. Five examples, in two sessions, of this attempts to produce *tenant* in spontaneous conversation were recorded, and are reproduced in example (2).

(2) ['emnənt ... 'semənt ... 'tenənt ... 'tenəmən ... 'təneit]

Each attempt came from a separate sentence, so the sequence cannot be regarded as conduite d'approche. Taking the incorrect attempts as a whole, one can see that each segment, /t, e, n, ə, t/ is produced in its correct word position at least once, and no error is produced more than once; and one attempt was fully correct. It is reasonable to suppose, therefore, that the PLR of the word has not suffered permanent corruption, either through the replacement of a target segment by an intrusion, nor by the loss of a phonetic feature on one or more of the segments. Leaving aside arthric or praxis problems, from which DJ did not suffer, some problem in translating an intact PLR into a phonetic plan seems the most likely explanation, though the variability of error forms precludes proposing a specific locus in the translation process. The most likely account is that on each attempt to say the word, some of the information about the segments was lost in transmission, but in a rather unsystematic, perhaps random, way. (This is not, of course, to say that DJ had a fully preserved vocabulary that sometimes got scrambled in his attempts to talk, but only that for this target, the translation explanation appears the most consistent with the evidence so far presented.)

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

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

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

(3) Target: eskimo
Session 1: ['esiməu]
Session 2: ['æstiməu]
Target: hedgehog
Session 1: ['dɪdʒɒg]
Session 2: [ɪg, ɒs, 'hɪdʒɒg, 'egɒg], H-E-
Target: jacket
Session 1: ['dʒækə, dʒæk]
Session 2: ['dʒæk], zipper, [zɪpəweiz]

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

In any event, the model presented in Figure 21.6, and its interpretative apparatus, allowed us to explain the pattern of paraphasic errors in a single patient in a plausible way and it

stressed the need for data on item-consistency that is usually neglected. (Cp. Butterworth 1992)

6. Conclusion

To say that a model is deficient in obvious and important ways, is not to say that it is without value to a science. Indeed, according to Lichtheim the very act of schematization in diagrammatic form (Figure 21.1.) historically turned out to be important:

> "The necessity of differentiating still further types [of disorder] struck me on attempting to schematize the forms hitherto known, for the purposes of instruction." (Lichtheim 1885, 435)

Howard/Franklin (1988, 99) note that they are using the model in Figure 21.5. "because it is the only lexical model that specifies all the word processing routines we have investigated with (the patient) MK in sufficient detail to permit a discussion" of his problem in term of a small number of information-processing impairments. They conclude that this patient has impairments to four boxes, two arrows and to the cognitive system itself (Howard/Franklin 1988, 111). Such a conclusion, if not inconceivable without this kind of diagram, would at the very least have been hard to imagine.

The neuropsychologist faces a dilemma: on the one hand, the aphasic patient arrives in the clinic with interrelated deficits in more than one modality, and to understand what is wrong, and just as importantly, what is right, a multi-modal model is needed; on the other hand, to understand the fine structure of each behavioural abnormality a detailed, dynamic model of the processes of constructing continuous speech output, or interpreting continuous speech input, will be needed.

7. References

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