

## **STUDIES ON THE "SPEECHLESS MAN": The Case of Speech Automatism**

GERHARD BLANKEN – JÜRGEN DITTMANN – CLAUS-W. WALLECH  
*University of Freiburg i. Br.*

### *1. Introduction*

Looking up the term "aphasia" in lexica or dictionaries, one often finds a reference to the meaning of the ancient Greek word *aphasia*, which is translated as speechlessness or, rather more loosely, as the loss of the ability to speak or of the capacity for language. As further information it may be noted that this condition is a case of a neurogenous language deficiency. This widespread conception of the phenomenon of aphasia contrasts crassly with the experiences gained in conversations with aphasic patients. Aphasias can be described more appropriately as disturbances of language or rather of the processing of language, and in sub-groups such as Wernicke's and anomic aphasics the fluency of the production of language is usually well preserved. In other aphasic patients, however, suffering from Broca's or global aphasia, speech production is reduced in fluency and effortful. The errors observed in aphasic patients' speech productions affect in particular the grammatical organisation of speech (agrammatism, paragrammatism), phonological planning (phonemic paraphasias and neologisms), and semantic-lexical processes (word finding impairment, semantic paraphasias) (see Huber, Poeck & Weniger, 1982, for a detailed neurolinguistic description of aphasic syndromes and symptoms).

Indeed, as almost all aphasics can produce speech, although perhaps in distortion, and some of them even tend to speak more than before (logorrhea), one can hardly speak

of "speechlessness" in the vast majority of cases. However, the term "speechlessness" has been used by a number of authors who were fully aware that aphasic patients are not mute (Jackson, 1879). In fact, the English neurologist Hughlings Jackson introduced the term in order to indicate that useful, propositional speech could not be produced by those patients he called "speechless" and he investigated in great detail those utterances which were not propositional, such as neologisms ("jargon") and automatisms ("recurring utterances"). Jackson interpreted speech automatisms as elements of utterances pathologically preserved in *statu nascendi*:

Thus the disease causes loss of speech; it permits the increased dischargeability of the right half .... The greater excitability of a centre uncontrolled by removal of its higher centre is supposed to be temporary, but the particular activity may be kept up by repeated use; the speechless man's recurring utterance is being uttered very often; it keeps up what was doubtless at first a temporary organization. (p.192).

Therefore, although the conception of aphasia as a loss of speech is misleading for the vast majority of cases of aphasia, there are patients whose linguistic processing mechanisms only permit the production of a few or individual words, syllables, or vowels and who, instead of being able to use linguistic forms in accordance with their intentions, only have at their disposal the repetition of these stereotyped linguistic elements.

In its prototypical form this condition is infrequent. De Bleser and Poeck (1985) indicate that only 2% of the aphasics investigated by them revealed the production of language which exclusively consisted of fluently uttered chains of consonants and vowels (CV-recurring utterances, e.g., di-di-di). If one does not restrict the symptomatology to the CV-type of utterance, the proportion of patients with exclusive production of stereotyped utterances might be estimated as being below 5% of patients suffering from chronic aphasia. Exclusively stereotypical productions may also occur with the advanced breakdown of language and other higher mental functions in dementia (Leischner, 1951).

In the second half of the last century, cases with stereotypical productions were for a short time the focus of neuropsychological research, which was just beginning to develop (cf. Lebrun, 1986, for a review). Thereafter, efforts to describe the ways of appearing, the causes, and the course of this strange aphasic symptomatology were made only quite recently. Taking particularly into account the most recent results of research, the present article intends to provide a survey of the empirical results, the theories, and hypotheses

which could have explanatory value, and the general conceptual models concerning the production of language in the context of which the special results of these neurolinguistic studies can be placed.

## 2. Definitions

Up till now there is no consensus among aphasiologists with regard to how stereotypical linguistic remnants can be denominated. We favour the neurolinguistic term "speech automatisms" as it was introduced by the Aachen group of researchers. Huber et al. (1982) defined the linguistic automatism as "a constantly recurring utterance which is formally rigid, consists of neologistic sequences of syllables, interchangeable words or phrases, does not fit into the linguistic context either lexically or syntactically, and which the patient produces in contrast to the intention expected by his interlocutor" (p. 81). The term "recurring utterances" is used for a sub-group of linguistic automatisms. It refers to utterances "which consist exclusively of syllables or sequences of syllables which are lined up in their production (e.g. dododo, tatata, tautau, gogogogo, manne, manne)" (ibid). In most of the English literature the term "recurring utterances" is used for any speech automatisms. Code (1982) defined "recurrent utterance" in the spirit of Jackson (1879) as "an utterance made up of either real words or a non-meaningful string of speech sounds which some aphasic patients produce either every time they produce speech or just sometimes" (141). Alajouanine (1956) used the concept of verbal stereotypy in order to designate the symptomatology and further qualified it as "a permanent stereotyped verbal expression with or without linguistic meaning unconsciously and involuntarily uttered" (6).

## 3. The Historical Point of Departure: Broca and Hughlings Jackson

It is astonishing to see to what extent the descriptions of the neurolinguistic status of patients with linguistic automatisms diverge. Broca described two patients whose speech production was reduced to minimal remnants: Leborgne, whose phonological utterances consisted only of the CV-syllable "tan" (ta), and Lelong, who could only produce "oui", "non", "tois" (for "trois"), and "toujours" as well as his name, partially, ("lelo"), upon being asked. Broca (1861) believed

...la faculté du langage ... persiste évidemment tout entière, puisque les malades comprennent parfaitement le langage articulé et le langage écrit; ... puisque enfin ceux qui sont lettrés, et qui ont le libre usage de leurs mains, mettent nettement leurs idées sur le papier. Ils connaissent donc le sens et la valeur des mots, sous la forme auditive comme sous la forme graphique. .... Ce qui a péri en eux, ce n'est donc pas la faculté du langage, ce n'est pas la mémoire des mots, ce n'est pas non plus l'action des nerfs et des muscles de la phonation et de l'articulation, c'est autre chose, c'est une faculté particulière considérée par M. Bouilland comme la faculté de coordonner les mouvements propres au langage articulé, ou plus simplement comme la faculté du langage articulé, puisque sans elle il n'y a pas d'articulation possible. (333)

Although Jackson (1879) agreed with Broca on some points, for example, on the fact that the articulatory and vocal instruments of these patients were usually intact and that linguistic comprehension was not extinguished, he disputed the retention of the capacity for language, which was maintained by Broca. This becomes particularly clear in his conception of the written language capabilities of the aphasic with automatisms:

He cannot write. - That is to say he cannot express himself in writing. ... Since he cannot write, we see that the patient is speechless, not only in the popular sense of being unable to talk, but altogether so; he cannot speak internally" (p.163). And at another point: "I know that cases of loss of speech have been recorded by eminent physicians, in which ability to write was not lost. The chronic cases of this kind that I have seen have been mostly cases of pretended loss of speech. Besides, how is it conceivable that a person who has lost speech should be able to express himself in writing? If a person can express himself in writing he gives proof that he has not lost speech. We must speak internally before we write - before we express ourselves in writing. (1874:131)

#### 4. Types of Speech Automatisms

Jackson was the first person who carried out a detailed neurolinguistic analysis of automatisms. He distinguished four types: firstly, those consisting of jargon utterances (e.g. "yabby, me committimy, pittymy, lor, deah"); secondly, those formed of real words (e.g. "man", "one", "awful"); thirdly, those consisting of short phrases (e.g. "come onto me"; "yes, but you know"); lastly, he emphasized that the particles "yes" and "no" can frequently be observed as automatisms alongside other automatisms or as a unique type of utterance. Alajouanine (1956) distinguished two forms of occurrence, firstly, those with, and secondly, those without linguistic meaning. He subdivides the latter type into "iterative stereotypies", which are syllables or sequences of syllables constantly repeated, and "jargonised stereotypies" which he understands as being the constant reproduction of formally constant neologisms or meaningless chains of words.

Code (1982) reports on 97 speech automatisms in a sample of 75 patients (more than one automatism may be produced by a single aphasic patient). "Real word recurrent utterances" formed the substantially larger group in his observations. They consisted of proper names, curses, fixed expressions, single words of different grammatical categories, composite nouns, and a number of phrases which mostly began with "I". The list of automatisms without linguistic meaning ("non-meaningful recurrent utterances") predominantly included CV-syllables or their combinations. In a recent review, Code (1987) observes that real word automatisms were non-propositional and "either emotionally charged phrases or expletives or serial-automatic in nature for the part, and ... appear to be holistically produced" (69) and assumes that they could well be manifestations of right hemisphere speech. "Non-meaningful recurrent utterances", on the other hand, are supposed by Code to result from a severely compromised left hemisphere phonological system without any right hemisphere input.

Blanken et al. (1987) investigated 30 aphasics with automatisms. Three patients uttered real word automatisms exclusively and two other patients exhibited both lexical and non-lexical automatisms. One patient, for example, produced alongside the very frequent sequence of syllables "di:bn-di" the further automatism "bitte sehr". The majority, however, was characterised by automatisms of the syllabic type (e.g. "do-do"; "na-na"). These findings are discordant with Code's results summarised above. One reason for the divergence of results may be the restriction to patients with predominant production of speech automatisms (more than 45% in spontaneous speech) in the study of Blanken and co-workers. In addition, the different results might be due to the exclusion of "verbal stereotypies", i.e. empty phrases which recur persistently during conversation, but are, for the most part, appropriate in the context and which can also occur in milder expressive aphasic disorders (e.g. "well I know", "so and so", "oh my god"; cf. Huber et al., 1982).

The longest automatism known to us is the sentence "Ich geh mit dir in 20 Jahren" (I shall go with you in 20 years), which was the sole spontaneous speech production of an aphasic patient over many years. Another aphasic utters "Ich möchte sprechen" (I should like to speak) whenever he starts speaking, upon which fragmentary, but possibly task-related productions may follow. The word sequence within such multiple word automa-

tisms may be fixed, but this is not necessarily the case. A patient of ours showed the following variations:

arbeiten (work (infinitive))  
 und (and)  
 ich will (I want)  
 ich arbeiten und (I work (infinitive) and)  
 und arbeiten (and work (infinitive))  
 ich will arbeiten (I want to work)  
 ich will arbeiten und arbeiten (I want to work and work)  
 ich will arbeiten und lernen (I want to work and learn)

The CV-syllable or the only rarely observed VC-syllable (e.g. "it-it") may not constitute the smallest automatised elements. Kremin (1987) reports on an aphasic with the stereotyped vowel sequence "ah-oh-oh" as an automatism (VVV-automatism). Blanken et al. (1986) described a patient who produced the utterances "oh-ih" and "oh-si" alongside the CV-automatisms di-di and si-si.

##### 5. "Language" Beside the Automatisms

Many aphasics with automatisms exhibit exclusively stereotypical spontaneous speech. On the other hand, others produce real linguistic forms beside their automatisms. Quite often series, for example the number series 1-10 or the days of the week can still be recited rather well, prompted or even spontaneously. Familiar proper names can often still be produced, too. "Yes" and "no" may be used appropriately, although the differentiation from the homonymous automatism is often difficult. Finally, interjections, particularly particles with a high frequency of occurrence in spontaneous speech (e.g. "also" (thus)) as well as expressions (such as "ah", "oh", "mhm") can be observed frequently. Moreover, there are patients who can produce lexical units in propositional application, however, to a severely restricted extent. One can frequently observe further pathological linguistic symptoms in patients with a non-exclusive production of automatisms. Some patients are characterized by a high incidence of non-stereotypical neologisms. Perseveration is frequent, and echolalia can also be occasionally observed.

Jackson emphasizes that the non-stereotypical utterances, the so-called "occasional utterances" of severely aphasic patients, frequently consist of non-propositional speech. In particular, he found patients who often uttered curses beside their recurring utterances. In addition to this, he found that the linguistic performances of patients improved when they were excited. He concludes from this that only the voluntary and "intellectual" language is lost, but that their "emotional language" is retained. With the latter he understands, apart from curses and interjections, wide areas (with the exception of mime) of non-verbal and paraverbal communication such as laughing, smiling, wrinkling one's forehead, prosodic characteristics of utterances, and variations of the voice's qualities, as well as the ability to sing, which may be retained to the extent of correct production of the words of familiar songs.

De Bleser and Poeck (1985) pursued the question of whether patients with recurring utterances can utilize their prosodic means for communicative goals. They found that the global aphasics they investigated, who exclusively produced CV-automatisms, showed stereotypical non-communicative behaviour both on the level of variations of the length of utterances and of variations of the pitch of sounds, and that the recurring utterances, in contrast to the clinical impression, possessed no communicative function.

Blanken et al. (1986) reported that a group of aphasic patients with predominant automatism production was extremely inhomogenous with respect to prosodic characteristics of the production of automatisms. Some patients showed a very high degree of prosodic stereotypicality. One patient, e.g., uttered his automatisms with the predominant constant accent structure na-tá-ta-tá-ta, so that an extreme restriction of prosodic capacities can be assumed. On the other hand, other patients showed a greater variation of pitch and accent. The elements of these patients' utterances were connected by a pervading prosody which reminded one of patterns of sentence intonation and was at the same time unexaggerated, similar to, e.g., the use in narrative speech.

In amplification, it should be mentioned that patients with severe aphasia, including those with automatism production, are far better in their communicative performances than in their linguistic performances. They may have at their disposal communicative strategies which they use in a compensatory fashion where non-verbal communication can be employed (cf. Herrmann, 1987, particularly chapter 4.3).

### 6. *On the Course of the Symptoms*

Alajouanine (1956) describes observations on the development and remission of repetitive stereotypical speech. In a collective of 30 aphasics with automatisms, 19 showed improvement of this symptomatology, 6 showed spontaneous remission in less than a month. The latter group consisted of 5 patients with Wernicke's aphasia and one patient with anarthria (in modern terminology probably: Broca's aphasia with predominant speech apraxia). All these cases had initially exhibited automatisms with linguistic significance. The remaining patients with remissions, who showed improvement after a period lasting up to 2 years, were all classified as Broca's aphasics. Eight of these produced lexical and 5 non-lexical automatisms. According to Alajouanine's nomenclature, these patients were particularly severe cases of Broca's aphasia with persevering production of automatisms, although he does not define this aphasic syndrome with reference to an agrammatical symptomatology, rather by pointing to the phonological-phonetic deficiencies.

Alajouanine describes a process of regression usually passing through a number of stages for automatisms which remit slowly, although he adds in qualification that not all remissions necessarily follow this scheme. In a first stage, the use of automatisms becomes differentiated according to prosodic characteristics such as intonation and the speed of utterance. In a second stage, the patient begins to realize the inadequacy of his utterances and attempts to gain control, e.g., by slowing or terminating them. A third stage is reached when changes and variations in the linguistic form occur, be it that a new automatism appears which replaces the old one or that non-stereotypical utterances increasingly appear beside the production of automatisms. In the final stage the automatism disappears completely. Instead of the compulsive automatisms, which are, however, uttered fluently, voluntarily produced linguistic expressions can now be formed. These can, however, only be brought forth slowly and only with distinct phonetic disturbances. Alajouanine reports that only a sub-group of patients shows agrammatical symptoms, a second sub-group is characterised by its strenuous and phonologically distorted production of speech, but follows the rules of the grammatical organisation of utterances.

Blanken et al. (1986) report on 4 aphasics who produce automatisms transiently. Only 2 exhibited aphasia with clear agrammatical symptomatology in the remitted stage.

The remaining patients revealed symptoms of a non-severe Wernicke aphasia or transcortical-sensory aphasia after rapid remission of automatism production.

Since agrammatism is known as a rather stable and slowly remitting complex of symptoms, the observations on patients with terminated automatism production allow the inference that agrammatism was not present in the status of automatism production and thus agrammatism does not constitute a necessarily co-occurring disorder in patients with speech automatisms.

### 7. *Where Do Speech Automatisms Come From? Neurological and Neuropsychological Evidence*

As has been reviewed in the introduction, speech automatisms raised considerable interest in the early days of aphasiology. Broca (1861) viewed their presence as indicative of the severe disruption of processes of the production of articulated speech, the faculty of which he localised in the foot of the third convolution of the left frontal lobe of the brain. Transposed into modern concepts and theories of speech production, he would interpret speech automatisms rather as a symptom of speech apraxia than of aphasia. Jackson (1879, see above) explicitly stated his view that recurring utterances were a symptom of the severe breakdown of language proper, their basis being a disinhibition of lower, right hemisphere systems, resulting in the permanent establishment of originally temporary language fragments, frozen in the process of production when means of their modulation were destroyed by disease. Whereas Broca postulated a left hemisphere generator for speech automatisms, Jackson proposed a right hemisphere one. The question of the localisation of the automatism generator is still unresolved.

Interest in speech automatisms faded when in the last two decades of the 19th century neurologists set out to explain the normal functions of the brain and literally dozens of centres for higher mental functions were localised. This period was followed by a fierce, but probably premature, debate as to whether or not modular theories of these functions were appropriate at all and whether cognitive modules, if they existed, could be localised in discrete brain regions.

In more recent years, Kornhuber (1977) speculated on the basis of clinical observations that the production of speech automatisms indicated a dysfunction of a "program generator" situated subcortically in the basal ganglia. This hypothesis would link speech

automatisms with repetitive and iterative speech and motor phenomena found with extra-pyramidal diseases, such as Parkinsonism. It was confirmed by Brunner, Kornhuber, Seemüller, Suger & Wallesch (1982) who, in a series of 40 aphasic patients suffering from the consequences of cerebral infarctions, found speech automatisms in 9, all of whom had lesions of the basal ganglia and the temporal language area. Poeck, De Bleser & Keyserlingk (1984) presented a number of cases with automatism production but with conflicting neuroradiological evidence. In the last few years neurologists have become increasingly critical of the localising value of neuroradiological information as the commonly used CT-scans are able to show unequivocally only those areas of the brain where there is complete tissue destruction but not those where only the neurones or their connections are destroyed. Consequently, it has been argued (Wallesch and Papagno, 1988) that only careful analysis of the vascular pattern underlying CT-pathology may provide evidence of the anatomical damage.

Following these lines, Haas, Blanken, Mezger & Wallesch (1988) undertook an analysis of the CT-scans of 18 aphasic patients with speech automatisms and another 14 with infarctions of similar size and aphasia of similar degree of severity. They found a) that all patients suffering from automatisms exhibited infarctions which involved the area of supply of deeply subcortical arteries but that most of the control aphasics showed similar lesions, and b) that in patients with identical lesions the production of automatisms depended on the patient's age. Result a) would support Kornhuber's theory but is also compatible with Code's (1987) assumption that a subgroup of automatisms may reflect right hemisphere language, as commissural fibres are also involved in the subcortical lesion. More important are the negative aspects of the same finding. Haas et al. point out that only 15.5 % of the variance in automatism production can be explained by parameters of lesion anatomy. Therefore a certain anatomical configuration of the lesion may be a necessary, but is definitely not a sufficient cause for automatism production. Finding b) can be interpreted along two lines: 1) the cerebral representation of language functions may change with advancing age towards further focussation which would make it more vulnerable (Brown and Jaffe, 1975), or 2) advancing age could lead, either on its own or by prolonged exposure to risk factors, to diffuse brain damage not visible neuroradiologically which contributes to automatism generation in those patients who eventually suffer a

stroke. As has been described in the introduction, diffuse brain damage alone may lead to automatism production (Leischner, 1951).

We assumed that these two hypotheses lead to different predictions concerning the accompanying neurological and neuropsychological symptoms in aphasic stroke victims with and without speech automatisms, and compared two respective groups of patients who were matched for age, sex, lesion size, and severity of aphasia (Wallesch, Haas & Blanken, in press). Conclusive evidence for the regular presence of diffuse brain damage or dysfunction in patients with automatisms was not found. This finding is supported by the study of Herrmann (1987) who described a high standard of communicative competence in some patients with predominant automatism production. The neurological and neuropsychological analysis of the Wallesch et al. (in press) study revealed the presence of more severe hemiparesis and ideomotor apraxia in patients with speech automatisms. Ideomotor apraxia is currently viewed as a disorder of the motor programming involved in the execution of actions (Poeck, 1982). Both these symptoms indicate damage to the periphery of the cerebral executive system. If one does not assume a focussation of motor functions with advancing age (which position to our knowledge has never been advanced), one would be inclined to link automatism production with a deficit rather in an output system than in central processing components, and probably rather look for its mechanisms of generation on its periphery.

#### 8. *Where Do Speech Automatisms Come From? Neurolinguistic Evidence*

Blanken, Dittmann, Haas & Wallesch (1988) attempted to delimit the functional locus of the generation of speech automatisms within a model of oral language production. Nine patients with a predominant production of automatisms of the syllabic type (e.g. "do-do-do", "weddi-weddi") were investigated. A standardised interview was carried out in order to elicit dialogical productions in a differentiated manner. The catalogue of questions consisted of 18 wh-questions, 18 yes/no questions, and 9 narrative requests. The latter consisted formally of a wh-question and an appended yes/no question with the request, "Could you please tell me?". The stimuli were offered in a pseudo-randomised sequence. The analysis of the patients' response behaviour revealed that the length of their verbal reactions was orientated towards the type of question and was not organised stereotypically. The authors conclude that form-related decisions on the type of speech-

act are possible in the case of aphasics who produce automatisms and, thus, that planning processes on a pragmatic and conceptual level of the production of speech are not organised stereotypically. A second experiment was directed at the patients' abilities in "deblocking" situations. A sub-group of aphasics could be cued into correct phonological utterances in a series completion task (number series, days of the week, months) and by syntagmatic stimuli with a missing final element (e.g. "day and ... (night)"; "he hits the nail right on the ... (head)". The authors interpret this result as evidence against the assumption of stereotypical production resulting from disturbances of the articulatory apparatus. They conclude that the generation of automatisms results from a disturbance which is functionally situated between the "higher" pragmatic-conceptual planning processes and the "lower" articulatory processes (see Fig. 1).

In a cognitive single case study, Kremin (1987) reported on Michel, whose spontaneous speech was restricted exclusively to the vowel sequence "ah-oh-oh". However, only few automatims occurred in oral naming. Here he attempted, with great effort of speech, to approach the target word syllable by syllable. Written naming was far better preserved. Moreover, on the single word level he was able to attain relatively good results in writing to dictation, reading aloud, and repetition. The experimental investigations showed word frequency effects for reading aloud, writing to dictation, and repetition; abstractness effects for writing to dictation; and non-word effects in reading aloud and writing to dictation, but not in repetition. Semantic errors had only been discernible in the initial phase of aphasia. On the background of the logogen model (cf. e.g. Morton, 1980; Morton and Patterson, 1980), Kremin assumes that the cause of this patient's automatisms is a disconnection between the semantic system and the phonological output logogen system. She attributes the superior performance in reading aloud, writing to dictation, and repetition to direct input/output logogen connections which can circumvent the cognitive and semantic system, respectively. In particular, she pursues the hypothesis that Michel used a non-semantic lexical pathway (cf. also Patterson, 1986; see Fig. 2), which, she considers, explains both the lack of semantic mistakes and of word class effects in reading and repetition and lexical effects (word versus non-word).

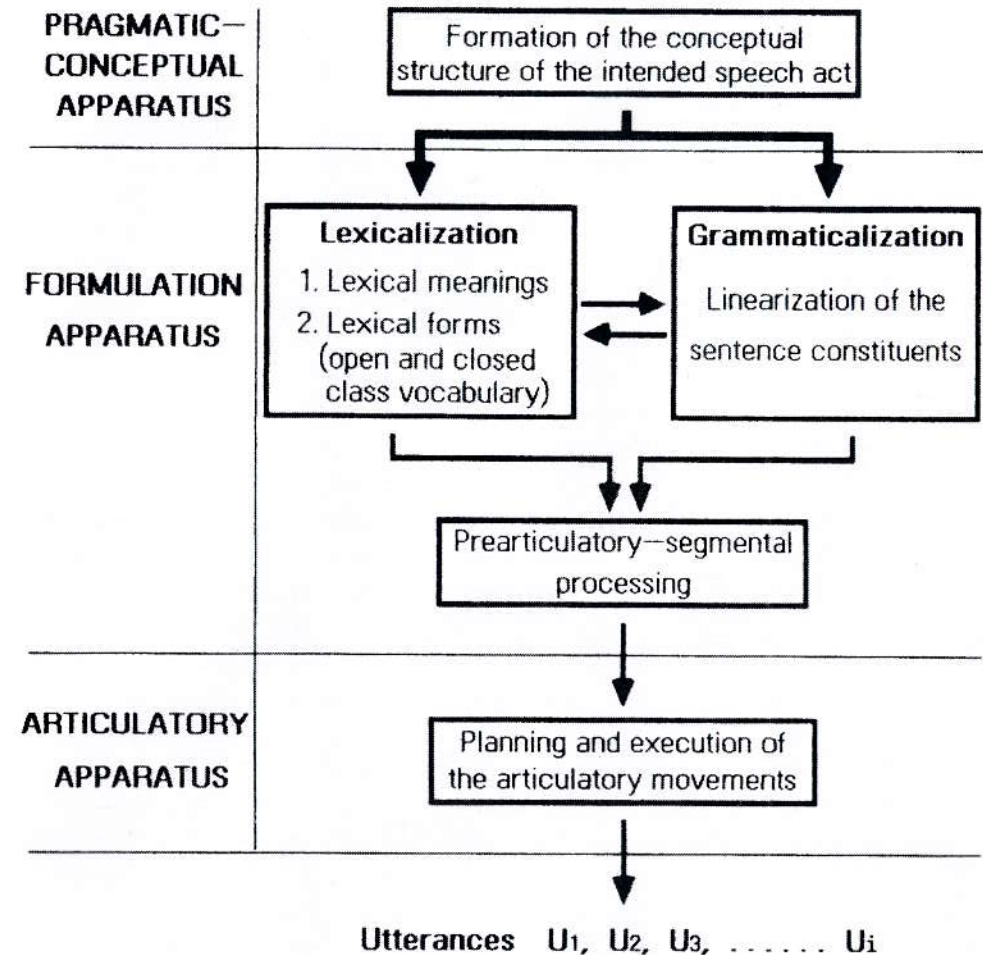


Fig. 1: Neurolinguistic working model of oral language production (from Blanken et al., 1988)

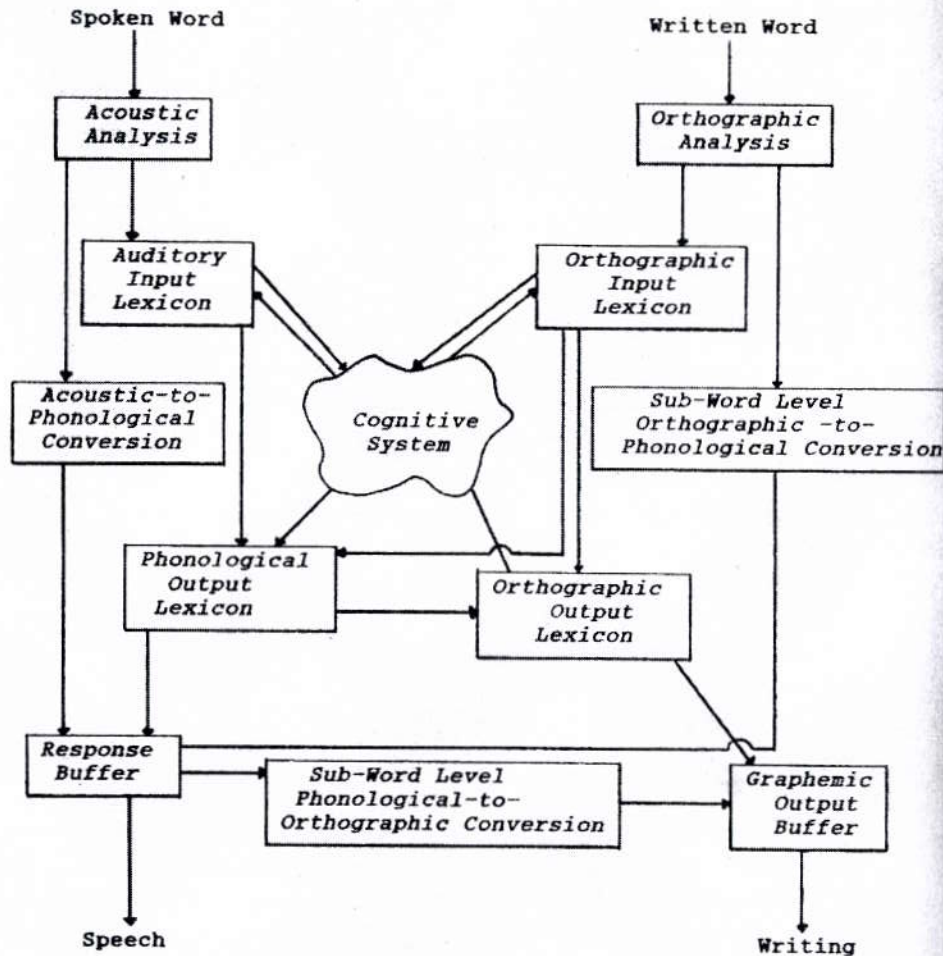


Fig. 2: Language processing model for the recognition, comprehension, and production of spoken and written words (from Patterson, 1986)

There are only few systematic studies on the written language abilities of aphasics with automatisms. Peuser (1978) documented the written language performance of an aphasic who produced automatisms and whose written production appeared to be relatively intact, with, however, agrammatical symptoms. Blanken et al. (1988) found in 9 aphasics a modality specific basis for the production of automatisms. None of these patients repeated the verbal automatisms in the production of written language and there were also no new and genuine written automatisms. It should be noted, however, that the written language abilities of most of the patients investigated were very poor and only in individual cases could partially retained written language abilities be found.

Blanken, de Langen, Dittmann & Wallesch (in press) investigated the relatively spared written language abilities of a patient with severe chronic aphasia and with no other oral output than speech automatisms in spontaneous speech, oral naming, repetition, and reading aloud. His written language production was, in contrast, far better preserved. His performance was predominantly correct on the single word level in the writing to dictation of monosyllabic and bisyllabic words. He was also frequently able to name object pictures in written language. A more exact examination of the "lexical route" revealed good performances in writing orthographically ambiguous stimuli (e.g. Tod (death), Uhr (clock), Volk (people)) and partial capacities in the writing of orthographically irregular nouns (e.g. Chef (boss), Orange (orange)). Although his performance in writing non-words was grossly off-target so that severe damage to the "phonological route" of writing must be assumed, the authors found that writing to phoneme-grapheme conversions was not wholly impossible. In the case of the non-words there were occasional correct results and frequent close approximations to the target forms, and the patient also occasionally disregarded the critical irregularities in writing irregular nouns and carried out a direct translation from sound into writing (e.g. Scout - Skaut; Cello - Schello) instead. The fact that writing to dictation according to phonological rules was possible, but not oral repetition or other types of verbal language performance, provides evidence within the framework of the logogen model or of similar models of a functional localisation of this patient's production of automatisms beneath the "phonological buffer". Apart from this, upon being presented simultaneously with 3 pictures of objects in connection with a stimulus named by the investigator, the patient was able to find the



phonologically related item. The authors evaluate this as evidence for the patient's capacity to operate his phonological lexicon (see Fig. 3).

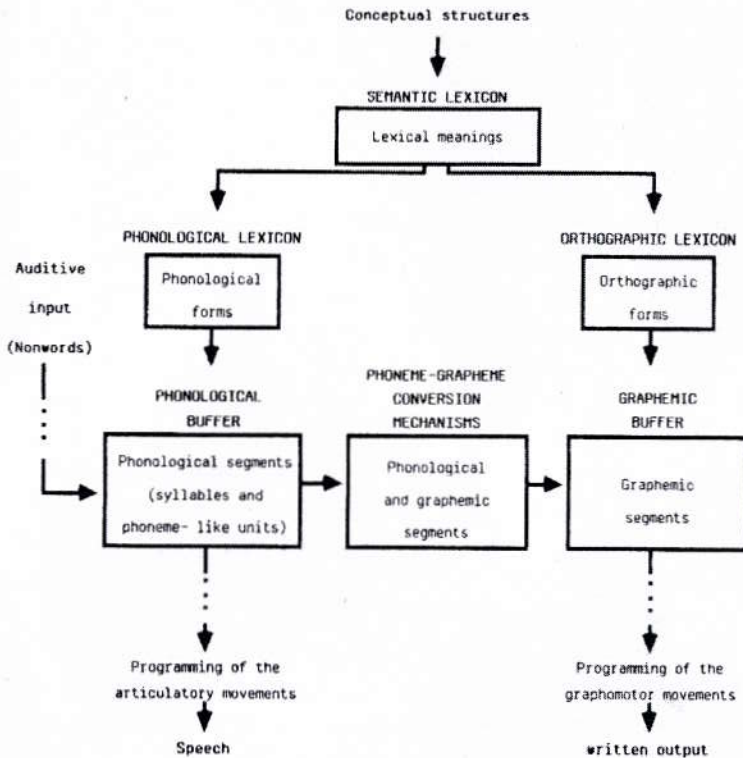


Fig. 3: Segment of a production model

The results of both case studies suggest that there may be a multiple potential basis for the generation of speech automatisms, one which is related to the (deficient) activation of lexical units and another which has to be situated near the periphery of the language

production system. This latter type could be viewed as a special form of speech apraxia. However, the more exact relationship between speech automatisms and speech apraxia has yet to be clarified.

#### 9. Perspectives on Cognitive Models of Language Processing

In this final section we shall briefly discuss the place of the research reviewed so far within the realm of cognitive neurolinguistics. This subdiscipline of linguistics aims at relating patterns of deviant language processing caused by brain injuries or lesions to models of normal language processing developed in the theoretical frameworks of cognitive psychology, psycholinguistics, or artificial intelligence research. According to this methodological paradigm we discussed hypotheses about the functional localisation of speech automatisms in the previous sections. Both models of language processing we referred to there consist of "boxes", representing components of the language processing apparatus, and of lines connecting these boxes, representing the pathways of information flow between the components. The logic of this model type implies that it is only said what these components do - processing of phonological information, e.g. - but not how they accomplish processing (i.e., their internal structure and functioning). Two proposals have been drawn up concerning the direction of information flow between the components: Garrett (cf. 1975; 1984) hypothesised a production apparatus with autonomous components connected in series, permitting one-way (top-down) flow of information only. However, both by a priori reasoning and by evidence from psycholinguistic and neurolinguistic data we have to assume feedback and therefore interactive processing between at least some of the components. It is, e.g., reasonable to postulate simultaneous and heavily interacting processing of information in the lexical and syntactic component during language production (cf. Bock, 1982; Stemberger, 1985, and others). Therefore "boxologists" models, although showing a clear preference for serial, top-down processing, have to take into account interactive connections, i.e., bidirectional information flow between (at least some of the) components (cf. Blanken, 1988).

In these models, however, the components are still drafted in a black-box. One way to obviate this disadvantage is to decompose the processing apparatus in a 'network'-like fashion. The basic ideas behind models of this type, e.g. the interactive activation model developed by McClelland & Rumelhart, 1981, are the following (cf. Dell, 1984; Ditt-

mann, 1988): The speaker's knowledge of language is represented as a network of linguistic unit-types like morphemes and phonemes, connected by activating and inhibiting links. Language processing consists of spreading activation between these units ("nodes") which guarantees information flow through all of the system, top-down as well as bottom-up. Although the units ("nodes") can be defined as constituting "components" or "levels" of processing, these components clearly do not have the property of black boxes. It is possible to analyse component-internal processes, e.g. activation of single phonemes by higher-level units like morphemes and syllables, and bottom-up activation of morphemes by way of "positive feedback" (cf. Dell, 1985) from the phoneme-level. Without discussing further details here, it should be stressed that some open problems concerning speech error data have been explained in the recent years (cf. Berg, 1985; Stemberger, 1985) and a quite elegant description of monitoring processes has been presented within this theoretical framework (cf. Berg, 1986).

Future research in cognitive neurolinguistics should take into account network modeling of language processing for several reasons. In our opinion, one important point is the possibility of describing disordered language processing in terms of partially damaged links between unit-types in the network, leading to disturbances of activation and inhibition, therefore causing insufficient or misdirected flow of information between nodes of the same level and of different levels of processing. By this means a more detailed analysis of the functional locus and structure of processing deficits seems to be possible as compared with boxologists' models, which can only describe the respective deficiencies in global terms, that is as "malfunctioning" of whole components or as interruption of connections between whole components.

## REFERENCES

- Alajouanine, T. 1956. Verbal realization in aphasia. *Brain* 79.1-28.
- Berg, T. 1985. Is voice a suprasegmental? *Linguistics* 23.883-915.
- Berg, T. 1986. The problems of language control: Editing, monitoring, and feedback. *Psychological Research* 48.133-144.
- Blanken, G. 1988. Zur Ausgrenzbarkeit der linguistischen Formulierungsprozesse. *Neurolinguistische Evidenzen*. G. Blanken, J. Dittmann and C.-W. Walleesch (eds.), *Sprachproduktionsmodelle. Neuro- und psycholinguistische Theorien zur menschlichen Spracherzeugung*. Freiburg.
- Blanken, G. - De Langen, E.G. - Dittmann, J. - Walleesch, C.-W. (in press) Implications of preserved written language abilities for the functional basis of speech automatism (recurring utterances): A single case study. *Cognitive Neuropsychology*.
- Blanken, G. - Dittmann, J. - Haas, J.-C. - Johannsen-Horbach, H. - Walleesch, C.-W. 1986. Sprachautomatismen im Sprachproduktionsmodell. *Arbeitsberichte der Linguistischen Sozietät zu Freiburg* 2.32-65.
- Blanken, G. - Dittmann, J. - Haas, J.C. - Walleesch, C.-W. 1987. Varietäten linguistischer Leistungen bei Aphasikern mit Sprachautomatismen. Poster. 14. Jahrestagung der Arbeitsgemeinschaft für Aphasierecherche und -behandlung, München.
- Blanken, G. - Dittmann, J. - Haas, J.C. - Walleesch, C.-W. 1988. Producing speech automatism (recurring utterances): Looking for what is left. *Aphasiology* 2.545-556.
- Bock, K. 1982. Toward a cognitive psychology of syntax: Information processing contributions to sentence formulation. *Psychological Review* 89/1.1-47.
- Broca, P. 1861. Remarques sur le siège de la faculté du langage articulé, suivies d'une observation d'aphémie (perte de la parole). *Bulletin de la Société Anatomique de Paris* 36.330-357.
- Brown, J.W. - Jaffee, J. 1975. Hypothesis on cerebral dominance. *Neuropsychologia* 13.107-110.
- Brunner, R.J. - Kornhuber, H.H. - Seemüller, E. - Suger, G. - Walleesch, C.-W. 1982. Basal ganglia participation in language pathology. *Brain and Language* 16.281-299.
- Code, C. 1982. Neurolinguistic analysis of recurrent utterance in aphasia. *Cortex* 18.141-152.
- Code, C. 1987. *Language, Aphasia, and the Right Hemisphere*. London.
- De Bleser, R. - Poeck, K. 1985. Analyses of prosody in the spontaneous speech of patients with CV-recurring utterances. *Cortex* 21.405-416.
- Dell, G.S. 1984. Representation of serial order in speech: Evidence from the repeated phoneme effect in speech errors. *Journal of Experimental Psychology: Learning, Memory, and Cognition* 10.222-233.
- Dell, G.S. 1985. Positive feedback in hierarchical connectionist models: Applications to language production. *Cognitive Science* 9:3-23.
- Dittmann, J. 1988. *Versprecher und Sprachproduktion*. G. Blanken - J. Dittmann and C.-W. Walleesch (eds.), *Sprachproduktionsmodelle. Neuro- und psycholinguistische Modelle der menschlichen Sprachproduktion*. Freiburg i. Br.
- Garrett, M.F. 1975. The analysis of sentence production. G. Bower (ed.), *The Psychology of Learning and Motivation: Advances in Research and Theory*. Vol. 9. New York.
- Garrett, M. F. 1984. The organization of processing structure for language production. Applications to aphasic speech. D. Caplan, A.R. Lecours and A. Smith (eds.), *Biological Perspectives on Language*. Cambridge, Mass.
- Haas, J.-C. - Blanken, G. - Mezger, G. - Walleesch, C.-W. 1988. Is there an anatomical basis for the production of speech automatism? *Aphasiology* 2.557-565.
- Herrmann, M. 1988. *Psychosoziale Veränderungen und kommunikative Fertigkeiten bei chronischer schwerer Aphasie*. Phil. Diss. Freiburg i. Br.
- Huber, W. - Poeck, K. Weniger, D. 1982. *Aphasie*. K. Poeck (ed.) *Klinische Neuropsychologie*. Stuttgart.
- Jackson, J.H. 1874. On the nature of the duality of the brain. Reprinted in: J.Taylor (ed.), *Selected writings of John Hughlings Jackson*. Vol. 2. New York 1958.
- Jackson, J.H. 1879. On affections of speech from diseases of the brain. Reprinted in: J.Taylor (ed.), *Selected writings of John Hughlings Jackson*. Vol. 2. New York 1958.
- Kornhuber, H.H. 1977. A reconsideration of the cortical and subcortical mechanisms involved in speech and aphasia. J.E. Desmedt (ed.) *Language and hemispheric specialization in man: Cerebral ERPs*. Basel.
- Kremin, H. 1987. Is there more than ah-oh-oh? Alternative strategies for writing and repeating lexically. M. Coltheart - G. Sartori and R. Job (eds.), *The Cognitive Neuropsychology of Language*. London.
- Lebrun, Y. 1986. Aphasia with recurrent utterance: A review. *British Journal of Disorders of Communication* 21.3-10.
- Leischner, A. 1951. Über den Verfall der menschlichen Sprache. *Archiv für Psychiatrie und Zeitschrift Neurologie* 187.250-267.

- McClelland, J.L. - Rumelhart, D.E. 1981. An interactive activation model of context effects in letter perception. Part I: An account of basic findings. *Psychological Review* 88.375-407.
- Morton, J. 1980. The logogen model and orthographic structure. U. Frith (ed.), *Cognitive Processes in Spelling*. London.
- Morton, J. - Patterson, K.E. 1980. A new attempt at an interpretation, or, an attempt at a new interpretation. M. Coltheart - K.E. Patterson - J. Marshall (eds.), *Deep Dyslexia*. London.
- Patterson, K.E. 1986. Lexical but nonsemantic spelling? *Cognitive Neuropsychology* 3.341-367.
- Peuser, G. 1978. *Aphasie*. München.
- Poeck, K. 1982. *Apraxie*. K. Poeck (ed.), *Klinische Neuropsychologie*. Stuttgart.
- Poeck, K. - De Bleser, R. - von Keyserlingk, D. 1984. Neurolinguistic status and localization of lesion in aphasic patients with exclusively consonant-vowel recurring utterances. *Brain* 107.199-217.
- Stemberger, J.P. 1985. An interactive activation model of language production. A.W. Ellis (ed.) *Progress in the Psychology of Language*. Vol. 1. London.
- Wallesch, C.-W. - Haas, J.C. - Blanken, G. (in press). On the neurological status of speech automatisms and its significance for neurolinguistic models. *Aphasiology*.
- Wallesch, C.-W. - Papagno, C. 1988. Subcortical aphasia. F.C. Rose - M.A. Wyke - R. Whurr (eds.), *Aphasia*. London.

## MULTILINGUALISMUS UND SPRACHLICHE STÖRUNGEN Physiologische, aphasiologische und linguistische Aspekte\*

BELA BROGYANYI

Freiburg i. Br.

### 0. Vorbemerkung

Ausgehend von einem Selbstzeugnis eines polyglotten Sprechers sollen in diesem Beitrag Probleme des Multilingualismus in Zusammenhang mit neurophysiologischen und aphasiologischen Aspekten behandelt werden. Besonders beziehe ich mich dabei auf Störungen der Sprache von gesunden Mehrsprachigen. Es wird dabei auch notwendig sein, auf definitorische Fragen des Bi- und Multilingualismus und seines Grades einzugehen.

### 1.0. Mehrsprachigkeit und sprachliche Störungen: Ein typischer Fall

Im folgenden soll hier ein interessanter Fall eines polyglotten Sprechers angeführt werden.

In der amerikanischen Zeitschrift *Maine Times* (3. April 1981, 2 ff.) wurde über Persönlichkeiten aus dem Bundesstaat Maine berichtet, die als außergewöhnliche Talente bezeichnet wurden. In diesem Zusammenhang wurde ein Bürger vorgestellt, der durch seine Mehrsprachigkeit hervorragte. Im Interview sprach er über seine Situation als Mehrsprachiger und berichtete auch über Störungen seiner Sprachen, die durch diesen Umstand hervorgerufen wurden.

Der Interviewte unterrichtete an einem College des Landes als Professor für Romanistik. Er sprach folgende fünf Sprachen (in der Reihenfolge des Erwerbs): 1. Ungarisch, 2. Deutsch, 3. Englisch, 4. Französisch, 5. Italienisch. Alle diese fünf Sprachen