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Major Review

Old solutions to new problems: a contribution to today's relevance of Carl Wernicke's theory of aphasia

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Abstract

This article discusses Wernicke's theory of the cognitive architecture of language and relates it to models used in contemporary neurolinguistics. In addition, Wernicke's view of a theory-guided patient classification is discussed. Special emphasis is placed on his theory of speech production that can be understood to a large extent as a theory of 'conscious' and 'unconscious' correction of speech output. Finally, similarities between Wernicke's theory and current models of language production and speech monitoring are pointed out.

Introduction

Carl Wernicke is considered to be a forerunner and anticipator of modern aphasiology. Even when one is aware of Wernicke's importance for modern aphasiology, the level of his theoretical insight and his clarity of clinical observation in his 1874 study 'The symptom-complex of aphasia' is amazing. From today's perspective, one starts to understand why the high level of aphasiology research in the last quarter of the past century could only develop out of the fruitful synthesis of both theory and observation. Wernicke himself was probably one of the first aphasiologists who recognized the relatedness of both aspects so sharply. This is reflected in the fact that he did not draw on any of the clinical descriptions of aphasic patients existing at the time; rather he judged them as 'doubtful material'. Instead, he relied on cases whose symptoms and development he documented on the basis of his own observations, 'because a precise examination particularly of psychological symptom-complexes cannot be devoid of an already completed theory which prescribes its direction'¹ (p. 38). Indeed, Wernicke's theory of the aphasias, as set out in his 'psychological study on an anatomical basis' (the subtitle of his work which comprises only 72 pages) was revolutionary; it brought about a radically new approach to aphasic symptoms and their configurations.

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In which respects was Wernicke's theory so novel that to his mind the previous descriptions on aphasic patients had to be completely reclassified and reinterpreted? What was entirely new, and looking back, also very successful, was his combination of assumptions about the loci of neuroanatomical brain lesions with a complex and explicit theory of psycholinguistic language processing. This step towards a functional, cognitive model of language processing was stimulated by Wernicke's conviction that Broca's theory of a sole centre for the human language ability (cf. Broca, 1861) was wrong. Rather, he proposed that one had to distinguish two anatomically and functionally distinct language centres, a motor and a sensory centre. According to Wernicke, all aphasic symptoms could be considered as disorders between these two extremes.

From today's viewpoint, what turns out to be even more interesting than his two-centre-theory and its relevance for the science of neuroanatomy, are the neurolinguistic and clinical implications of his functional theory of language processing, in particular, for the description and categorization of aphasic symptoms. Besides other forms of aphasia, he described sensory aphasia, as it was called then (later termed Wernicke's aphasia), which is marked by impaired auditory speech comprehension and fluent, but paraphasic speech production. Sensory aphasia was considered by Wernicke to be caused by a lesion of the sensory speech centre. Another symptom-complex, conduction aphasia (a term which Wernicke himself had used), could also have been named after Wernicke, since he provided an equally thorough and precise description as for sensory aphasia, and assumed it to be due to the injury of the association fibres which connect the two speech centres.²

The vigorous position Wernicke advanced on the relation between thinking and speaking is still heavily disputed today. He says: 'Thinking and speaking are two completely independent processes which may even impede one another' (p. 33). According to Wernicke, aphasic disorders are not necessarily accompanied by thinking disorders, and the latter are well compatible with a retained 'formal language ability'. Also in this respect, Wernicke had anticipated much of what was later described in detail about cases with so-called 'isolation of the speech area' (see Whitaker 1976).

The psycholinguistic model by Carl Wernicke

As stated above, Wernicke postulated two functionally distinct centres of language processing which he assumed to be situated at different loci in the cortex. The centres were considered to be connected to each other by a pathway or conduction (anatomically realized by association fibres), allowing an exchange of information between the two systems. The motor speech centre comprises mental representations about the movements necessary for producing speech, so-called 'motor images'. The sensory speech centre encompasses mental representations about the sounds of words involved in the perception of speech, called 'sound images'.³ These linguistic representations are separated by Wernicke from so-called 'images of visual and tactual sensations' which are necessary for concept formation and do not belong to the realm of language, but to that of thought ('intelligence', to use Wernicke's term). Wernicke's proposal on the architecture of the human language system is illustrated schematically in Figure 1.

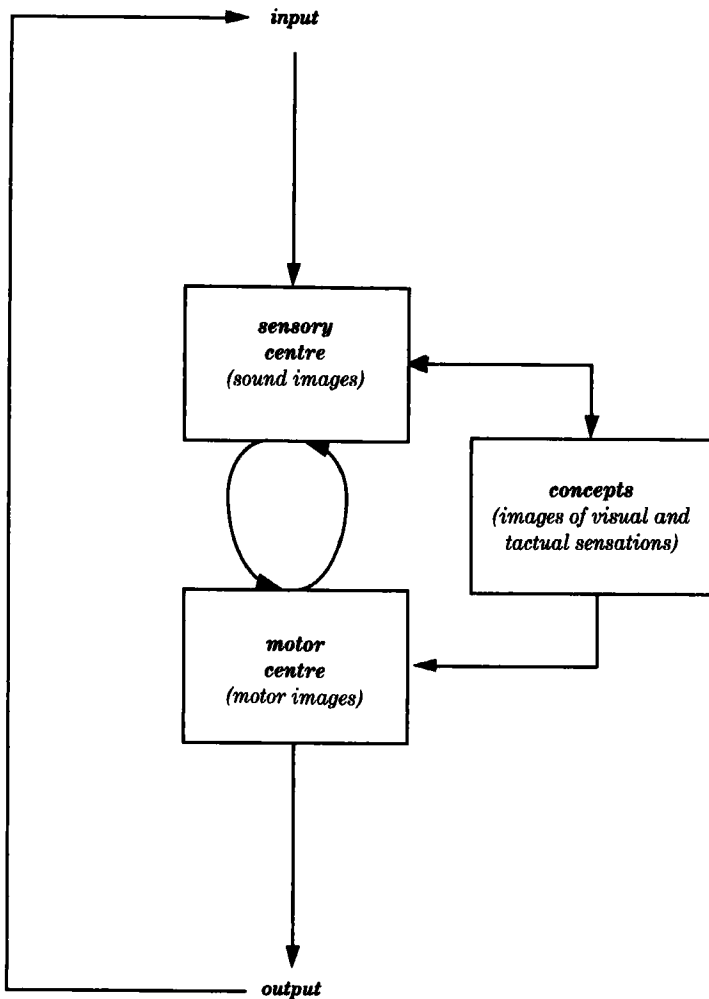


Figure 1. Wernicke's psycholinguistic model (without written language) including unconscious and conscious correction.

Wernicke's methodological procedure in developing his model followed that of other researchers at his time, e.g. Broca's; namely, to exploit language deficits in aphasia, or more specifically, particular patterns of aphasic breakdowns, as a source of evidence for postulating a certain functional system in healthy brains. The circumscribed pattern of breakdown was thought to correspond to a distinct language function in the unimpaired condition. Wernicke, however, went beyond Broca by demonstrating that a double dissociation of certain language functions may occur. Indeed, his observation that sensory and motor speech abilities can be doubly dissociated formed the basis of Wernicke's model.

A comparison between Wernicke's model and modern models of single word processing, such as the Logogen model (see Figure 2), reveals several similarities. Figure 2 shows the relevant part of the Logogen model (here, according to Patterson and Shewell 1987). It is easy to see that the architecture of the language processing system is similar to that of the Wernicke model. Both models graphically

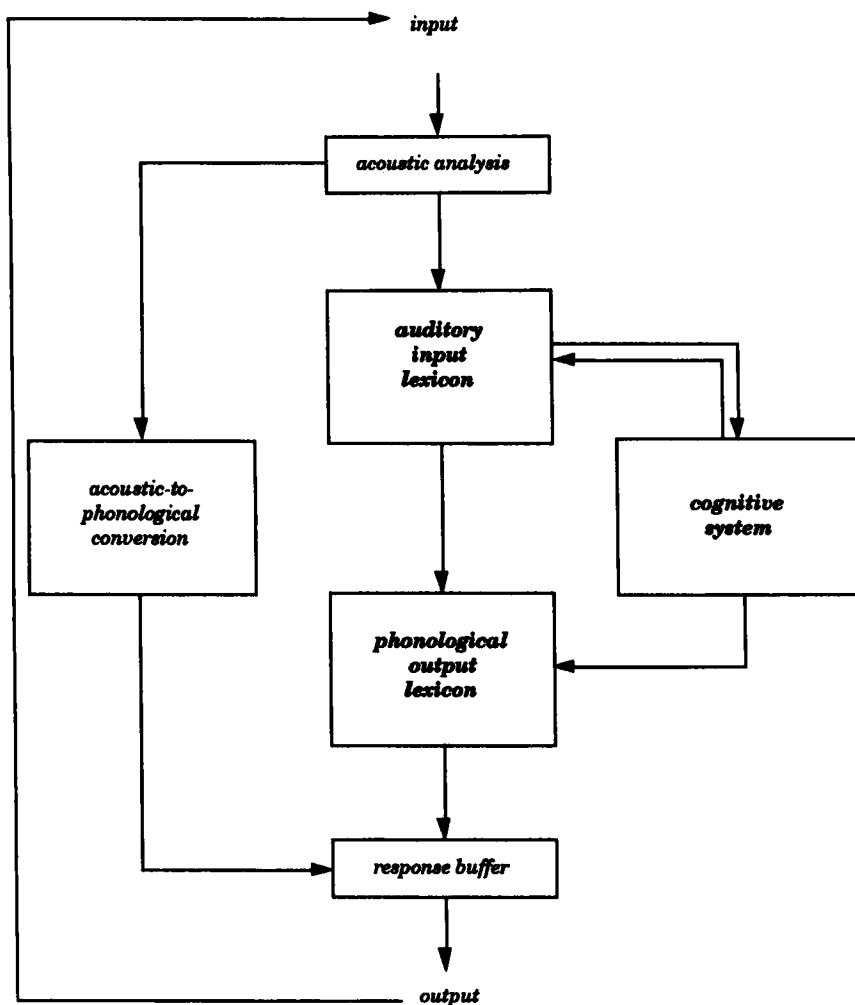


Figure 2. The logogen model (without written language) (according to Patterson and Shewell, 1987).

depict separate language components for linguistic input and output information. Both models make an attempt at distinguishing the processing of meaning from that of form, reflected in the postulation of a separate cognitive and/or conceptual system, respectively.

But there are also many differences between the two models. The most important ones are the following: Wernicke did not know of prelexical ('acoustic analysis') and postlexical ('response buffer') processing devices. To Wernicke, the crucial-linguistic units for speaking were the word or the syllable (cf. Wernicke, 1874, p. 14) and the sentence (and for writing, the letter). Smaller linguistic units, e.g. phonemes, were not yet known to him in a theoretically precise sense, which is why Wernicke could not postulate a separate non-lexical route for the repetition of words (via 'acoustic to phonological conversion'). The assumption that such a non-lexical route exists, however, is not shared by all modern psycholinguists and neurolinguists (see e.g. Kohn 1993). Second, Wernicke's model does not

furnish a locus for representations of verbal meaning. Concepts would only be linked to visual and tactual sensations. Obviously, Wernicke did not see much of a need to explain semantic errors in aphasia, and accordingly, to postulate a system of semantic representations, whereas the 'cognitive system' in Figure 2 is supposed to contain verbal meanings, among other cognitive representations. But how semantic representations are organized in detail, and how verbal and non-verbal semantic representations are related to each other, are issues that are still being hotly disputed (cf. Job and Sartori 1988). Third, there are significant differences between the two models as far as the involvement of the knowledge stores in language processing and the processing characteristics themselves are concerned. These aspects will be further described below.

Aphasia diagnosis in Wernicke's model

Wernicke was a neurologist and not a psycholinguist. Thus, he was mainly interested in applying his theory to the diagnosis of his own patients. In his 1874 study he discusses 10 cases on the background of his model, all of which he had examined himself in the 'Allerheiligen Hospital' in Breslau. It is surprising that Wernicke was already able to distinguish all diagnostic categories for aphasic disorders which still seem relevant to us today.

As a counterpart to motor aphasia going back to Broca's description (and now referred to as Broca's aphasia), which Wernicke attributed to an impairment of the motor images, Wernicke postulated a sensory aphasia, now referred to as Wernicke's aphasia. He assumed this latter form of aphasia to be caused by an impairment of the (sensory) center for sound images. The symptoms of a (severe) sensory aphasia were characterized by Wernicke as follows: 'The patient is . . . neither able to repeat the spoken word, . . . nor to comprehend it. All the patient perceives of what is spoken to him is an indistinguishable noise which does not make any sense to him' (p. 22).

Furthermore, Wernicke introduced the so-called conduction aphasia, which he thought to be caused by a disruption of the pathway (or conduction)⁴ between the sound and the motor images, whereby the sensory and the motor centre themselves may be spared. He emphasized that these patients can be characterized by preserved speech comprehension ('The patient understands everything' (Wernicke, 1874, p. 26)), by certain problems in speech output, namely, paraphasias and word-finding difficulties, as well as by their ability to correct themselves ('The patient knows, if he is paying attention, that he has spoken incorrectly and gets upset about it' (1874, p. 26)). Although Wernicke draws attention to the 'crucial task' (1874, p. 22) the pathway between the two language centres fulfils, namely, the repetition of words, he does *not* assume that a repetition disorder may form the predominant symptom of conduction aphasia. Wernicke's view, which in modern aphasiology is also shared by Kohn (1992), is contrary to how conduction aphasia has been viewed by many subsequent authors. Only later, Wernicke (1906, p. 230 f.; quoted from Eggert, 1977), like most of the subsequent researchers, defined the inability to 'mimic', that is, a repetition disorder, as one of the leading symptoms of conduction aphasia, the others still being paraphasias, word-finding difficulties and preserved speech comprehension. Despite this definition of predominant symptoms, Wernicke (1906, p. 230) still hesitated to posit 'a unified empirically-based clinical picture' of conduction aphasia.⁵

Wernicke also knew about another form of aphasia, today commonly referred to as anomic or amnesic aphasia. However, he regarded it as a mild form of conduction aphasia: 'In milder cases, one has to be content with the assumption of an impaired conduction due to poor blood circulation. These cases do not exhibit hemiplegia. What stands out as a striking feature in this disorder is that rather than confusing words the patients have much more trouble in finding the right word. The patient's speech is then full of pauses, in which he obviously struggles to express himself' (p. 27). In sum, Wernicke distinguishes anomic and conduction aphasia by two criteria: (1) (the lack of) hemiplegia, and (2) word-finding problems versus confusion of words.⁶

Moreover, Wernicke had already described a case of transcortical aphasia, as it was called later by Lichtheim (1885), and more specifically, transcortical motor aphasia: 'He comprehends all simpler sentences within his intellectual range, he correctly responds yes and no, and does each task correctly. In spontaneous speech, however, he can only utter yes and no . . . Still, he can repeat everything correctly' (p. 60). Wernicke regarded this kind of aphasia as a complement of the classical form of conduction aphasia due to a disruption of the pathway between the (non-verbal) concepts and the motor images.

Furthermore, Wernicke was familiar with the picture of global aphasia with recurring utterances (see his Case 8).

It has to be added that Wernicke not only documented (wherever possible) alectic and agraphic disorders in his patients, but also integrated the processing of written language into his model (see de Bleser in this volume for more information on written language).

Sensory and conduction aphasia: errors in speech production

The following discusses the two types of aphasic patients which are clearly foremost in Wernicke's 1874 investigation: patients with sensory and conduction aphasia. Both kinds of aphasia are not caused by impairments of the centre for motor images. Nevertheless, Wernicke describes a number of symptoms to be observed in the speech output of these patients. Tables 1 and 2 present the most important observations in the case of Adam (sensory aphasia) and in the case of Bechmann (conduction aphasia), respectively. Both patients are among those who have been documented by Wernicke in detail. The dates of examination indicate that both cases must have still been very vivid in Wernicke's mind when he completed his study in 1874.

Table 1. The case of Susanne Adam (sensory aphasia; Wernicke's Case 1)

A summary of the most important linguistic symptoms as described by Wernicke (pp. 39 ff.) upon four dates of examination

Widow of a worker, 59 years old, disease onset: 1 March 1874

Date of examination: 7 March 1874

Auditory speech comprehension

'She understands absolutely nothing of what is spoken to her.'

Oral speech production

'The sentences she produces are often incorrect in that they contain meaningless or distorted words.'

'Very often, especially in emotional states, she succeeds in producing entirely correct sentences.'
 'At some times, she is able to name the objects presented to her correctly . . . , at other times she no longer knows the names of the same objects.'
 'Through the day, when she is with other patients and is not under any pressure, she correctly names most of the objects. Thus, one almost has to assume that an unlimited vocabulary is at her disposal.'

Writing

' . . . she only writes basic lines and hairlines.'

Reading

'There is complete alexia, digits are also not understood correctly.'

Date of examination: 15 March 1874

'Her general state has rapidly improved.'
 'But the overall picture was still typical: she spontaneously used most of the words she produced correctly, whereas she comprehended only very few, and if so, only with great difficulty.'

Date of examination: 25 March 1874

'further progress'
 ' . . . it was striking that in her first attempts, she incorrectly repeated the words that were repeatedly presented to her, but in the end, she could repeat them correctly, and she was also able to correct herself spontaneously on many occasions.'

Date of examination: 20 April 1874

Auditory comprehension

'She has made further progress, comprehends almost everything that is repeatedly spoken to her.'

Oral speech production

'Her speech is still filled with pauses, but is almost correct.'

Writing

'Agraphia is . . . her most strikingly impaired language ability.'

Reading

' . . . she reads fluently.'

Table 2. The case of Beckman (conduction aphasia; Wernicke's Case 3)

A summary of the most important linguistic symptoms, as described by Wernicke (pp. 47 ff.); Wernicke noted only the first date of examination

Pharmacist, 64 years old, disease onset: 15 March 1874

Date of examination: 20 March 1874

Auditory speech comprehension

'He comprehends everything precisely, and always responds correctly to suggestive questions.'

Oral speech production

'There are no signs of motor aphasia, since his vocabulary is unlimited.'

Word-finding difficulties

' . . . he lacks the words for many objects he wants to name; he tries hard to find them . . . '

Repetition

'He can repeat a word named to him without hesitation.'

Commenting aloud to oneself (or speaking aside)|overlearned material

'He is quite good at speaking when making asides, especially when they consist of idioms and common sayings.'

Self-corrections/corrections by others

'At times, he reaches a difficult word, he gets stuck, struggles, even gets angry, and almost every word he eventually utters, which is interrupted then by many pauses, is meaningless; he corrects himself again and again, and the greater his effort is, the worse it gets. If one guesses what he intended to say and tells him that, he heaves a sigh of relief: "Yes, that's what I wanted to say." . . . Very often he asks something about what he has uttered right before: Was that correct?'

' . . . and sometimes it happens, if he gets carried away that whole sentences which are grammatically correct just slip out of him, but their meaning differs completely from what he intended to say; in this case . . . he disavows the sentence he has just uttered.'

Written language

' . . . His alexia and agraphia are of different severity.'

Reading of single letters

'A capital (printed) letter is presented to him, and he is to name it. Despite great effort, he cannot identify it.'

Identification of single letters

'Among a number of letters . . . written for him he finds the one asked for, slowly, but always correctly; and he usually notices, if the letter (that has been asked for) is not among the presented ones. He also recognizes each letter, when letters are pronounced for him.'

Reading of words

'[Beckmann's] glance falls upon a book whose title is printed in golden letters, he points at the title and says: That I can recognize, that means "Goethe". Right next to Goethe, there is another book, one by Schiller; he is asked to read the title, but despite all efforts he does not succeed; only by comparing both titles does he assert that the latter is not a book by Goethe. He also reads the street signs in passing, without particularly searching for the words; but if he is presented a certain word, a certain letter, he never succeeds in finding it.'

Writing

'Agraphia indeed exists.'

' . . . he is unable to write without any help.'

'He can hardly write any letter successfully, and despite all his effort he only produces basic and hairlines.'

Copying

' . . . with some effort, he is not able to copy single letters or entire words he cannot name.'

Further examinations (without exact dates):

Severity/Progress of illness

' . . . the severity of his aphasia shows great variation He was much less aphasic with his relatives than with persons he did not know well. His aphasia was always most severe in doctoral examinations.'

Word finding difficulties

' . . . that only nouns, and among these, particularly names of people and places were occasionally missing.'

Reading

'The examination in May of the same year [1874] revealed significant progress. The patient now showed the interesting symptom that he correctly read whole words, such as his own name, and those of his relatives, but he could not read the individual letters of those names.'

Last examination (after May 1874) (Wernicke, 1874, p. 72):

'[the patient] is now able to write without any problems, the only residual symptom is severe alexia.'

As Tables 1 and 2 show, Wernicke keenly recognized which kinds of problems sensory and conduction aphasics might exhibit in speech production. As an example, he mentioned 'the confusion and distortion of words' (p. 44), errors

were also referred to as 'paraphasias'. He also diagnosed word-finding difficulties, a disorder which in his view should occur primarily in mild cases of conduction aphasia. Wernicke furthermore seems to have made observations about differences in the availability of particular grammatical categories. Thus, he drew attention to forms of sensory aphasia 'where not only the sound images of concrete objects and actions, but also the words necessary for conjoining clauses ["Bindewörter", to use Wernicke's term] have been lost' (p. 24), and spoke of 'curiosities in the field of aphasia . . . , e.g. the selective loss of nouns, or verbs, and so on' (p. 69).

The amount of words available to the patient was regarded by Wernicke as a characteristic feature of both sensory and conduction aphasia. In both forms of aphasia there is an abundance of words which can be spoken, whereas in motor aphasia only few simple words are preserved. Despite the above-mentioned possible (word-) category-specific differences in speech output (or within their-limits) Wernicke underlined a general feature of the speech production of sensory and conduction aphasics, namely, the high degree of fluctuation in their speech output: 'The same words are once produced correctly, once incorrectly, and no regularity can be observed. A definite vocabulary, one which comprises only correctly used words, does not exist' (p. 24). Wernicke also calls this huge vocabulary which can be used only unreliably in both types of aphasic patients, the 'virtual vocabulary'.

Although Wernicke did not explicitly state whether such variables as word frequency or word familiarity could influence the error pattern of aphasic speech, he was definitely aware of them, and may even have considered them as self-evident, as suggested in part I of his study.⁷ Wernicke refers to a further important variable which influences aphasic speech production, namely, the affective state of a patient. In highly affective situations, aphasics produce correct utterances much more often than in emotionally neutral situations.

Wernicke thus had to explain errors in linguistic output in patients who have either suffered from a lesion of the sensory centre or of the pathway between both linguistic centres, but whose motor centre was regarded as virtually unimpaired. Wernicke's solution to this problem is of great interest to us, in as much as it closely resembles recent approaches in modern psycho- and neuro-linguistics. In the following, we will focus on Wernicke's assumptions about the process of speech production, and about the cause of paraphasias in fluent aphasias. In our view this concerns issues that have hardly received any attention so far. Despite the fact that the architectures of the models in Figures 1 and 2 appear to be similar at first glance, it will also become clear that there are significant differences as to how speech production is conceived of by proponents of the Logogen model and by Carl Wernicke.

'Correction' as a constitutive part of speech production

Wernicke embedded his view on speech production into a theory on how the ability to speak is acquired. His basic idea can be sketched as follows: the child learns to speak by repeating words, that is, by using the pathway between the sound and the motor images. Thus, at first the acquisition of speech is guided by the correct reproduction of words. Only at a later stage is speaking governed by concepts; then, a direct connection between concepts and motor images is established, serving the purpose of (e.g. communicative and intentional) speech

production. Wernicke's position is that the influence of the sound images on the motor images, that is, the influence of the sensory centre on the process of speech production, retains its constitutive role even in adult speech. In highly affective states, however, only little influence is exerted, there 'the [direct] expulsion in speech movements is favored when no other associated memory images interfere' (Wernicke, 1874, p. 24).

On the grounds of this position, it could indeed be expected that any impairments of the sensory centre would lead to disorders in speech production. This does not mean that a sensory impairment necessarily yields limited motoric abilities, but rather it leads to a lack of control in the selection of words and an uncertainty as to the sounds of the words. The missing corrective function performed by the sound images would inevitably eventuate processing errors in the motor centre which is being left on its own, either due to an impairment of the sensory centre, or due to a disruption of the pathway from the sensory to the motor centre. In order to describe the impact of the sensory on the motor centre, Wernicke uses the term 'Correctur' ('correction'). He says: 'Since in usual speech—as is easily recognized from how speaking is learned—the sound image always seems to be *unconsciously* [our emphasis] innervated, and to—so to speak—hallucinate simultaneously, thereby having a constant corrective influence on the succession of the motor images' (p. 23).

Thus, output disorders are correction disorders. Indeed, Wernicke's explanation of the error patterns in the speech output of conduction and sensory aphasics is exclusively based on this assumption of a correction deficit⁸ (cf. Wernicke, 1874, p. 23).

Unfortunately, Wernicke does not offer more information about the exact nature of unconscious correction, he only says that 'the mere existence of the pathway a**1**b [from the sensory to the motor centre], without its being innervated intentionally, suffices to ensure the selection of the correct motor image' (p. 23). Wernicke assumed that the pathway between the sensory and the motor centre, being crucial to the child's language acquisition process, loses importance in adult intentional speaking. There, the directer, or as Wernicke says, the 'shorter' (Wernicke, 1874, p. 23) path from the concepts to the motor centre is taken. That is, when adults produce speech, the sound images are not intentionally activated. Obviously, Wernicke conceived of both speech centres and their respective representations as being intrinsically related to each other such that the sensory component is always automatically (co-)activated in the process of speech production.

Thus, according to Wernicke, a single lexicon exists which comprises complete phonological representations; namely, the store for sound images. In contrast to the logogen model, the centre for motor images in Wernicke's model is not an autonomous output lexicon, but depends strongly on the synergetic support by the sensory system. In this respect the motor centre is more like an epiphenomenon of the sensory centre. Nevertheless, in the production process the motor centre is activated directly by the centre for concepts, whereas the sensory centre is merely automatically (co-)activated. Obviously, Wernicke did not favour the possibility that a sound image is first selected from a sole lexicon, namely, the sensory centre, and is then passed on to the motor centre. Instead, he argued for the solution that a constant information exchange between the sound system and the system of the motor images is possible. Put in modern terms, Wernicke's

notion can be best expressed as an interactive or a two-way connection. That is, the pathway between the two speech centres permits an information exchange in both directions, an idea that comes up again when he discusses the connection between the concepts and the sensory centre (cf. below). In other words, as soon as the output (motor) centre has become activated via conceptual information, a connection to the sensory system is immediately established, and this latter system, in turn, feeds back activation to and thereby stabilizes the motor images. According to this interpretation, correct speech production is ensured by this feedback loop between the output and the input centre.

Still another aspect, which is closely related to correction, as we will see, of Wernicke's theory of speech production arouses interest, namely, the notion of a possible mechanism of addition of activation (or 'innervation', to use Wernicke's term) in the motor centre. If e.g. an object is to be named, both the tactual image (c) and the visual image (d) of the object exert influence on the motor centre (b). Furthermore, motor information is activated and stabilized by the sensory centre (which Wernicke refers to as 'a') via feedback. Thus, there are three sources of activation. In unimpaired speech—according to Wernicke—'the sum of $d + c + a_1$ acts in combination, and each contributes with the same intensity, finally resulting in the correct selection of the word' (p. 23). If the sensory centre is lesioned, however, the case is different: 'If a_1 does not function, then only the sum of $d + c$ has an innervating effect on the motor centre, the powerful influence of the pathway a_1b ceases' (p. 23). Conduction aphasia is caused by a different lesion, but the consequences are similar: 'Though the sound image is retained, it is also innervated by the remaining sensational images [visual and tactual sensations] which make up the concept. However, since the pathway a_1b [between the sound and the motor centre] is disrupted, the sound image cannot bear its weight to tip the scales in favour of the correct choice of the motor images, or at least its impact will have much less intensity' (Wernicke, 1874, p. 26).⁹

Wernicke indeed proposes the principle of local summation of activation being fed by different sources. This principle is not only similar to the threshold hypothesis (in the logogen model), but also—because it embraces different sources of activation—to recent theoretical developments of modular accounts in cognitive neurolinguistics, in particular, to the 'summation hypothesis', as put forward by Hillis and Caramazza (1991). Moreover, Wernicke's notion is well compatible with assumptions proposed in the context of interactive network theories.

To Wernicke, a feedback loop—as described above—is not necessary in emotionally produced speech, since there the motoric activities would be expelled in a most direct and fast way. Wernicke takes this assumption that the production system could—under emotional conditions—function independently from the input system as an explanation for why the speech of sensory and conduction aphasics show much fewer errors when being in an emotional state. It is important to note that Wernicke advances a psycholinguistic and not a neuroanatomic (e.g. hemisphere-specific) position to explain his observations in aphasic speech production under emotional conditions.

In Wernicke's correction theory, errors in speech production are viewed as being dependent on the extent to which the sensory centre and/or the pathway between the two centres has been lesioned. On this basis the development and/or course of a certain aphasia can be predicted. For sensory aphasia this implies that if speech comprehension improved, the rate of errors in speech production ought

to decrease. Wernicke's observations on how his patients developed confirmed this prediction (cf. e.g. Table 2).

Wernicke's notion of 'unconscious correction' and/or feedback, respectively, was later adopted by Lichteim (1885) (see Butterworth 1993). In modern psycholinguistics we also encounter similar notions, in particular in the framework of interactive network theories or connectionist models (cf. e.g. Dell, 1988), notions which also are increasingly gaining in importance in neurolinguistics (cf. e.g. Martin and Saffran 1992).

Wernicke's monitoring theory

An impaired (unconscious) correction and/or feedback loop has similar consequences for both conduction and sensory aphasics. Although both show the same above-mentioned error patterns in their speech output, conduction aphasics do so to a lesser extent, as Wernicke points out. Still, both groups of patients differ in another respect, namely, in how they react to the errors they make. Sensory aphasics who suffer from severely impaired speech comprehension are 'unconscious of their correct and incorrect use of words' (Wernicke, 1874, p. 24). Conduction aphasics, in contrast, who comprehend both their own utterances and those of other persons quite well, can employ 'another correction which is only rarely used in normal speech production and which may eventually replace the unconscious correction completely by a *conscious* [our emphasis] one. The acusticus is intact and passes on the sound of the spoken word to the unscathed locus of the sound images. The word that has been spoken is hence perceived, and judged as correct or incorrect, respectively. . . . and if he [the patient] is a strong-willed man, capable of great attentiveness, he will be able to offset his deficit by conscious correction, which is of course much more arduous and time-consuming' (pp. 26 f.).

Whereas 'unconscious correction', that is, the feedback loop between the motor and the sound centre, marks the normal case, 'conscious correction' is only rarely used in normal speaking, as Wernicke states. Conscious correction is rather a strategy employed by conduction aphasics to compensate their impairment of unconscious correction. Since the latter takes place before any overt speech is produced, it can hence be regarded as a feedback loop that is internal to the language processing system ('internal correction'). Conscious correction, in contrast, necessitates that the word/sentence is first output, and is subsequently fed into the sensory speech centre as input. For this reason it can hence be regarded as a loop that is external to the language processing system ('external correction'; cf. Figure 1).

It is striking how much similarity the external correction loop ('conscious correction') bears to current psycholinguistic conceptions of 'monitoring' one's own speech (in particular, to the one put forward by Levelt, 1983, 1989).¹⁰ These modern theories also assume—as did Wernicke—that speech production may be controlled and monitored via self-perception and via the speaker's speech comprehension system, although 'inner speech', in as much as it is phonologically encoded, can also be accessed by the monitor that is based on speech comprehension (cf. Schlenck *et al.* 1987, for aphasiological evidence). Whether all control and monitoring devices are carried out via a comprehension-based monitoring system, or whether it is necessary to also assume a 'shorter' feedback loop (e.g.

Wernicke's internal correction loop), is a matter of current dispute (cf. Levelt, 1989, pp. 460 ff.). At any rate Wernicke was obviously acquainted with both control possibilities.

Concluding remarks

It has become clear how modern problems currently discussed in psycholinguistics and neurolinguistics, such as those concerning the number of lexica, the role of speech comprehension in monitoring one's own speech, or the role of feedback between different components of language processing, have already been pinpointed by Wernicke with amazing clarity. He also tried to provide solutions to these problems with his theory of language processing. In fact, one can say that the old problems identified by Wernicke appear in new guises in modern aphasiology. Our task today is to challenge the viability of Wernicke's old solutions to these current problems.

Wernicke was ahead of his times in many respects: He laid the theoretical foundations for viewing aphasic language deficits as a central disorder, and not only a deficit concerning merely speech output. He emphasized that a close interaction between explicit model formation and empirical observation and/or documentation is indispensable for any progress in aphasiology. He chose the single-case study and not the group study as the methodological means to empirically support his theory, and also deliberately avoided hypostasizing a symptom-complex to a rigid syndrome. Finally, he made a strict distinction between problems that are related to thinking and those related to speaking, and this conviction formed the grounds for a theory of aphasia as a 'psychic focal disease' (or put in German, 'psychische Herderkrankung'; Wernicke, 1874, p. 70). The best thing is: it may well turn out that Wernicke could have been right in many of his assumptions underlying his theory of language processing, which has not yet been falsified in its basic outline. It may well be that there is only a single lexicon for spoken language, namely, a lexicon which contains abstract sound representations. The conceptions that speech production is monitored by speech comprehension, and that there is an (automatic) feedback between phonological output representations and higher-level lexical representations are also plausible. Future research will help decide these issues.

Endnotes

1. All English translations of quotations taken from Wernicke (1874) are ours, those taken from Wernicke (1906) and Wernicke (1885/1886) are Eggert's (1977).
2. However, Wernicke was aware that the syndromes he distinguished on an anatomical basis rarely occurred in pure form; much more often 'two or three of the designated symptom-complexes' (p. 32) would merge.
3. Wernicke (1874, p. 18 f.) hypothesized that the centre for sound images was located within the cortex of the first temporal gyrus. Later (cf. Wernicke, 1906, p. 224) he confirmed this claim, and added that the adjacent parts of the second temporal gyrus were also involved, an assumption he based on two autopsy studies.
4. Wernicke (1874, p. 18 f.) located this pathway in the cortex around the Sylvian fissure (insula). Later (cf. Wernicke, 1906, p. 231), he conceded that the available autopsy findings did not suffice to corroborate his conception of conduction aphasia on anatomical grounds. This argumentation, however, is not consistent, since on the other hand Wernicke (cf. 1906) pointed out the significance of the insula for 'speech function' in that it forms a focal point for the

- union of the association tracts connecting the two speech centers and whatever other regions may be related to speech function' (quoted from Eggert, 1977, p. 274). From today's perspective, a lesion in the insular cortex area may indeed result in conduction aphasia (cf. Palumbo *et al.* 1992).
5. We think that Wernicke's hesitation to sharply define the syndrome of conduction aphasia could be due to several things. First, the two cases he classified as conduction aphasia (his Cases 3 and 4) showed different disorders in processing written language. Case 3 (cf. Table 2) had a 'chronic alexia' (Wernicke, 1874, p. 72) whereas Case 4 had an 'isolated agraphia' (cf. Wernicke, 1885/86; quoted from Eggert, 1977, p. 199). Second, the chronic alexia of Case 3 could have also been caused by hemianopia. Third, Wernicke was hesitant whether Case 3 was 'particularly typical' of the syndrome (cf. Wernicke, 1874, p. 47: 'prägnanter Fall'). Fourth, the anatomical findings available at that time did not unambiguously confirm his (anatomical) view on conduction aphasia. From today's perspective, impairments of spoken and written language are regarded as distinct disorders. Therefore, Wernicke's prudence is, though laudable, unwarranted, since his diagnosis of the two cases as conduction aphasia is not called into question by their different disorders in processing written language.
 6. A further reason why Wernicke was indecisive about his conception of conduction aphasia could lie in the difficulty of delimiting conduction from anomia, a disorder to which he devoted only one paragraph.
 7. 'It can easily be asserted that the central nervous system has some kind of "memory". Experience shows that this is reflected in the resistance of a certain path to nervous excitations which can be lowered by being used more frequently. Reflex movements thus arise most easily on frequently used paths and in a form which has been practised most often.' (Wernicke, 1874, p. 4).
 8. This aspect is expressed very explicitly in a later study (cf. Wernicke, 1906) where Wernicke stated that in sensory aphasia being due to an impairment of the sensory speech centre 'articulate speech remains essentially preserved and is disturbed only in so far that there is impairment in the regulating influence of the sensory speech center which safeguards the selection of the correct motor images' (quoted from Eggert, 1977, p. 226).
 9. It is interesting to note that, in addition to the activation flowing from the sound images to the concepts, Wernicke also assumed that the activation can flow in the opposite direction. So, this route is bidirectional, as is the route between the sound and the motor images.
 10. Note that Levelt (1989) regards monitoring via the speech comprehension system as a constitutive part of his psycholinguistic model of normal speech production. Wernicke applies his conception of 'conscious correction' to a strategy extensively used and gradually built up by (conduction) aphasics to compensate for their output deficit.

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