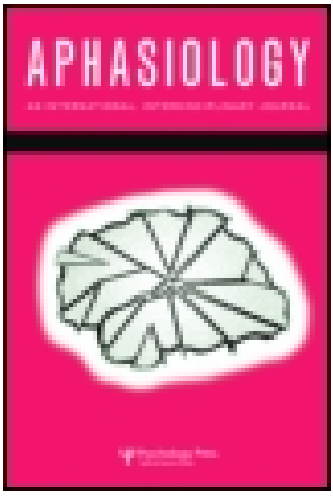

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Review

Conduction aphasia—11 classic cases

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Abstract

Eleven cases of conduction aphasia derived from a survey of the literature of the classic period of German aphasiology are reviewed. They were published between 1885 and 1934. Only those case reports were included that presented sufficient data concerning spontaneous speech, repetition and comprehension and gave examples of the patients' verbal behaviour. In summary, conduction aphasia was characterized by; (1) phonemic paraphasia with conduite d'approche, (2) preserved auditory and reading comprehension, (3) a repetition impairment that depended upon word length, (4) paragraphia with writing to dictation, and (5) a tendency in some patients to produce the unusual symptom of form-related semantic paraphasias. As anatomical explanations, it was proposed that there existed two routes, a phonological and a semantic, for repetition and that the right hemisphere may contribute to comprehension performance. Functionally, a dissociation between phonemic components of words, a disorder of the generation of the temporal sequence of speech sounds or deficient transcription of speech sound images into speech motor images, an impaired production of the single phoneme, a disorder of the associative basis for phonemes, impaired phoneme perception (incomplete sound deafness), an impairment of word sound control and the contribution of a memory deficit were assumed as relevant preconditions. The theories of the classical writers are compared with modern accounts of the pathogenesis of conduction aphasia.

Introduction

Within the framework of Wernicke's model (Wernicke 1874) of the representation of language functions in the brain, conduction aphasia ('Leitungsaphasie') was predicted as the consequence of a disconnection between the sensory and the motor language centres:

> The patient comprehends all (...). He can say everything, but the choice of correct words is impaired in a similar manner as just described (*with aphasia resulting from lesion of the sensory*

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> *language centre—the authors*). The auditory word image ('Klangbild') is preserved and can be accessed from those associations that form the word concept, but it cannot determine the correct choice of motor concepts ('Bewegungsvorstellungen') (...). Therefore, words are confused (...). However, hearing is intact and the auditory perception is transmitted to the centre for auditory word images. The spoken word is perceived and found correct or wrong. The patient (...) knows that he spoke wrongly (...); if he is a strong-willed and attentive person, he will be able to compensate his deficit by conscious, laborious and time consuming correction (pp. 26–27).

Contrary to recent conceptions, Wernicke, in his 1874 thesis, did neither predict nor even mention a prominent repetition deficit in conduction aphasia (for a lucid discussion of these 'misconceptions and misrepresentation' see de Bleser *et al.* (1993)), although he drew attention to the 'crucial task' of the direct pathway between the two language centres, namely, the repetition of words (see Blanken *et al.* 1994, p. 211). In his case descriptions, Wernicke (1874) focused on the patients' failing attempts at self-correction, resulting from lack of control of the motor centre:

> Many productions succeed, especially small talk; but then a critical word comes up and he gets stuck; he struggles, gets angry, and almost every word that is now haltingly produced is meaningless; he continues to correct himself, but the more he tries, the worse it gets (Wernicke 1874, pp. 47–48).

Wernicke viewed the presence of attempts at output correction in conduction aphasia and their absence in sensory aphasia as the main distinguishing feature between the two syndromes.

Describing a patient with what he held to be conduction aphasia within the framework of his model of the physiological basis of language disturbances, Lichtheim (1885) concluded that a defect of repetition should be an inherent symptom of the syndrome resulting from the interruption of the direct pathways from the sensory to the motor language area. His patient, however, exhibited paraphasia with repetition only to a similar extent as in spontaneous speech (see de Bleser *et al.* (1993) for a translation of the relevant passages).

In his seminal paper 'Disconnexion syndromes in animals and man', Geschwind (1965) resuscitated the classical German (Wernicke 1874, Lichtheim 1885) interpretation of conduction aphasia resulting from a disconnection of the anterior from the posterior language area by lesion of the arcuate fasciculus. Damasio and Damasio (1980) added empirical support to this claim when analysing the lesions as shown by the CT of six patients with fluent speech with phonemic paraphasia and a repetition deficit that spared digits. On the other hand, the Damasios also pointed out that the lesion of quite a number of functional components of the language apparatus can account for deficits of repetition, so that the number of lesions possibly involved render it unlikely that all forms of conduction aphasia can be related to a single anatomical structure. Kertesz (1977) described on the basis of numerical taxonomic studies a bimodal distribution of conduction aphasics along the fluency dimension. Caramazza *et al.* (1981) suggested two types of conduction aphasia differing with respect to phonemic paraphasias in speech output and concluded that patients with repetition deficit but without output problems suffered from a pathological limitation of auditory-verbal short term memory.

Like Wernicke, Kohn (1984) focused her interest in conduction aphasia upon phonemic paraphasia in non-repetition tasks. She proposed a disruption of a pre-articulatory programming stage so that the phonological representation of a word

cannot immediately be transcribed into a phonemic string. Errors in these translations together with deficient output monitoring and/or error correction result in the characteristic, laborious sequences of self-corrections, the 'conduite d'approche'.

Benson *et al.* (1973) proposed the following criteria for diagnosis of conduction aphasia: (1) fluent, paraphasic conversational speech; (2) absence of significant comprehension impairment; (3) repetition disturbance of a significant degree. Obviously, 'significant' is a statistical term, rendering a qualitative diagnosis of conduction aphasia difficult (compare Wallesch and Kertesz (1993)).

The modern literature is divided with respect to the critical symptom(s) of conduction aphasia with neurologists focusing on the repetition deficit (e.g. Geschwind 1965, Benson 1973, Kertesz 1977) and neurolinguists on the phonemic disorder (e.g. Joanette *et al.* 1980, Kohn 1984, 1992, Buckingham 1992a, b).

The present study attempts to analyse the classical German papers from 1874 (Wernicke 1874) to 1934 (Kleist 1934) aiming at a description and interpretation of which symptoms and mechanisms were originally thought to constitute conduction aphasia. In the international literature, the topic was only rarely discussed until Weisenburg and McBride (1935) probably following the taxonomy of Kleist (1934) included 'repetition aphasia' in their classification system (but compare Pershing 1900). Most English, French and American aphasiologists of the time did not subscribe to localizationism, therefore the theoretical necessity of a disconnection syndrome was lacking. Bastian (1887), whose theory was based on the assumption of centres, suggested that the disconnection of the auditory-kinaesthetic route produced a syndrome similar to Broca's aphasia. Wyllie (1894) postulated a syndrome of Wernicke's aphasia without word deafness. The French literature includes a highly interesting discussion of the genesis of phonemic paraphasias which, however, found no solution as linguistic models were lacking (e.g. Pitres 1899, and the debate in the Société de Neurologie de Paris 1908, see Lecours *et al.* 1992, Leischner 1992).

Two cases from Wernicke (1874)

(for an extensive English translation of the relevant passages see de Bleser *et al.* 1993)

The concept of conduction aphasia was a consequence of the model Wernicke developed in his 1874 MD thesis. The syndrome was outlined with reference to the theory. In the last section of his book mainly dealing with case studies Wernicke described two patients fitting in this context:

Wernicke (1874): Beckmann

This 64 year old pharmacist had suffered a stroke, which according to Wernicke left his comprehension and the contents of his lexicon intact:

> His knowledge of words is without limits. However, for many objects that he wants to name, the words are missing; he tries hard to find them, gets angry, and if the name is given to him, he can repeat it faultlessly (...) On the street, he can read the signs in passing; however, when a specific word or letter is indicated, he is unable to produce it. (...) There is agraphia, he can copy everything, but cannot write on his own. (...) He is more successful with digits, but even two-digit numbers are a hard task for him (p. 47–49).

This patient, explicitly called a 'telling case' ('prägnanter Fall') of conduction aphasia by Wernicke, was able to repeat with ease. Instead, Wernicke's description and theoretical account focus upon Beckmann's futile attempts at production, the 'conduites d'approche'. The passage relating to this symptom has already been quoted ('Many productions succeed...', see above).

Wernicke (1874) : Kunschkel

Kunschkel was a 50 year old goldsmith who had suffered a blow to his right ear that rendered him aphasic and right hemianopic:

> He spoke adequately for quite a while, until finally at the end of a sentence there was an incorrect word (...). When asked specifically about this word he had just spoken, he attempts to correct, and produces a jumble of wrong words and syllables (...), a jargon that can hardly be repeated. (...). He comprehends all and with ease. There is alexia (...) and also agraphia (p. 51).

Wernicke notes rapid improvement of Kunschkel's aphasia. Again, Wernicke's description is focused upon the patient's presumably phonemic paraphasia and his futile attempts at correction.

Eleven cases of conduction aphasia from the German literature between 1885 and 1934

The 11 following cases are included because (1) they were presumed by the respective authors to represent conduction aphasia and (2) the descriptions contain information about spontaneous speech, repetition and comprehension together with examples of productions that allow confirmation and reanalysis.

Lichtheim (1885): Samuel Berger

The 46 year old farm hand had suffered a cerebral infarction. Autopsy about 1 month later demonstrated a lesion in the area of supply of the left arteria fossae sylvii (in modern anatomical nomenclature the middle cerebral artery).

Comprehension, reading and copying were found intact, Berger's symptoms included phonemic paraphasias, paralexias and paragraphias in spontaneous speech, repetition, reading aloud and writing, respectively.

> With repetition of complete sentences, the same deficits were present as in voluntary speech. On the other hand, single words were repeated correctly (p. 217).

Kleist (1905): Lina S.

Following an evening in the dance hall, the 20 year old housemaid awoke with aphasia and right hemiparesis. Endocarditis was suspected.

Comprehension was only mildly impaired and, in case of failure, was found to improve after repetition. Spontaneous speech was telegraphic and halting with word finding difficulties. Kleist describes a prototype of agrammatism:

> ... almost exclusive use of the nominative and accusative case and the infinitive and participle forms with deletion of function words (p. 504).

Phonemic paraphasias were frequent, especially with longer words. Reading comprehension was intact, reading aloud was paraphasic, writing spontaneously

and to dictation was paragraphic. Repetition of sounds, syllables and short words was intact. There were effects of frequency and word length upon repetition performance. Repetition of pseudowords was especially impaired but not impossible.

Heilbronner (1908): Alwine S.

During a severe illness with jaundice and delirium, the 54 year old merchant wife developed a language disorder.

Comprehension was only mildly impaired and improved rapidly. In naming, word finding problems were frequent and could often be solved by descriptions of use or category. Repetition was massively impaired. The patient clearly followed a semantic strategy with descriptions of use: ('chair'—'you are sitting on it'), context ('sky'—'with the almighty') or semantic conduite d'approche ('child'—'animal, small girl, a child'). Frequently, she constructed a personal context ('foot'—'my foot here'; 'dog'—'a dog, our dog, that is running along here'). The patient refused to repeat pseudowords ('I cannot, these things are alien').

Reading aloud was relatively well preserved and clearly better than repetition. Reading comprehension was found almost intact. Brief conversations were impaired. Heilbronner does not state why, although he notes that the patient was quite talkative ('schwatzsüchtig'). When investigated 6 months later, the deficits were all improved, but the pattern of symptoms had remained stable.

Stertz (1914): Hermann G.

A week before admission, the 52 year old shipbuilder suddenly developed a language disorder, apraxia and headaches. He stayed in hospital for several months, but diagnosis could not be secured.

Comprehension was initially impaired but improved markedly. Spontaneous speech was stereotyped at the beginning. Eight months post onset, it was characterized by some word finding difficulties, semantic paraphasia and paragrammatism.

Two months post onset, naming resulted in phonemic conduite d'approche. Repetition was partially preserved, especially for short high frequency words with few consonants. Errors mainly consisted of phonemic paraphasias and perseverations. Repetition of pseudowords was massively impaired. Reading comprehension was found intact, writing to dictation was grossly deficient with frequent literal paragraphias.

Försterling and Rein (1914): Heinrich Roskosch

The 28 year old chronically alcoholic tramp was admitted to the mental asylum because of drowsiness. When consciousness cleared 2 weeks later, he was aphasic, acalculic and amnesic. He was assessed in some detail 2 months post onset.

Auditory and script comprehension was almost intact. Spontaneous speech exhibited mild word finding problems, in which case HR used adequate circumscriptions, and few phonemic paraphasias.

Repetition was markedly impaired. HR repeated more than 50% of words, pseudowords and syllables incorrectly. Phonemic paraphasias and conduites

d'approche were prominent. Oral naming was impaired in similar fashion and degree. Written naming and writing to dictation contained paragraphias but were better preserved than oral performance. Spontaneous writing contained paragrammatic errors. Reading aloud was intact.

Liepmann and Pappenheim (1914): Maximilian Ernst Biermann

This 62 year old patient was also a tramp. One or 2 weeks before admission he was suddenly rendered speechless. He died 2 months later. Autopsy revealed a left temporoparietal infarct. His language deficits were reported as stable during his 2 months in hospital.

Comprehension was described as intact. Spontaneous speech was characterized by severe word finding deficits and phonemic paraphasia.

Naming of short target words was relatively intact with 3/17 failures, 6/17 successful conduites d'approche and 8/17 instantaneously correct productions. Monosyllabic words were repeated almost without fault; with bisyllabics, frequent phonemic paraphasias and conduites d'approche were noted. Repetition of pseudowords was grossly phonemically distorted. Script comprehension was found intact, reading aloud only mildly impaired. Writing to dictation was found similarly impaired to repetition.

Kleist (1916): Friedrich S.

The 52 year old bookseller suddenly developed aphasia and headache in 1908. Initially, he was found word-deaf and could neither read nor write. He was investigated 3 years later. Repetition was characterized by marked phonemic paraphasia with 15 successful conduites d'approche among 50 attempts (18 failures, mainly after conduite) and 17 correct performances. Kleist noted effects of word length and frequency. Repetition of pseudowords was even more impaired.

Comprehension was mildly affected (and awkwardly assessed by asking the patient to explain verbally abstract words that were presented auditorily—Kleist notes a deficit of auditory word form processing by this method).

Spontaneous speech was scarce. The naming disorder was characterized by length and frequency effects. Short and well known words were correctly produced in most instances. Kleist notes phonemic and occasional semantic paraphasias.

Reading comprehension is described as superior to auditory comprehension, reading aloud was found almost intact. Writing to dictation is reported as impaired in a similar fashion as repetition. The patient was found able to write a short letter with only few errors of word position.

Bonhoeffer (1923): LH

One month before admission, the 31 year old wall painter fell aphasic after chloroform analgesia for a tooth extraction. An autopsy report is quoted describing lesions of the middle part of the left postcentral gyrus and of the inner part of Heschl's gyrus. Date and cause of death are not given.

Comprehension was found intact. Spontaneous speech was halting and laborious with phonemic paraphasia and conduite d'approche. Naming was massively

phonemically distorted. Letters and monosyllabic words were repeated correctly. With increasing word length, phonemic paraphasia increased and there were frequent sequences of phonemic approximations. Writing to dictation was grossly paragraphic. Reading comprehension was intact, reading aloud mildly impaired at first, later intact.

Grubel (1925): E. Roetter

This 44 year old merchant's wife had suffered a stroke with aphasia and transient right hemiparesis 5 months before admission.

Comprehension was found intact. Spontaneous speech contained phonemic and less frequently semantic paraphasias. Naming was relatively well preserved (20 phonemic paraphasias with 120 items, mostly followed by self-correction). ER was able to repeat correctly 12 out of 35 mono- and polysyllabic pseudowords. Repetition of words contained some phonemic paraphasias (obviously not many as Grubel can give only seven examples). Reading comprehension, reading aloud, spontaneous writing, written naming and writing to dictation were only mildly impaired.

Hilpert (1930): Max K.

In 1914, at the age of 22, this 38 year old teacher had been wounded in the left parietal region by a shrapnel. He developed a chronic abscess in the left lower parietal lobe which led to seizures and progressive hemiparesis with ataxia.

Comprehension of longer sentences was impaired by delay. The patient explained that he knew the words, but needed time to recognize them, i.e. access the underlying concept (patient's explanation). Spontaneous speech was scarce, with, short phrases, lacking or wrong inflectional endings, word finding difficulties and frequent phonemic paraphasia.

Repetition of monosyllabic words was good. Word length was noted as the critical variable for repetition impairment: 'The disorder is absolutely dependent upon the number of syllables and not upon whether he has a concept of the word meaning or not. Repetition of nonsense syllable sequences is difficult only because he is unable to imagine the written representation' (p. 231). Conduite d'approche is demonstrated by many examples. Hilpert draws attention to the patient's ability to repeat longer words correctly syllable by syllable. He also notes that MK was unable to repeat words upon command that he had just produced fluently in conversation. Naming was severely disturbed. Hilpert states that MK was almost regularly able to arrive at the target words after numerous attempts and gives an example of a semantic conduit:

> Federhalter (pen): pencil, Blei... nein, pencil, Schreib... Feder... Feder... Feder... Hand... Halter... Federhalter (p. 232).

MK frequently produced English words which Hilpert found difficult to explain. He notes that the patient had neither spoken nor read English since his schooldays.

Reading comprehension of single words and short sentences was prompt. Hilpert describes difficulties with irregular words ('words not written phonologically'). In some instances, reading comprehension could be established only by moving the finger along the outlines of the letters. Spelling of printed words was found possible only with pantomimic copying. Reading aloud was massively

impaired with frequent phonemic paraphasias and conduite d'approche. MK could write to dictation only single words and relied upon oral repetition.

Stengel (1933): Marie B.

The 40 year old woman had an egg-sized cyst operated upon in the lower part of the left central sulcus. Following the operation right facial weakness and right hemianopia were noted.

Comprehension was found almost intact. Spontaneous speech was fluent with phonemic and semantic paraphasia, word finding difficulties and grammatical errors. A relative lack of nouns is noted. Naming was massively impaired by phonemic paraphasia. Stengel notes phonological similarities between the neologisms that were produced and the word form of the target words (formal paraphasias—for a discussion compare Blanken (1990)).

Repetition was massively impaired. Stengel describes occasions of complete lack of response or fruitless lip movements. Letters and short words were repeated usually correctly. With longer words, the productions frequently contained the initial and final letters of the target word. Writing to dictation was similarly impaired to repetition, and spontaneous writing resembled spontaneous speech. Reading comprehension was almost intact.

Table 1 gives an overview of the language symptoms of the 11 post-Wernicke cases presumed to represent conduction aphasia that were described above. These certainly do not constitute all or even the majority of cases in the German literature of the period, but were chosen because of their adequate assessment and description.

As far as can be derived from the mostly incompletely and unsystematically presented case descriptions, phonemic paraphasia with conduite d'approche, as originally highlighted by Wernicke, is the most consistent symptom reported in seven out of 11 cases. A prominent deficit of repetition was noted only in one case (Wernicke 1906, not included because of insufficient details). An effect of word length upon repetition performance was described in seven cases and explicitly denied in two. An effect of word frequency was found in three patients and was not present in another three. At least two patients exhibited the relatively rare symptom of form related semantic paraphasia with repetition. It is supposed to reflect an instability in word-form access as a variant of a phonological disorder (Blanken 1990). One additional patient showed this symptom with naming (Stengel 1933). Semantic paraphasia with repetition was noted in five patients, in two of these of the form-related variant, and explicitly stated as absent in two. The majority of patients had relatively well preserved comprehension. Naming performance was variable and was characterized by phonemic paraphasia in the majority of cases. Spontaneous speech ranged from nonfluent agrammatic (Kleist 1905) to fluent paraphasic. With the exception of Hilpert's (1930) case, reading comprehension was well preserved. Reading aloud was variable ranging from the almost intact (Kleist 1916) to most impaired (Hilpert 1930). Writing to dictation was found paragraphic when analysed in detail.

In summary, conduction aphasia in the classic German literature is characterized by:

- Phonemic paraphasia with conduite d'approche;
- Preserved auditory and reading comprehension;

Table 1. Overview of language symptoms in 11 classic cases of conduction aphasia

	Lichtheim (1885)	Kleist (1905)	Heilbronner (1908)	Stertz (1914)	Försterling and Rein (1914)	Liepmann and Pappenheim (1914)	Kleist (1916)	Bonhoeffer (1923)	Grubel (1925)	Hilpert (1930)	Stengel (1933)
Repetition:	'single words good'		'most impaired'					'altogether not too bad'			
Effect of word length	?	short > long	'fails even short'	short > long	no	monosyll. correct	short > long	short > long	no	short > long	short > long
Effect of frequency	?	frequent > infreq.	frequent words	frequent > infreq.	no	?	frequent > infreq.	?	no	?	no
Phonemic conduite d'a.	?	+	+	?	+	+	+	self-corr.	?	+	?
Semantic paraphasia	?	form-rel. paraph.	+	no	+	form-rel. paraph.	+	?	no	?	?
Comprehension	good	mildly impaired	good	mildly impaired	almost intact	good	mildly impaired	good	intact	impaired, delayed	intact
Naming	WFD	?	some phon. & sem. paraph.	WFD, paraph.	severely impaired, phon. paraph.	severely impaired phon. paraph.	phon. & sem. paraph.	phon. paraph.	phon. & sem. paraph.	good, some English words	form-rel. paraph.
Spontaneous speech	phonemic paraph.	nonfluent WFD, phon. paraph.	fluent	stereo-typed, nonfluent	WFD, phon. paraph.	WFD, phon. paraph.	scarce, nonfluent	halting	rel. well preserved	WFD, phon. paraph.	WFD, pho & sem. paraph.
Reading comprehension	intact	intact	intact	good	intact	good	better than audit. compre.	good	good	reads by writing	good
Reading aloud	paralex.	paralex.	literal & sem. paralex.	intact	almost intact	good for letters	almost intact	lit. paralex. at onset	?	most sev. impaired	impaired
Writing to dict.	paragraph.	paragraph.	(refused)	impaired	paragraph.	good for letters	paragraph. similar to repetition	?	?	by spelling aloud	?

WFD: word finding difficulties; phon.: phonemic; sem.: semantic; paraph.: paraphasia; paragraph.: paragraphia; paralex.: paralexia.

- A repetition impairment depending upon word length;
- Paragraphia with writing to dictation; and
- A tendency in some patients to produce the unusual symptom of form-related semantic paraphasias,

but none of the symptoms was found in all 11 cases.

After Lichtheim (1885), impaired repetition as a key symptom of conduction aphasia was next highlighted by Heilbronner (1908). From then on, it took more than another quarter of a century, until Kleist (1934) and, in the first attempt at psychometric classification of aphasia, Weisenburg and McBride (1935) coined the term 'repetition aphasia', elevating repetition impairment to the cardinal symptom of conduction aphasia.

Theoretical accounts of conduction aphasia in the classic German literature

> Rarely the contrast of conceptions advocated in language pathology shows up so clearly as with regard to the discussion of the complex of those findings, that since Wernicke is called conduction aphasia, and that has been coined prominently as central aphasia by Goldstein. (Isserlin 1929)

As stated above, Wernicke in his 1874 thesis deduced the symptoms of conduction aphasia from his model and described two cases with paraphasia together with attempts at correction as the clinical correlate. According to him, there is a qualitative distinction between conduction and sensory aphasia. In the latter,

> ... the correct or wrong use of the words remains unconscious (p. 24),

whereas in the former

> ... the spoken word is judged as correct or wrong (p. 26).

(See Blanken *et al.* (1994) for an extensive discussion.)

Highly important for his theory of conduction aphasia was the co-occurrence of alexia and agraphia, as reading and writing, in Wernicke's conception serial associations of letters and sounds, are completely dependent on a direct connection of motor and sound images. Consequently, Wernicke (1886) rejected Lichtheim's conduction aphasia case as not representing the syndrome, because this patient had been able to read with full comprehension, but made phonemic errors when reading aloud as a form of 'amnesic aphasia'.

In a later paper, Wernicke (1906) included repetition performance in his description of conduction aphasia and states that automatic (echolalic) repetition and repetition of pseudowords are rendered impossible by the lesion of the direct route between the sensory and motor speech centres. Meaningful words, however, were spared because of the intact indirect semantic route via the centre for concepts. This theory would predict meaning-related semantic paraphasias with repetition. A number of authors tested these predictions in their case analyses. Near semantic paraphasia with repetition was found by Heilbronner (1908) and Kleist (1916), but only occasionally. A marked impairment of the repetition of pseudowords was described by Heilbronner (1908), Liepmann and Pappenheim (1914), Försterling and Rein (1914), Kleist (1916) and Grubel (1925).

In line with the Wernicke-Lichtheim model, Heilbronner (1908) points out that the presence of semantic paraphasia in repetition tasks in his patient Alwine S. supports the assumption of two routes for repetition, a semantic via the concept

centre and a direct phonemic one (compare Howard and Franklin (1988), for a recent review). In a later handbook article, Heilbronner (1910) presents a new description and analysis of conduction aphasia:

> ...occasionally—although not too often—one encounters patients with little or no comprehension impairment, without deficit of speech production, the intactness of which is documented by intact production of series, usually quite talkative, but with severe disturbance of spontaneous speech, with prominent word distortions and frequently with marked word amnesia in naming. These components vary in degree, and depending on these components there may be similarities to transcortical motor or transcortical sensory aphasia (...). One aspect, however does not comply with such classification. In accordance with an assumption later suggested by Wernicke, these (patients) exhibit a severe disorder of repetition, the function whose intactness characterizes the 'transcortical' forms. (...) indeed, the grimacing movements which accompany these patients' attempts to produce or repeat single words suggest confusion with motor aphasia; however, the ease with which the patients speak otherwise, especially (overlearned) series, excludes even stages of restitution of motor aphasia (p. 1032).

This description is the first that emphasizes repetition as a prominent distinguishing feature.

In the analysis of his case Lina. S., Kleist (1905) focused on an explanation of phonemic paraphasias. Contrary to his later positions (e.g. Kleist 1934), he argues in 1905 that auditory and motor word images 'must be merged together at each locus in the whole area of the brain that subserves language functions' (p. 522). Kleist (1905) explains phonemic paraphasia and paragraphia as resulting from dissociations between the phonemic components of words. A similar account based on Bastian's (1887) theory of the various levels of impairment has been proposed by Lewy (1908), who stated that phonemic paraphasia resulted from a 'functional reduction of the arousability of centres that can result from direct lesion or from diaschisis' (p. 856). Stertz (1914) assumes a 'disturbance of the complex associative interplay of various groups of sensory and motor components of excitation' (p. 358) to form the physiological basis of paraphasia.

In 1916, Kleist gives an anatomo-physiological explanation for phonemic paraphasias by assuming a disorder of generation of the temporal sequence of speech sounds:

> If the acoustic element does not correspond with its (kinaesthetic-motor) counterpart, how can this result in a disorganization of the sequence of the temporal order of the sounds of the spoken word? One cannot explain the disturbance of a temporal structure (paraphasia) on the basis of a disturbance of sensory connections. (...) Dissociation in time is not a consequence of sensory disintegration (p. 166).

Kleist assumes that this deficit had a defined neurophysiological correlate, the impairment of the transcription of speech sounds into speech motor images. A similar locus of disorder, together with a feedback impairment, was later suggested by Dubois *et al.* (1964), and Kohn's (1984) recent account of the phonemic disorder of conduction aphasics resulting from a postlexical transcription deficit below the phonological but above the motor level representation seems also related.

While Kleist in his earlier paper (Kleist 1905) had come to the conclusion that conduction aphasia was the 'pure aphasia of word concepts' ('Wortbegriffs-aphasie') and had supposed all other aphasic syndromes to be combinations of this and word deafness or word muteness, he now in 1916 (Kleist 1916) views the clinical syndrome of conduction aphasia as a combination of paraphasia with mild word deafness and not as a pure (monocausal) syndrome. This explanation was supported by autopsy data from Pick (1898, pp. 123–133) and Liepmann and

Pappenheim (1914) who found damaged not only the region intermediate between the motor and sensory speech centres but also the left auditory and sensory language areas. The position of Kleist (1916) was shared or only slightly modified by Bonhoeffer (1923) and Grubel (1925).

In 1934, Kleist drastically altered his views with respect to conduction aphasia again. In his monumental book 'Gehirnpathologie' (1934), he now uses the term 'repetition aphasia' ('Nachsprechaphasie'). He assumes a disorder of the production of the phoneme to be the core deficit and speculates that an apraxic component could be a constituent of the syndrome. So, although Kleist accepts the impairment of repetition as the distinguishing feature, his explanatory attempts continue to focus on the functional basis of phonemic paraphasia. A resemblance to the modern account of Kohn (1993) must be noted.

Kleist (1934) gives an anatomical explanation for the dissociation between repetition and comprehension in conduction aphasia. He assumes that the right hemisphere homologue of Wernicke's area is able to take over perceptive language functions but cannot control the left motor speech area. Paraphasias occur, because 'regulations from the right temporal lobe cannot pass undisturbed' (p. 134). A similar but less elaborated proposal had earlier been made by Liepmann and Pappenheim (1914). In more recent years, this anatomical theory of Kleist has been revived by Benson *et al.* (1973).

Phonemic paraphasias (the term 'paraphasia' was coined by Kussmaul in 1877, who called them 'literal paraphasias'; for the term's further history cf. Buckingham 1989: 94ff.) were an enigma to classic aphasiology, partly because no appropriate linguistic theory was available. According to Robins (1979), during the 19th century phoneticians were concerned with the improvement of orthography, aiming at the goal of 'one sound, one symbol'. But in the latter half of the century, it became evident that this goal was unattainable: Every useable orthography would omit many observable phonetic differences, and every orthography satisfying the demands of narrow transcription would be too complicated for practical use. So it was Henry Sweet, who in his 'Handbook of phonetics' of 1877 (Sweet 1877) drew the distinction between sounds, whose characteristics depend on their phonetic environment (like [ç] and [χ] in German *ich* and *ach*) and which therefore are non-distinctive, and sounds which can establish lexical contrasts, therefore being distinctive (like /m/ and /h/ in English *mouse* and *house*). Only the latter would be relevant for orthography and would have to be noted separately. Clearly, this was the concept of 'phoneme', although Sweet did not use the term which was introduced by Baudouin de Courtenay, at first in the Russian form 'fonema'. Baudouin de Courtenay (1895/1972, p. 152) proposed an interpretation of 'phoneme' as 'the psychological equivalent of a speech sound', thus existing 'in the mind'. And this author was the first who saw parallels between deviations in normal speech and pathological phenomena in aphasia (Baudouin de Courtenay 1886/1972, p. 121; cf. Buckingham 1992b, p. 42).

In the first third of this century, the status of the phoneme was debated controversially, and 'it was variously held to be a psychological entity, a physiological entity, a transcendental entity, and just a mere descriptive invention' (Robins 1967, p. 204). Nikolai Trubetzkoy, member of the Prague school of linguists, in his 'Grundzüge der Phonologie' (Trubetzkoy 1939/1958) then developed an extensive phonological theory, in which the phone is defined as an entity of 'la parole' in the sense of de Saussure, while the phoneme is defined as a

functional entity of 'la langue', the system of a language. The function is a distinctive one, namely: 'Contrasts of sounds which differentiate the intellectual meaning of two words in the language in question, we call phonological [...] or distinctive oppositions' (Trubetzkoy 1939/1958; p. 30; translated by the authors). While in his earlier work Trubetzkoy, like Baudouin de Courtenay, proposed a psychological interpretation of phonemes as 'Lautvorstellungen' ('sound images') behind the performance of sounds, he eventually dropped this psychological view (cf. Trubetzkoy 1939/1958, p. 37) arguing that the term 'Lautvorstellung' was misleading because 'acoustic-motor images', controlling speech, would have to be associated with every phonetic variant. This would contradict the definition of 'phoneme' and obscure the distinction between sound ('Laut') and phoneme. So he concludes: 'The phoneme is, above all, a functional concept which must be defined with regard to its [distinctive] function. With psychologistic terms such a definition cannot be realised.' (Trubetzkoy 1939/1958, p. 38; translated by the authors.) Indeed, Trubetzkoy's argument is right if one defines 'phoneme', like Baudouin de Courtenay did, as the psychological equivalent of the concrete speech sound.

Nevertheless, a psychologistic conception of the phoneme can make sense: Jakobson (1941/1969, p. 36f.), following Liepmann and Kleist, distinguishes between 'aphasic disturbances of sound [aphasischen Lautstörungen]' on the one hand and dysarthria and anarthria on the other. Aphasic disturbances according to Jakobson are characterized by the loss of parts of a 'mnestic possession [mnestischer Besitz]'. But what, Jakobson asks, is this possession? He then argues that during language acquisition it is not about [geht nicht um] learning the capacity to produce or perceive sounds ('Laute') *per se*, but about learning the 'distinctive linguistic value of the respective sounds, and thus, for the unlearning ('Verlernen') of the aphasic not the restriction [Einschränkung] of the pronounceable or hearable sounds is essential, but that of the *functional distinctive sounds*'—say: the phonemes (Jakobson 1941/1969; p. 37; translated by the authors). This is the theoretical justification of using the term 'phonemic paraphasia', and, surely, it was not at hand in the days of Wernicke and his successors.

For the early Wernicke (1874), the syllable or the word is the basis of language production, and the centre of sound images ('Klangbilder') of the words, localized in the superior temporal lobe, may be affected by an aphasic disturbance, later called 'cortikale sensorische Aphasie' by Lichtheim (cf. Wernicke 1886) or 'Wernickesche Aphasie' (Freud 1891/1992), resulting in confusions of words ('Verwechseln der Wörter'; Wernicke 1874). In the description of his two cases of sensory aphasia, Wernicke (1874/1974, p. 40) speaks of senseless and distorted words ('unsinnige oder entstellte Wörter'), what can be interpreted as the occurrence of phonemic paraphasias and neologisms. (Cf. Wernicke 1906, p. 495, for the same characterization). Following Wernicke (1874), a similar, although less severe, disturbance may result from a disruption of the connection between the centre of sound images and the centre of motor images of the words (localized in the first frontal convolution), causing what was later called 'conduction aphasia' by Lichtheim (cf. Wernicke 1886). In his later case-descriptions, Wernicke (1886), again, speaks of 'senseless words', in the second case of 'wrong words and syllables'; this patient showed neologisms in his spontaneous speech. What is the mechanism which produces distorted or senseless words under the condition of sensory or

conduction aphasia? It is Wernicke's (1874) hypothesis, that during speech production the sound images have an influence on the motor images and they have to be corrected permanently, that is: controlled. In case of destruction of the centre of sound images or disruption of the connection between this centre and the centre of motor images, the lack of control causes errors of production in the centre of motor images. (Cf. Blanken *et al.* 1994, for further discussion.) Clearly, this is a hypothesis about deviancies in word form production in general, not especially about the origin of phonemic paraphasias.

The problem of the generator of paraphasias was raised again in the Dejerine/Marie debate in 1911 but instantly dropped. Marie, who was certainly not unimaginative, confessed that he had no speculation on the generation of paraphasias.

An explanation off the Wernicke—Kleist mainstream and more in line with Goldstein, who saw the cause of phonemic paraphasia in a disorder of the successive associative basis of phonemes by a 'loosening of the association complexes in the language field' (Goldstein 1912, p. 742) is given by Hilpert (1930). In his view, production of phonemic paraphasias resulted from the 'inability to activate kinaesthetic speech motor engrams ('kinästhestische Sprecherinnerungsbilder') from acoustic or optic stimuli. The associative connection between kinaesthetic, acoustic and optic memory images is impaired' (p. 243). All symptoms of conduction aphasia including the word finding impairment could be explained as resulting from a deficit of associative mechanisms. Hilpert also drew attention to the possible role of memory deficits in the pathogenesis of conduction aphasia (compare Shallice and Warrington 1977, Caramazza *et al.* 1981).

Another original account was advanced by Klein (1931) who proposed an impairment of word sound control as the core deficit. His empirical evidence for this assumption was the presence of 'iterations' in his patient Mrs L. (not included in the review of cases because of insufficient details). Without giving examples, Klein terms as 'iteration' the multiple repetition of a paraphasically produced word in phonemic variation but without improvement. These were present in spontaneous speech, with naming and repetition. His observations probably correspond to conduits d'ecarte. In modern terms, Klein seems to suggest a monitoring deficit to underlie the conduction aphasics' conduits. The role of output monitoring in conduction aphasia was recently reviewed by Buckingham (1992a). On the basis of Joanette's *et al.* (1980) finding of regularly successful approximations in conduction aphasics, he rules out a defective monitor underlying this syndrome. However, as Kohn (1984) points out, in sequences of phonemic approximations there are rare instances, when patients do not realize, that they already had produced the right response. This indeed might reflect a momentary breakdown of a phonemic monitoring system, due to a disruption of phonemic discrimination in the face of repetitively failed trials for correct pronunciation of the target word, but cannot account for the generally good ability of conduction aphasics to recognize phonological errors.

The most detailed classical theory of the production of phonemic paraphasias was given by Kleist (1934):

> ... there is a parallelism between the degree and type of phoneme sequence paraphasia and word deafness; both symptoms rely in exactly the same way upon whether long or short words shall be comprehended or spoken, or whether a reliable, less reliable or no memory trace is present for the respective words or sound sequences (...). One is lead to assume that the production of

words and sound sequences succeeds or fails to the extent that the patient is able to internally activate the acoustic word image... (p. 720).

What is control and regulation of the act of speaking by the acoustic word image in psychological terms, is physiologically the coordination of the language movements ('Sprachbewegungen') by the acoustic word engrams. Similarly, Dejerine has called paraphasia a kind of disorder of coordination, an ataxia of speech movements. This view has the advantage that the psychopathological symptom of paraphasia can be subsumed under a well-established term of general neuropathology, namely the dependence of movements from sensations or from their engraphic representation, i.e. images (p. 721).

In summary, it can be stated that Wernicke's theory of the connection of literal paraphasia with word deafness and of both with the first temporal convolution is unhampered (p. 724).

On the basis of this theory, Kleist gives his account of conduction aphasia:

...a partial disturbance of language comprehension is a regular feature of conduction aphasia. (...) not so much word comprehension but a deeper level of language comprehension is affected, so that it is rather an incomplete 'pure language deafness'—today, we call it more precisely incomplete sound deafness (p. 725).

Justified criticisms have been raised against the interpretation of repetition aphasia as a conduction aphasia or a 'central aphasia'. Wernicke suggested the insula as its location which could not be, as there are no or other aphasic disorders with its lesion, but depend upon whether the insula alone is lesioned or the insula together with parts of the adjacent motor or sensory language zones. (...) Secondly, the arcuate fasciculus must be considered as a connection between the temporal and centro-frontal language areas. However, in the case Liepmann-Pappenheim the arcuate fasciculus was not relevantly lesioned and thus could not be the critical brain structure for the symptomatology. Consequently, one must assume that excitations are transmitted from the sensory to the motor language area by a chain of shorter fiber bundles and via the cortex of the (...) most inferior portion of the parietal lobe. (...) this would imply an autonomous participation ('selbsttätige Mitwirkung') of the inferior parietal praxis area in language. (...). It has to be investigated whether the clinical picture of repetition aphasia contains an element of apraxia and whether cortex or white matter of the most inferior anterior parietal lobule is regularly involved and whether isolated lesions of this area result in apractic-phasic disorders—or whether the lesion of Sm [i.e. supramarginal gyrus; the authors] is inconsequential for repetition aphasia. In this case, all symptoms of repetition aphasia must be derived from the lesion of the temporal language zone. Repetition aphasia would then be an incomplete language deafness, a speech sound deafness, the impressive part of which—comprehension deficit—would be masked by functions of the right temporal lobe, while the expressive part—paraphasia and speech sound amnesia, especially with repetition—resulted from the left temporal lesion (p. 726/727).

In summary, the following main explanatory accounts have been proposed in the classic German literature:

(1) Anatomical theories:
- two routes, a phonological and a semantic, for repetition (Wernicke 1906, Heilbronner 1908);
- right hemisphere contribution to comprehension performance (Liepmann and Pappenheim 1914, Kleist 1934);

(2) phonological explanations:
- dissociation between phonemic components of words (Kleist 1905, Lewy 1908);
- disorder of the generation of the temporal sequence of speech sounds/deficient transcription of speech sound images into speech motor images (Kleist 1916);
- impaired production of the single phoneme (Kleist 1934);

- disorder of the associative basis for phonemes (Hilpert 1930);
- impaired phoneme perception (incomplete sound deafness, Kleist (1934));
- impairment of word sound control (Klein 1931);

(3) contribution of a memory deficit (Hilpert 1930).

Modern research, based on more precise linguistic models and modern imaging methods has added little to the framework of theories that have been documented in the classical German literature.

Anatomical theories

Wernicke's (1906) multiple route model was revived and elaborated further by modern neuropsychology (e.g. Morton 1980, Patterson and Shewell 1987, Blanken *et al.* 1994). Wernicke's prediction of the inability to repeat pseudo words together with semantic paraphasia with repetition was confirmed repeatedly, the syndrome has now been termed 'deep dysphasia' in analogy to deep dyslexia (Michel and Andreewsky 1983).

The anatomical theories of Wernicke (1874, 1906), Lichtheim (1885) and Kleist (1934) concerning both the arcuate fasciculus as the anatomical connection between the sensory and motor speech areas and the role of the right hemisphere in the restitution of comprehension have been reestablished by Geschwind (1965, 1973). Geschwind's papers proved very influential and resulted in a change of paradigm in behavioural neurology (c.f. Schacter and Devinsky 1997). A multitude of studies attempted to empirically support the role of the arcuate fasciculus in the pathogenesis of conduction aphasia. The topic remains controversial.

Phonological explanations

The majority of the classic cases exhibited sequences of phonemic approximations or attempts at approximation. Modern investigations confirm that conduction aphasics exhibit this type of behaviour more frequently than other aphasics (Joanette *et al.* 1980, Marshall and Tompkins 1982, Kohn 1984, Valdois *et al.* 1988). Kohn and Smith (1992) differentiate two basic classes of conduction aphasics. A nonfluent type is characterized by prominent phonemic approximations, the fluent variety by form-related paraphasia (compare table 1). In the former, the deficit is situated postlexically in the phonemic buffer, in the latter within the lexicon. According to Kohn and Smith (1992), the fluent variety occurs in the remission of Wernicke's aphasia.

The major progress made in the last 60 years lies in the availability of detailed neurolinguistic theories that form a framework for the analysis of aphasic symptoms (e.g. Buckingham (1993), Kohn (1993) for reviews, but compare also Butterworth (1993) for criticism).

The contribution of a memory deficit

Hilpert (1930) had speculated that a memory defect may contribute to the symptomatology of conduction aphasia. The modern literature contains a number of cases with a disorder of repetition that can be attributed to an auditory-verbal memory deficit (e.g. Shallice and Warrington 1977, Caramazza *et al.* 1981). These patients do not exhibit the phonological features of conduction aphasia. Further,

Strub and Gardner (1974) demonstrated that a number of aspects of conduction aphasia such as effects of word frequency cannot be explained by short-term memory deficits.

The syndrome of conduction aphasia

As has been pointed out above, none of the cases included in this review for their detail of description exhibited all symptoms of conduction aphasia, and no symptom was present in all cases. As was the case in the first two decades of this century, a deficit of repetition is today held as the cardinal symptom by some researchers, and various disorders of phonology by others. Do these controversies suggest that the concept of a 'syndrome of conduction aphasia' is obsolete? The status of syndromes and classification systems in aphasiology have been critically reviewed in the 1980s (Caramazza 1984, Schwartz 1984). Schwartz (1984) emphasized the polytypy of aphasic syndromes:

> By the criteria the taxonomists use (...), the aphasia classification has evolved from a 'typology', defined over generalized or idealized patterns shared by all members of the group, to a 'polytypic' structure in which groupings represent majority of shared characteristics. The important point about such polytypic structure is that, like the family resemblance categories of modern psychology, members need not share any single attribute, nor any patterns of attributes (p. 6).

Caramazza (1984) terms this type of syndromes 'psychologically weak' and 'defined loosely as the co-ocurrence of impairments to grossly defined functions'. In his view, groups of patients collected on the basis of such syndromatic categories are unsuitable for research into mechanisms underlying the pathological behaviour. Although the patients reviewed here probably all belong into a category 'conduction aphasia' defined along Schwartz' definition, Caramazza is obviously supported by the wide variation in symptomatology. Wernicke's patient Beckmann (Wernicke 1874) is able to repeat faultlessly, Kleist's Lina S. (Kleist 1905) is nonfluent and agrammatic, Stertz's Herrmann G. (Stertz 1914) and Stengel's Marie B. (Stengel 1933) are fluent and paragrammatic.

On the other hand following Caplan's (1991) argument discussing the value of agrammatism as an aphasic category conduction aphasia is a 'broad category' without any doubt. Nevertheless it can be taken as a collection of possible deficits resulting in production disturbances, which can be mapped on language production models, and which thus help to further enlighten the enigma of language production. In fact these categories are invaluable starting points both for linguistic theory and clinical practice of assessment and rehabilitation.

In his discussion of the validity of diagnostic categories in aphasiology, Poeck (1983) points out that the clinical syndromes of aphasia are not natural combinations of symptoms '... reflecting features of language that necessarily occur together by virtue of a disturbance of partial mechanisms within the brain system processing language as a linguistic communication device. They are, to a large extent, artifacts produced by the vascularization of the language area. They are, however, useful artifacts...' (p. 84).

Both these reflections on the status of syndromes in aphasiology and the variation in symptomatology encountered as well in the classic cases as in those seen in today's practice of neurology lead to the same conclusion: it cannot suffice

to describe an aphasic patient by a syndromatic label, but rather the patient's performance in various tasks and modalities must be assessed, analysed and documented.

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