

CARL WERNICKE AND THE NEUROBIOLOGICAL PARADIGM IN PSYCHIATRY

Frank Pillmann

Department of Psychiatry and Psychotherapy, Martin Luther University
Halle-Wittenberg, Halle, Germany

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SUMMARY

Carl Wernicke (1848-1905) was among the most outstanding and influential neuro-psychiatrists of the 19th century. He made numerous contributions to both clinical neurology and psychiatry. His network view of brain function, in some ways premature at the time, foreshadowed today's connectionist concepts. To understand the nature and importance of his findings, the scientific background during the second half of the 19th Century shall be briefly reviewed.

INTRODUCTION

Over the course of the 19th century, the large psychiatric hospitals ceased to be the driving force in psychiatry, in favor of the newly founded university hospitals. Repeatedly, psychiatry had been said to lack scientific foundation and to be dependent on other medical disciplines (e.g. Kahlbaum 1863). As a consequence, there was a strong tendency to fill university chairs of psychiatry with scientists of neuroanatomical, neuropathological or neurophysiological fame. Good examples of this include the brain pathologist Theodor Meynert (1870) in Vienna or Paul Flechsig, also with anatomical-pathological tendencies, who in 1887 became professor of psychiatry in Leipzig.

By the middle of the 19th century, enormous empirical and conceptual progress had taken place in the understanding of brain functioning. Cortical localization, which had fallen into disfavor after Gall's pseudo-science of phrenology had been discredited, found renewed interest when in Paris the surgeon and anthropologist Paul Broca localized "aphemia." In 1861 he reported a disturbance of speech resulting from a lesion of the second and

third frontal convolution of the left hemisphere. Since then, all over Europe, there was a strong interest in aphasia, the first instance of the empirically corroborated localization of a higher mental function (Hagner 1997).

In 1870, the Berlin neurologist Eduard Hitzig together with the anatomist Gustav Fritsch first reported animal studies showing that limb movements could be elicited by electrical excitation of the prefrontal cortex (Fritsch and Hitzig 1870). This finding further stimulated interest in cerebral localization and fostered the idea that brain research could contribute to the unsolved questions of psychiatry. Hitzig himself was appointed director of the University Department of Psychiatry and Neurology in Halle, where he was later superseded by Wernicke. As these examples illustrate, the 1870s, when Wernicke entered the medical profession, can well be characterized as a great period for cerebral localization.

APHASIA

Carl Wernicke was born in 1848 in Tarnowitz/Tarnowskie Góry, now in Poland but then part of Prussia, into modest circumstances. His father, a clerical assistant in the Prussian mining administration of Lower Silesia, died when his son was still a young man. Wernicke studied medicine in Breslau/Wrocław. He entered the municipal All Saints Hospital, where he served in the psychiatric department as assistant to Heinrich Neumann, associate professor of psychiatry. Wernicke was stimulated by the fascinating findings of the developing neurosciences (Lanczik and Keil 1991). He undertook a scientific visit to Theodor Meynert in Vienna, who had just begun to establish his fame as the principal authority in brain psychiatry. Back in Breslau/Wrocław, within a few months Wernicke developed an extended theory of aphasia. He collected a number of supporting cases from the wards of the All Saints Hospital and published a monograph of 72 pages: *The Aphasia Symptom Complex* (Wernicke 1874).

Virtually overnight, Wernicke assumed a leading role in aphasia research. The book opened Wernicke the opportunity to enter the Psychiatric Department of the Berlin Charité Hospital under Karl Westphal. In 1878, Wernicke's academic career suffered a major disruption when he came into conflict with the hospital administration. He lost Westphal's support and had to retreat to private neurological practice for the next several years.

The important point of Wernicke's work was not just the identification of yet another cerebral localization. It was his general approach to explaining higher cognitive functions by the interaction of spatially distributed but interconnected centers (Graves 1997). Wernicke integrated three different elements to develop his scheme of aphasia:

- the separate cortical representation of sensory and motor functions;
- the existence of association fibers;
- the concept of a reflex arc in higher cerebral functions (see Fig. 1).

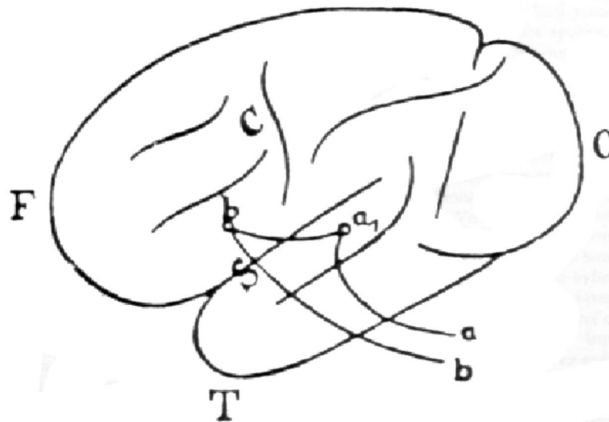


Fig. 1. Wernicke's schematic drawing from "The Aphasia Symptom Complex"

According to the scheme shown in Fig. 1, the sensory and motor aspects of an idea are stored separately in the temporal and frontal cortex, connected by association fibers running in proximity to the insular region. A lesion of the sensory cortical center leads to loss of speech comprehension and, by impaired sensor-motor monitoring via the association fibers, also to secondary motor symptoms in the form of paraphasic speech.

Some insight into Wernicke's way of thinking can be gained by an analysis of the empirical basis to his theory. As a scholar strongly grounded in scientific thinking, Wernicke strove to base his theory on clinical data, namely clinical-anatomical correlations. However, in the first and second parts of his monograph, Wernicke mainly unfolded his theoretical assumptions. In the third and last part of his monograph he lists ten case histories in order to substantiate his theory. These are summarized in Table 1.

On examination of Table 1, several points of interest can be observed:

- Only a few of the patients died and had post mortem examinations.

Table 1. Case histories from The Aphasia Symptom Complex

Clinical features	Examined	Post mortem
Sensory aphasia	March 1874	no
Sensory aphasia	Not extensively	December 1873
Conduction aphasia	March/May 1874	no
Conduction aphasia	March/May 1874	no
Predominantly motor aphasia	March 1874	April 1874
Motor aphasia and agraphia	April 1874	no
Conduction aphasia	May 1874	no
Global aphasia	March	June 1874
Motor (?) aphasia 3 years ago	?	no
Mixing up words	May 1874	May 1874

- Of the two patients diagnosed with sensory aphasia, one was alive at the time of publication. This patient presented clinically with a clearcut sensory aphasia, but the site of the lesion was unknown.
- The other patient showed a lesion more or less in the “right” location. However, this patient - according to Wernicke’s own words – had not been examined with the desirable comprehensiveness and exactness, because at that time Wernicke did not yet have available the correct concept of aphasia.
- A look at the dates when the clinical cases were seen by Wernicke (these dates are given in the histories) reveals that most of the work must already have been written when Wernicke collected the cases.

In summary, Wernicke’s way of reasoning can be described as mainly deductive. Rather than drawing conclusions inductively from observed clinical data, he started out from theoretical considerations and used the clinical material to confirm his views. This basically deductive procedure was justified by Wernicke in an explicit way. He programmatically stated that “...an exact examination, especially of psychic symptom complexes, cannot do without an already completed theory directing its way” (Wernicke 1874, p. 38).

FROM BRAIN RESEARCH TO PSYCHIATRY

From 1876 to 1885, Wernicke made his living as a practicing neurologist in Berlin. During his time in private practice, Wernicke’s productivity did not cease. He produced various neurological papers, including the description of the hemianopic pupillary response and wrote a well received *Textbook of Brain Diseases* (Wernicke 1881). It was this textbook that contained the description of “polioencephalitis haemorrhagica superior”, based on the examination of 3 cases – later called Wernicke’s encephalopathy. He inaugurated sterile ventricular puncture and external CSF drainage for the treatment of hydrocephalus (Aschoff et al. 1999), and in 1882 he reported the first case of surgical treatment of a patient with a brain abscess (Wernicke & Hahn 1882).

Finally, when Neumann died, Wernicke followed him in his position as head of the psychiatric department of All Saints Hospital and as professor of psychiatry and neurology (Kiejna & Wojtowicz 1999). Soon he set up a laboratory and began to fight for the funding of a neurological outpatient clinic, which was finally opened in 1887. The assistant in charge of the outpatient clinic was Ludwig Mann. In 1889 Wernicke had characterized the typical posture and gait in cerebral hemiplegia in a short article (Wernicke 1889), but he left it to Mann to publish in 1895 an in-depth analysis of what is since known as the “Wernicke-Mann” type of hemiplegia (Mann 1896). In the 1890s Wernicke embarked on a project to design a photographic atlas of stained brain slices. For years to come he and his assistants devoted a good deal of effort to handling the immense technical problems involved in producing, staining, and photographically reproducing whole-brain slices. In 1903 the last of three volumes was finally completed (Wernicke & Foerster 1903).

In Breslau/Wrocław (and later in Halle), Wernicke had many important pupils, who later made their careers in neurology or psychiatry, including Otfried Förster, Karl Bonhoeffer, Hugo Liepmann, Karl Heilbronner and Karl Kleist. During these years, Wernicke became more and more devoted to one pre-eminent goal: to base psychiatry on a neurobiological foundation.

Wernicke's interest in the neurobiological foundation of psychiatry was a logical extension of his earlier activities. Already the *Aphasic Symptom Complex* had been subtitled "A psychological study on an anatomical basis." Wernicke's aim was to give a neurobiological explanation of higher brain function and its disorders. In 1879, only 5 years after the publication of the *Aphasic Symptom Complex*, Wernicke gave a programmatic speech at a meeting of the Berlin Medical Society "On consciousness." He advocated a strictly scientific explanation of mental activity. Further pursuing this goal, from 1894 to 1900 he published successive parts of his textbook, *Outline of Psychiatry*. This book is an heroic and – in its consequence – unprecedented attempt to base a psychiatric textbook wholly on recent brain research.

For the explanation of psychiatric diseases, Wernicke expanded the assumptions that already underlay his theory of aphasia. The cerebral cortex is

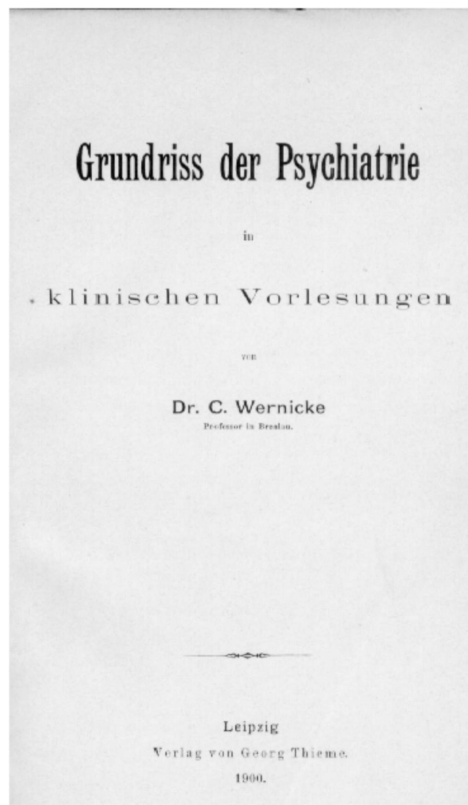


Fig. 2. The Outline of Psychiatry, original cover

thought of as the organic substrate of mental processes (of consciousness); ideas are cortically represented with separate localization of motor acoustic, visual etc. aspects in specialized cortical centers. Communication between these centers takes place via anatomically defined association fibers. Complex phenomena, e.g. the initiation of motor behavior by a voluntary act, can be explained by the concept of a “reflex arc” (Figure 3). This mental reflex arc is differentiated from simple, elementary reflexes by the addition of the association apparatus, in which individual experiences, ideas and images are stored. In analogy to the aphasic disorders, mental disorders were thought of as disorders of the “organ of association” (cf. Meynert). The basic pathomechanism was identified as the interruption of association fibers between higher cortical centers, a phenomenon called “sejunction” (i.e. disjunction or interruption) by Wernicke.

Wernicke was not able to assign definite localizations to the cortical representations of higher mental categories. For a more detailed classification of mental diseases Wernicke had to resort to more abstract categories. For the time being, these categories could only be connected to anatomical structures in a vague way. His classification differentiated the representation of the external world, the representation of the individual’s own body, and the representation of the self. The three corresponding fields of consciousness were called “allopsychic,” “somatopsychic,” and “autopsychic.” According to the respective domain showing psychopathological alterations, the psychoses were classified as “allopsychoses,” “somatopsychoses,” and “autopsychoses.”

Wernicke’s pathogenetic considerations were not confined to the three categories mentioned. He also considered further pathogenetic mechanisms linked to specific diagnoses (see Table 2 and section 5). In this scheme, for example, a disturbance (disconnection or sejunction) of higher motor fibers would lead to a psychosis predominantly characterized by motor phenomena. Such a disorder was named by Wernicke “motility psychosis.” He also

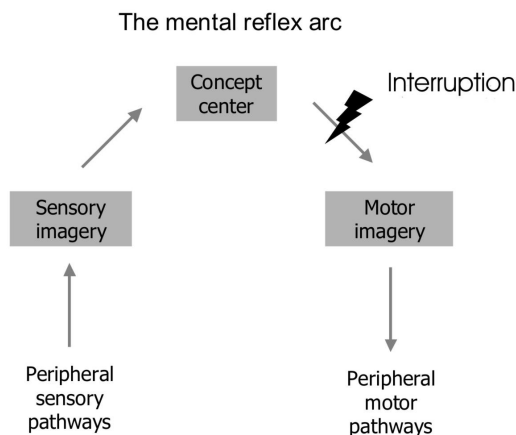


Fig. 3. The „Mental Reflex Arc” according to Wernicke

Table 2. Some of Wernicke's new diagnostic entities

- Anxiety psychosis
- Motility psychosis
- Expansive autopsychosis due to autochthonous ideas
- Autopsychosis
- Allopsychosis
- Somatopsychosis

described a disorder due to pathological irritation of association fibers, leading to the sudden appearance of erroneous thought contents, called autochthonous ideas. Thus a new disorder was born of the typically Wernickean name: "expansive autopsychosis due to autochthonous ideas."

One very important feature of Wernicke's nosological entities was that etiology was not a defining element. Just as a localized neurological syndrome could be caused by brain infarct, trauma or a tumor, in the same way a psychiatric disorder such as motility psychosis could have different etiologies, such as syphilis or alcohol. By excluding etiology from his definitions of disorders, Wernicke remained true to his pathological model, but differed markedly from his contemporaries, especially Kraepelin, who explicitly included etiology in his concept of natural disease entities.

In handling the political affairs of the clinic, Wernicke was less fortunate than in his research. There were long-standing conflicts of interest between the municipal authorities and the royal university. Wernicke, in his harsh and uncompromising way, more and more set the authorities against him; at the same time, support from the royal ministry in Berlin was half-hearted. In the course of this conflict, Wernicke first had to give up the function of Director of the clinic; later, he was denied the right to use patients for teaching purposes.

THE CASE NOTES IN HALLE

This state of affairs had lasted for several years when Wernicke was offered the chair of psychiatry and neurological diseases at the University of Halle-Wittenberg. Gratefully he accepted. In Halle, Wernicke initiated the use of brain puncture for the diagnosis and localization of brain tumors, and thus continued a neurosurgical tradition started by his predecessor Eduard Hitzig. He also recruited assistants for histological and pathophysiological studies and initiated psychopathological studies. After several years, during which Wernicke had no access to inpatients for treatment, teaching or research, he was now again the director of a full-scale, well organized university hospital. He saw a broad range of psychiatric and neurological patients and was able to apply his biologically based diagnostic concept within the setting of a busy university hospital.

The understanding of Wernicke's diagnostic concepts could be enhanced by information on the factual use of the concepts in his clinic. Fortunately, in Halle most case notes from Wernicke's time are well preserved. We therefore

used this material to examine how Wernicke's specific approach to diagnosis was used in clinical practice (cf. Pillmann et al. 2000). In particular, it is possible to ask whether Wernicke's nosological concepts can be validated by clinical data. Moreover, it seems to be worthwhile to examine a special group of disorders, namely "anxiety psychosis," "motility psychosis," and "expansive autopsychosis due to autochthonous ideas." In respect to these conditions it is of interest to note to what extent they actually represent predecessors of the later so-called "cycloid psychoses," as is commonly assumed.

All together, 889 case notes from the time of Wernicke's directorship could be evaluated. They were all drawn up under the supervision of Wernicke, most were initialed by him, and many showed corrections by his hand (Fig. 4). Diagnosis at admission, discharge diagnosis, outcome and demographic information were recorded in the case notes in a standardized form according to the usage of the time. The appearance of one of these files is shown in Fig. 4. A header contained in standardized form data concerning admission, date and mode of discharge, diagnosis, age, and some other data. Often both a nosological diagnosis, such as motility psychosis, and a specific etiology is recorded. In this case we have motility psychosis on a hebephrenic basis.

Of the 889 evaluated case records, 140 cases (15.7%) concerned patients with neurological diseases (peripheral palsies, apoplexy etc.), 564 (63.4%) concerned patients with psychiatric diseases (psychoses, neurasthenia etc.) and 167 (18.8%) could be allocated to both categories (epilepsy and psychosis, progressive paralysis). Another 18 (2.0%) suffered from other, mainly medical conditions. Thus the great majority of patients had at least one

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Psychiatrische und Nerven-Klinik der Universität Halle a. S.

Name: _____ Geschlecht: _____
 Alter: 31 J. Wohnort: Gotha Heimat: _____

Aufgenommen: 13. Juni 04 Entlassen: 1.8.04.
 Vorläufige Diagnose: Akinetische Motilitätspsychose (hebephrenische Etiologie) Definitive Diagnose: ebd.
 Bemerkungen: Beobachtete Schweregrade
 Gehfähig auf Nietleben

Datum	Ordination
13. Juni 1904	
Bemerkung zur 22. J. zeitlich krankheitlich	

Fig. 4. Header of the case notes of a 31-year-old woman and details from the draft for a standardized clinical report preceding the referral of this patient to a state asylum. The writing is in Kleist's hand and corrected by Wernicke (italics). Name:... / Occupation: - / Age: 31 years / Place of residence: Gotha / Place of birth: - / Admitted: 13 June 1904 / Discharged: 1 August 1904 / Preliminary diagnosis: Akinetic motility psychosis (hebephrenic etiology) / Final diagnosis: same / Remarks: Improved to Nietleben [state asylum][...]

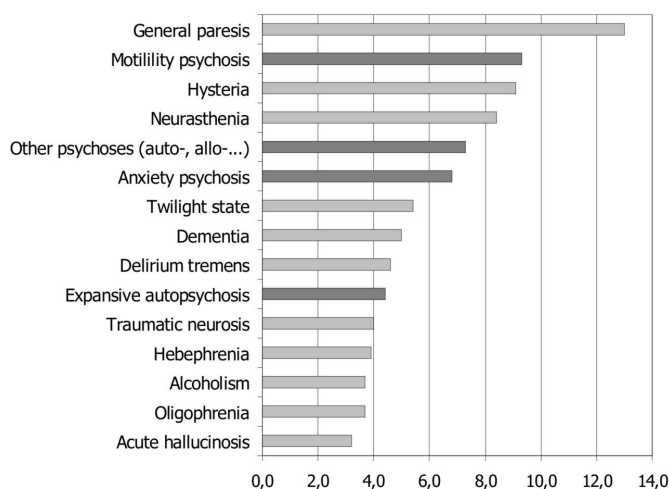


Fig. 5. The 20 most frequent diagnoses in 723 psychiatric admissions

psychiatric diagnosis. More than 45 different psychiatric diagnoses were used in the case records; the 20 most frequent are given in Figure 5.

Anxiety psychosis, motility psychosis and expansive autopsychosis due to autochthonous ideas were each diagnosed in 4.4-9.3% of psychiatric admissions. Although many 'traditional' diagnoses, such as neurasthenia, delirium, twilight states etc., continued to be used, the diagnoses of catatonia or dementia praecox were never made. A diagnosis of hebephrenia was made in 28 cases. Although Wernicke did not use his rival Kraepelin's term *dementia praecox*, he diagnosed hebephrenia with the connotation of a deteriorating disorder with an unfavorable prognosis and an outcome in dementia.

WERNICKE AND THE "CYCLOID PSYCHOSES"

With respect to psychiatry, Wernicke's name today is associated mainly with the Wernicke-Kleist-Leonhard school of psychiatry (Ungvari 1993). Karl Kleist (1879-1960) worked as a resident physician during Wernicke's directorship in Halle; later he became head of the university department of psychiatry in Frankfurt am Main. In 1924 he coined the term "cycloid psychosis" (Kleist 1924). Kleist shared his clinical interests with Karl Leonhard, his collaborator in Frankfurt from 1936 to 1950. In his 1957 textbook *Classification of Endogenous Psychoses* Leonhard cemented the nosological system largely identified with the Wernicke-Kleist-Leonhard school (Leonhard 1957). Wernicke's anxiety psychosis, motility psychosis and expansive autopsychosis have been regarded as predecessors of the "cycloid" psychoses introduced by Kleist in 1924 and reformulated by Leonhard in 1957 (Kleist 1924, Leonhard 1957, Leonhard 1961, Perris 1995). In Leonhard's system cycloid psychoses are divided into anxiety-elation psychosis, motility psychosis and

confusional psychosis (Leonhard 1961). They have in common a phasic course, a bipolar appearance, and, most important, they are thought invariably to remit without residual symptoms (Leonhard 1961). The concept of cycloid psychosis continues to be used and has been integrated into the ICD-10 concept of acute and transient psychotic disorders (Perris 1988). Though there has been some continuity in the use of the terms, anxiety psychosis, motility psychosis and expansive autopsychosis were not grouped together by Wernicke and he did not associate them with a good prognosis as decidedly as did later authors (Franzek 1990).

At present our knowledge of Wernicke's clinical classification derives primarily from the descriptions in his textbook and published selected case histories (Wernicke 1899/1900). *Anxiety psychosis* is characterized by Wernicke as a psychotic disorder lasting several weeks to several months with fearful affect and anxious ideation referring to the self ("autopsychic") or the body ("somatopsychic"). At its climax patients may show verberation, acoustic hallucinations, delusions of inferiority and delusions of reference. The subjective feeling of insufficiency that characterizes depression ("affective melancholia" in Wernicke's terminology) is lacking in anxiety psychosis (Wernicke 1900). Wernicke principally allows the occurrence of anxiety psychosis in the context of such specific etiologies as progressive paralysis and alcoholism (Wernicke 1900, Wernicke 1899/1900). The prognosis is described as favorable.

According to the case records examined, female patients predominated in anxiety psychosis, comprising 61.2% of the patients. This was markedly different from the gender distribution in the total population of psychiatric patients and from patients diagnosed as suffering from hebephrenia, only one third of whom were female. A specific etiology was only recorded in 32.7% of admissions with anxiety psychosis (Fig. 6). The most frequently stated etiology for anxiety psychosis was paralytic etiology, followed by alcohol consumption

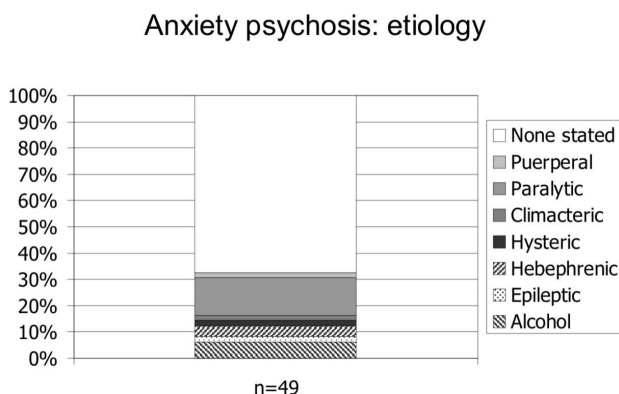


Fig. 6. Etiology as stated in the case records of 49 patients with anxiety psychosis

Motility psychosis is defined as a psychotic disorder in which all symptoms follow from pathologically altered motility. This entity was of high theoretical importance to Wernicke. He used motility psychosis as an example for the disconnection of higher centers in the brain and the motor association cortex. Wernicke described several forms: a hyperkinetic form, a hypokinetic form, and combinations of these called “complete” motility psychosis. In hyperkinetic motility psychosis young women predominate and onset is sudden, within a couple of days, but sometimes with prodromes. A wealth of motor phenomena affecting expressive and reactive movements are described by Wernicke. Prognosis is favorable. Akinetic forms are sometimes of ‘hebephrenic etiology’ and their prognosis is somewhat less favorable. This also applies to complete motility psychosis.

In the case records of patients with motility psychosis, as in anxiety psychosis, female patients predominated (40 of 67 case records, 59.7%). This again was markedly different from the gender distribution in the total population of psychiatric patients and from patients diagnosed as suffering from hebephrenia. This finding is remarkable because later concepts of such related disorders as cycloid psychosis or the acute and transient psychotic disorder of ICD-10 share this disproportion of female patients. Fig. 7 shows the etiologies held to be responsible for motility psychosis.

Most of the cases were not assigned a specific etiology. Some were associated with childbirth, a finding repeatedly replicated by later authors. Some cases were associated with progressive paralysis and some were judged to be of hebephrenic origin. The last statement sounds somewhat odd, because hebephrenia had already been used by Wernicke as a diagnostic term. Indeed, Wernicke is ambivalent on this issue. His double use of hebephrenia and hebephrenic both as a nosological term and as a form of etiology reflects the difficulty involved in accommodating endogenous psychoses in his neuropathologically oriented system.

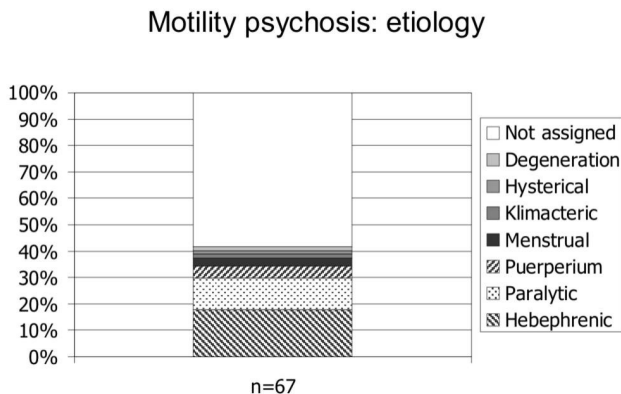


Fig. 7. Etiology as stated in the case records of 67 patients with motility psychosis

Expansive autopsychosis: etiology

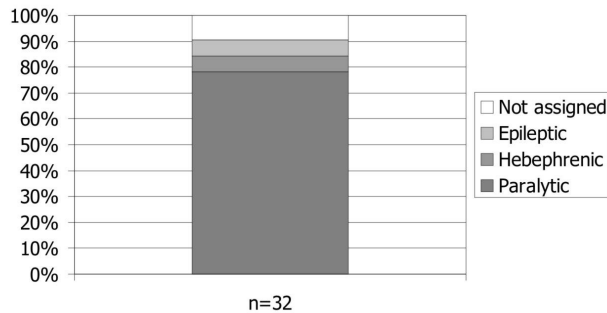


Fig. 8. Etiology as stated in the case records of 32 patients with expansive autopsychosis

“Acute expansive autopsychosis due to autochthonous ideas” was defined by Wernicke as a disorder characterized by the acute occurrence of autochthonous ideas, suddenly appearing ideas caused by irritation of association fibers. They are judged alien by the patient and lead to secondary hallucinations and delusions of interpretation. The course is intermittent and the outcome surprisingly favorable within a few months. Usually ideas of grandeur and feelings of elation are prominent, partly in combination with anxiety.

Our analysis of the 32 cases of expansive autopsychosis in Wernicke’s material revealed that the gender distribution in expansive autopsychosis was quite different from that of the disorders mentioned above, with a strong male preponderance: 30 of the patients (93.8%) were male (!). The spectrum of suspected etiologies was also rather specific, as can be seen in Fig. 8.

Expansive autopsychosis stands out as being most frequently of a stated specific etiology, namely paralytic. Most of the patients therefore suffered from progressive paralysis.

This again is an interesting finding for the following reason. The supposed pathogenesis of the disorder, i.e. the occurrence of nonsense associations leading to ideas of thought insertion or voices, sounds like a modern neurobiological explanation of first rank symptoms in schizophrenia. Instead, expansive autopsychosis is nearly exclusively diagnosed in male paralytic patients. The diagnosis seems to have been preferentially made in manifold psychosis in the presence of progressive paralysis.

SO, DID THE THEORY WORK IN PRACTICE?

Diagnostic patterns in the material analyzed confirm that Wernicke’s diagnostic scheme did not contain a well-structured system of mutually exclusive nosological categories and subcategories. Instead, there was a multitude of diagnoses that could not easily be grouped into major categories. These

diagnoses included both traditional terms (e.g. “hysteria” and “twilight state”) and typically Wernickean terms (e.g. “allopsychosis” and “motility psychosis”) without any obvious differences. Nosological distinctions such as the division into auto-, somato- and allopsychoses occur in all combinations. They thus seem to be heuristic ordering principles, but do not lead to a system of distinct nosological categories.

Nevertheless, some diagnostic prototypes described so vividly by Wernicke in his textbook can be identified in the case records, and there are common sociodemographic and clinical features. This is especially true of anxiety psychosis and motility psychosis, which may be compared to the later concept of cycloid psychosis.

As previously noted, Wernicke did not use his rival Kraepelin’s term *dementia praecox* (Kraepelin 1896), but he diagnosed hebephrenia (Hecker 1871) with the connotation of a deteriorating disorder with an unfavorable prognosis and an outcome in dementia. The distinction between motility psychosis and hebephrenia, however, is not always clear. Not only was the short term-prognosis of motility psychosis comparably unfavorable, but many cases of motility psychosis were judged by Wernicke to be of “hebephrenic etiology” (cf Fig. 7). This ambiguous use of ‘hebephrenia’ both as a syndromal and an etiological term may reflect Wernicke’s ambivalence on the question.

“Expansive autopsychosis due to autochthonous ideas” is characterized by revelation experiences and elated mood. Pathogenetically Wernicke linked it to the sudden emergence of pathological associations. In practice, expansive autopsychosis due to autochthonous ideas was mainly diagnosed in the context of neurosyphilis, and as a pure “endogenous” disorder was extremely rare in our sample. This indicates that in clinical practice etiology may have influenced diagnostic decisions more than Wernicke intended.

WHAT FOLLOWED

After only 14 months, these activities came to an abrupt end when Wernicke suffered a severe accident on a bicycle trip into the forest of Thuringia. He died the next day on June 15, 1905, at the age of 58. His aim of a pathophysiologically based classification of psychiatric disorders was to some extent carried on by some of his pupils, mainly by Karl Kleist. In his time, Wernicke’s biaxial diagnostic system was the most advanced application of neurobiological thinking to questions of psychiatry, but it never gained ground in mainstream psychiatry. On the whole, the strict concept of a biological psychiatry envisioned by Wernicke was taken over by the more clinical approach of Kraepelin. Wernicke’s neurobiologically based psychopathology came to be denounced as “brain mythology.” In fact, he himself had never been able to complete his theory into a coherent whole. As the analysis of the case notes showed, in practice Wernicke had to adopt a pragmatic use of diagnostic terms not always true to his original intentions. With its

emphasis on neuropathology Wernicke's theory encountered difficulties in accommodating the problem of "endogenous" psychoses.

Still, driven by a fervent "need for causality" and talents for both conceptualizing and observation, Wernicke was among the most outstanding and influential neuropsychiatrists of the 19th century. He left numerous contributions to both clinical neurology and psychiatry. Moreover, his network view of brain function, in some ways premature at the time, foreshadowed today's connectionist concepts. In a period when the expanding methods of brain research again increasingly influence our concepts of mental disorders, and where functional brain imaging revives old hopes for the localization of complex mental functions, Wernicke's legacy seems remarkably up-to-date. As biological findings lead to problems with traditional nosological boundaries, it seems well worthwhile to look back at Wernicke's attempt to create a truly integrative neuropsychiatry.

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Address for correspondence:

PD Dr. med. Frank Pillmann
Department of Psychiatry and Psychotherapy
Martin Luther University Halle-Wittenberg
Julius-Kühn-Straße 7
06097 Halle
Germany
frank.pillmann@medizin.uni-halle.de

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