besides syphilis. The pathological and technical details in this paper are scanty, and in light of the patient’s history of sudden onset of speech difficulty and “right facial deficit” one wonders if focal pathology could have been overlooked. The disorder Pick’s Disease, named after him, followed the description of a specific pathology and a specific cell change nineteen years later by Alzheimer.

About the time of Pick’s paper (1892) there were significant contributions by Blocq and Marinescu (1892), who described scattered silver staining plaques in the cortex; Binswanger (1894), who described a subcortical encephalopathy due to arteriosclerosis; and Alzheimer (1895, 1898), who described cortical changes of atrophy and focal cell changes in relation to arteriosclerosis and blood vessel thickening. Alzheimer did not feel these changes could account for senile changes and separated arteriosclerotic dementia from other cases. It is in this setting that we should read the simple, direct, and clear paper of Alzheimer, which clearly recognizes a unique pathology and puts the changes into their appropriate perspective. This paper was given at a clinical meeting in 1906 and published in 1907. It is clear that Alzheimer knew he was describing something specific which should be separated from other cases. The subsequent paper by Bonfiglio contributes little except for its excellent discussion and confirmation of Alzheimer’s findings. That this was not purely unbiased is obvious when one notes that the brain was given to Bonfiglio by Alzheimer. It apparently details the findings described by Alzheimer the previous year in a syphilitic male. Except for his observation that the plaques originated in nerve cells and for some excellent details, it is probably only of historic interest.

By 1910 Perusini had separated senile dementia from other mental illnesses, and Kneppel had suggested that the disorder be named after his associate, Alzheimer. Subsequently, Simonowicz (1911) described the granulovascular degeneration, and in 1914 Divry described the amyloid changes.

It is always interesting to review original articles when one has been using terms such as “Pick’s Disease” or “Alzheimer’s Disease.” Alzheimer clearly made not only major contributions in descriptive pathology but in the conceptual separation of a confusing group of diseases. Neither Pick’s nor Bonfiglio’s contribution appears to warrant the historical reward it has been given.

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**On the Relation Between Aphasia and Senile Atrophy of the Brain**

A. PICK

**Whereas it is now known** that aphasia and other clinical focal signs often found in early stages of general paralysis of the insane are only occasionally complicated by more prominent local anatomic involvement, the same has so far not been considered with respect to the underlying anatomical basis of simple, uncomplicated senile dementia, namely, cerebral atrophy. This is due in large part to the opinion that all aphasia which accompany senile dementia, with the exception of the so-called amnestic form of aphasia, are complicated by definite focal lesions other than senile atrophy. The contrasting viewpoint is most clearly enunciated by Wernicke in a work where the relationship between “aphasia and psychic disorders” is discussed, when he maintains—although mentioned from a different point of view from the present one—that general paralysis is the only mental disease which during its course can lead to cortical and subcortical focal manifestations and, therefore, it holds a unique middle position between psychosis and organic brain disorders. The following article is meant to show that the same holds true for the cerebral atrophy which underlies senile dementia, thus contributing to the effort to bring neuropathology and psychiatry into closer relationship, making the latter understandable in medical terms.

**Note:** Translated by W.C. Schoene from: Pick, A., “Über die Beziehungen der senilen Hemiatrophie zur Aphasia,” Psychiatrische Wochenschrift 17, 16 (1892), 168-67. This translation could not have been done without help from Ingo Winzer and Helmut G. Renke.
On November 11, 1891 the 71-year-old August H. was admitted to the clinic with the history that following two years of progressive mental deterioration he had begun to show rage and had threatened his wife with a knife. Historical review revealed the following: a family history of apoplexy; for the past three years the patient, who had until then been in good physical and mental health, exhibited progressive memory loss; at the beginning of November 1889 he suddenly became “unconscious” after dinner for several minutes and experienced a similar attack the following day; thereafter he is said to have “talked crazy” for a while, having been unable to express himself properly; he subsequently suffered an intestinal illness which left him much weakened. In January of 1890 he suffered a severe case of influenza, the effects of which lingered until April; during the initial fever he was delirious, misidentified persons and manifested speech difficulties which, according to his wife, became pronounced later; during recent days he threatened his wife, crying “I’ll kill you.” Aside from this he has been quite infantile of late, playing with suspenders and spoons.

On admission to the clinic the patient demonstrated a profound memory loss and throughout his hospitalization lay quietly on his bed without being very much concerned with his surroundings; he is of medium height; shows pronounced senility; except for a barely perceptible facial deficit on the right side, no motor or sensory disturbance; somewhat heightened knee reflexes; ankle clonus; temperature normal, pulse 72; emphysema and bronchial catarrh. Hearing not noticeably impaired. The patient showed a profound speech difficulty of an apathetic character; speech comprehension is significantly, though not totally, destroyed; simple questions about his general circumstances are understood; other questions he does not understand at all.

Speech: The patient possesses a considerable vocabulary and speaks a lot; however, although sentences are sometimes correct when dealing with simple matters, they generally are nonsensical, partly because of the incorrect arrangement of words, partly because the words themselves are unintelligible. This is due to times to transposition of consonants; e.g., he says “colmore” instead of “locomotive,” “redeklasten” instead of “kleiderkasten.”

**Question:** “What is your name?”
**Answer:** “August H.” (correct)

**Question:** “How old are you?”
**Answer:** “Ten or 12, I don’t know. I don’t understand, the young girl, the stone in the hall.”

**Question:** “What is your job?”
**Answer:** “Karkal... Kakar... a man who works with gold... my God” (angrily)... (the rest is entirely unintelligible).

To a random question he answers: “I understand, but I must stand up, it’s leaving my head, my head is made of iron, that’s how the beginning starts, I don’t know that, I can’t start, I don’t have it here.”

He partially recognizes objects shown to him, but often describes them incorrectly:

- Hat: “Felt.”
- Matchbox: “Filter paper.”
- Pocket knife: “Umbrella.”
- Given a woolen glove, he rubs the palm of his hand and says: “Wool.”
- Spoon: “I know that, that’s on some coffee.” (places the spoon in his mouth)

He repeats phrases correctly if they are spoken to him slowly; however, as soon as the speed quickens he reverts to the above-described form of spontaneous speech. Apparently, in the first example the patient can follow the individual syllables, whereas in the second example he immediately forgets what is said and continues spontaneously.

He reads aloud slowly, with difficulty and usually incorrectly. Oslede is read once as: “Oste... ost... u te te, Ostus, tentinde.” Another time: “Otto, Osto, Otto, tres, en, am de, el.” Prager Abendblatt: “Parger Pagelage Abeangust.” Prager Tageblatt: “Pag... tag... tatalak, te tult... ta... tel... tel... tel...” He reads written names correctly, rapidly skimming over them: Goldarbeiter: “August” (his Christian name!) “Gust, gold...goldvater.” He identifies individual letters partly correctly; he recognizes numbers, individual ones correctly, polysyllabic ones partly incorrectly, 1891=1848, 25=85.

Script is apparently not at all understood. Spontaneous writing, copying, and dictated writing all appear to be impaired in a similar manner, i.e., the patient often begins correctly, writes two or three correct letters, then attains regularly a series of illegible signs resembling an “L,” at which time the patient excuses himself, saying that his hand trembles too much.

The above-mentioned findings remained fairly constant throughout the patient’s hospitalization until the last days of November, when his pulmonary symptoms worsened; the patient died on November 27 of pulmonary disease, having become progressively more apathetic.

The clinical diagnosis in the preceding case was made without difficulty after the speech disorder was recognized. Since our main concern is with the pathological anatomical diagnosis of the secondary symptom, namely, the speech disorder, the primary diagnosis, that of senile dementia, will not be discussed.

In observing the speech disorder we lay greatest emphasis on the fact that we are not dealing with a disorder which can be exclusively, or even primarily, attributed to the simple amnestic effects of the senile process, but, rather, it more closely parallels those which are the result of focal
lesions; it resembles those disorders which Wernicke-Lichthein described as transcortical sensory aphasia insofar as we could determine that the patient’s primary symptoms were a loss of understanding of speech and writing, paraphasia and partially retained ability to repeat speech.

More difficult than the clinical diagnosis was the pathological anatomical diagnosis, which had to determine both the site and nature of the disease.

If one held to the prevailing readily available opinion expressed at the beginning of this article, it would seem that the vague prehospitalization history could be weighted to indicate that the patient had suffered one or more apoplectic attacks which left circumscribed focal softening in the left temporal lobe, leading to what was recognized as a primary sensory speech disorder; it need only be briefly mentioned that this localization accounts for the lack of other motor manifestations; it would also not be too bold to associate the recrudescence of the symptoms after the influenza with such localized foci.

Although the idea of focal softening would agree well with currently accepted views, we considered this only as a secondary possibility; rather, our diagnosis before considering the autopsy was Atrophie cerebri praeципie haemisphaerii sin. in regione gyri primi lobi sphenoidalis, with the latter region only considered a secondary possibility; this was based on the prior clinical observations which appear in the Archiv für Psychiatrie und Nervenkrankheiten (vol. 23, no. 3), and also on the fact that the history indicated a gradual development of the patient’s speech disorder to its final degree. The preceding statement, viewed in the light of our published observations on transitory postepileptic sensory aphasia, suggests the possibility that a similar mechanism, though much slower, had been in operation here, and that at a certain point in time a more or less circumscribed type of aphasia could result from a simple circumscribed atrophic process.

Autopsy performed on the 28th of November by my colleague, Dr. Chiari, yielded the following with respect to the brain: the scalp soft and pale, the skull 54 cm in horizontal circumference, thickened, compact; the dura tightly stretched, its sinus slightly filled with fluid, slightly coagulated blood; on the inner surface of the pachymeninges over the surface of the cerebral hemispheres delicate newly formed connective tissue membranes; the inner meninges somewhat thickened, quite edematous, of medium blood content, everywhere easily removable. The walls of the basal vessels unevenly thick. The weight of the brain after removal of almost all meninges (except those covering the cerebellum) was 1150g; the right cerebral hemisphere 500g; the left one 470; the gyri of the cerebral hemispheres clearly narrowed, the atrophy of the gyri of the left side, especially of the lobus temporalis sin., clearly more pronounced than the corresponding ones on the right side; brain substance generally tough, pale, moist. The ventricles enlarged, the ependyma thickened, partially granulated, no focal lesion demonstrated; fresh teased preparations of white matter from the lobus temp. sin do not reveal granulated cells.

If we now advance this new viewpoint of the diagnostic significance of the focal signs in senile atrophy of the brain, it obviously lacks that broad base of case documentation which is the foundation of the prevailing opinion; nevertheless, I find in the literature, which has not yet been thoroughly examined, individual cases which entirely support the previously discussed viewpoint, not only for aphasia but also for other localizing symptoms.

The first case was reported by Bevan Lewis in his Textbook of Mental Diseases, 1889, p. 411. A 52-year-old man who eleven months previously had suffered nystagmus and convulsions became ill with what appeared to be senile dementia, having previously shown progressive weakness in the right extremities and speech difficulties; he showed amnestic speech difficulty and ataxic aphasia. Autopsy revealed: brain weight 1270 g; right hemisphere 555, the left 515; the right frontal lobe 327; the left 198. Cloudiness of the meninges, perhaps a slight circumscribed adherence of the meninges to the left frontal and marginal gyri; high-grade atrophy in the frontal and parietal regions, especially on the left side; no focal softening.

The second case taken from the literature comes from the observation by Magnan, which is related to Ville. Skwurtzoff ("De la céphale et de la surdité des mots dans l’aphasie," 1881, p. 100) in the chapter on etiology by the few words: "We studied an aphasic patient, where subsequent autopsy revealed atrophy of the entire left hemisphere." The actual clinical observations were as follows: "37-year-old woman who apparently suffered a progressive psychic disorder after a profuse bleeding at age 27; following a psychic shock she became panic-striken, complained of pain in the right leg, eventually lost the use of nouns, exhibited paraphasia, later only affected speech (she could, however, complete the words of a song sung to her, with which she was familiar) and high-grade dementia, dulling of the senses, muscle weakness without paralysis.

Autopsy reveals a brain weight of 935 g; 770 of which is cerebrum, 415 from the right, 355 from the left hemisphere, 165 from the cerebellum and hydrocephalus externus. The thickened and edematous leptomeninges are easily removed save for some adhesions at the tips of the temporal lobes, especially on the left. The gyri of the left hemisphere are markedly atrophic, except in the motor region; microscopic investigation
of cortical grey matter from the third left frontal gyrus and temporal gyrus revealed granular degeneration of the ganglion cells, isolated granule cells and fat granules in the vessel walls.

Although there is no senile but, rather, simple atrophy, paralysis can be eliminated, so that this case, as well, represents support for our thesis that simple progressive brain atrophy can lead to symptoms of local disturbance through local accentuation of the diffuse process. In passing it should be mentioned that this evidence will significantly improve the understanding of other manifestations caused by the diffuse process; just as the understanding of aphasia is also advanced generally by considering the development of the aphasia in the presented case. We do not wish to elaborate further on the pathological-anatomical questions related to the above discussion; these questions should be placed side-by-side with Lissauer's recent attempts to explain similar conditions in general paralysis.

A Unique Illness Involving the Cerebral Cortex

A case report from the Mental Institution in Frankfurt am Main.*

A. ALZHEIMER

THE CLINICAL COURSE and path ology of this distinctive process separate it from the known neurologic disorders.

The patient initially presented at fifty-one years of age with jealousy of her husband. Rapidly progressive memory loss soon followed. No longer finding her apartment suitable, she dragged her furniture back and forth and concealed it. She began to believe that others wanted to kill her, and she would scream out loud.

Following institutionalization she appeared totally bewildered. She was disoriented as to time and place and occasionally stated that she did not understand events around her. She treated her physician as a guest, excused herself and said she was not finished with her work. Following this she would scream aloud that he was trying to stab her with a knife, or indignantly turn him away, fearing that he would violate her. She was intermittently delirious, dragged her bedding about, called for her

*The clinical examination and central nervous system autopsy were performed by Dr. Sidl, director of the institute.