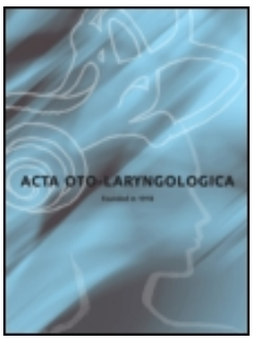

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On the hearing Sphere

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On the hearing Sphere [1].

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The starting point of this article is a paper, read at the Psychological Congress in Rome 1905 with the title "Surdité verbale pure" (Atti del Congresso di Psicologia. Roma 1906, p. 177).

The case at that time not being thoroughly examined microscopically, the paper was not published in the acts of the Congress, and the manuscript was laid aside.

Reclaiming a certain priority, I publish here for the first time my original paper, read at the Congress in French, on the purpose of showing that the ideas contained in the present text will not differ from those of the original paper of 1905. In the meantime, different researches on the matter have been published as:

Campbell, The localisation of cerebral function. Cambridge 1905.

Flechsig, Some communications in Neurol. Centralblatt 1908.

Mingazzini, Anatomia clinica. Roma 1913.

Liepmann's numerous papers.

Marie's and Déjérine's interesting discussions on Aphasia 1906—07.

Déjérine: *Surdité verbale pure.* Sémiologie. p. 414.

I. Sur la surdité verbale pure.

(Communication, faite au Congrès de Psychologie à Rome le 26 Avril 1905.)

Depuis 40 ans les études sur le cerveau s'occupent tout spécialement de la question de la localisation. On a réussi

[1] An auto-extract from the original paper, written in German: Journal f. Psychologie und Neurologie. Herausgegeb. v. Forel u. Oscar Vogt. redig. v. K. Brodmann 1918. Bd 22. Ergänz. Hft. 3.

à tracer les grands contours généraux des centres sensibles, sensoriels et moteurs. Le problème actuel est de préciser les limites de ces centres et de déterminer leurs fonctions. Ce problème a pour but d'examiner, s'il y a des centres psychiques séparés des surfaces sensorielles, et si ces centres psychiques ont des valeurs différentes, c. à. d. s'il existe des centres séparés pour les procès psychiques plus ou moins simples ou compliqués. Dans mon étude sur le centre de la vision et sur les voies optiques, publiée il y a 15 ans (1890, au Congrès internat. de méd. à Paris, 1900), j'ai prononcé mon opinion à ce respect, et par opposition à l'avis, alors au cours, j'ai démontré que le centre de la vision c. à. d. la surface qui reçoit en premier lieu l'impression de la lumière, est restreinte et qu'elle ne se répand pas sur le pli courbe et sur la corticalité entière du lobe occipital. Ce centre est limité à une très petite superficie et se trouve principalement placé dans la scissure calcarine, savoir la partie du lobe occipital, marquée par la strie de Gennari, comme je l'ai déjà répété tant de fois. En outre, j'ai réussi à démontrer qu'il y a sur cette corticalité limitée une projection de la rétine, vu que la lèvre supérieure de cette scissure correspond à la moitié supérieure de la rétine, le fond de la scissure correspond à la ligne horizontale et la lèvre inférieure à la moitié inférieure de la rétine. Au Congrès international ici à Rome (en 1894) j'ai déjà eu l'occasion de développer mes opinions hérétiques à ce sujet. Douze ans d'études anatomo-cliniques m'ont persuadé de la justesse de ma théorie. Il n'y a pas dans la littérature un seul fait qui soit en contradiction avec mon avis, et les preuves positives de l'exactitude de mon opinion se trouvent aussi dans les préparations que je suis prêt à soumettre ici aux experts du Congrès.

La destruction bilatérale du centre de la vision provoque une cécité corticale, mais n'efface pas les mémoires visuelles, et surtout pas la mémoire visuelle des mots. De même la destruction limitée au centre visuel des mots ne provoque aucune forme de cécité extérieure. La corticalité de la vision est donc localement séparée du centre supérieur

psychique de la mémoire des mots. Il y a donc relativement à la vision au moins deux centres corticaux; un inférieur qui reçoit immédiatement l'impression de la lumière et un supérieur, qui est la corticalité de la mémoire ou de la représentation.

La surface sensorielle reçoit l'impression sensorielle qui est envoyée et déposée en forme de mémoire et de représentation dans le centre supérieur psychique. La preuve de ce fait a été donnée par moi quant à la corticalité de la vision. Par une méthode, aussi originale que géniale, M. Flechsig est arrivé au même résultat quant à l'extension du centre optique et ses rapports à la corticalité psychique de la vision.

Or, est-ce que cette façon de voir peut aussi s'appliquer à la surface corticale de l'ouïe? La science a rencontré ici de plus grandes difficultés, savoir d'isoler le centre sensoriel primaire de l'ouïe du centre psychique, et la question ne peut pas se résoudre ici à l'aide des faits cliniques, car il n'en existe que très peu. Encore aujourd'hui on place le centre de l'ouïe et de la mémoire des mots à la partie postérieure de la première et de la seconde circonvolution temporale gauche, comme le prétendent M. Wernicke et M. Friedländer. M. Flechsig le premier a pris distance des autres savants dans sa méthode; il a volu démontrer que la surface auditive est minime et essentiellement limitée à la circonvolution transverse entre la circonvolution temporale et la partie postérieure de l'île de Reil. De même que le centre de la vision a une construction différente de la corticalité voisine, comme je l'ai prouvé, ainsi cette circonvolution diffère, selon M. Flechsig, de la corticalité voisine, et M. Ramòn y Cajal semble en avoir fait l'histologie en détail.

(Il n'est pas bien sure, que la description de M. Cajal s'applique à la circonvolution transverse ou, ce qui est plus probable, à la circonvolution première temporale. Remarque de M. Henschen, 1917.)

Ici, je vais communiquer à cette illustre assemblée un cas de surdité verbale pure, qui élucide ce sujet. Les cas

d'aphasie servent plus que d'autres à distinguer les surfaces sensorielles des centres psychiques.

Le cas est le suivant. Clara N., 54 ans, fut infectée de la syphilis par un enfant et fut en mai 1901 frappée d'un coup d'apoplexie, accompagnée de tiraillements et de faiblesse dans le côté droit; s'améliora. Le 1 Avril 1902 plusieurs nouvelles attaques, accompagnées d'une paralysie totale et permanente du côté gauche. Le 9 Avril elle n'avait pas encore d'aphasie, et l'ouïe était bonne des deux oreilles. Le 12 Avril nouvelle attaque, qui provoqua une surdité verbale complète. A partir de ce jour, son état restait constant, le côté droit n'était plus paralysé, mais le côté gauche l'était; pas d'hémianopsie.

Jusqu'à sa fin elle avait une bonne ouïe des deux oreilles: elle attrapait et comprenait le tictac d'une montre et le bruit d'un trousseau de clefs, elle pouvait entendre même un chuchotement et demanda alors: "Que dites vous, docteur?" Elle était lucide et s'intéressait à tout ce qui se passait dans la chambre, lisait son journal et racontait correctement les anecdotes lues. Quoique peu instruite, elle écrivait spontanément et copiait correctement et avec une certaine facilité. Elle parlait sensément et sans faire de fautes, mais ne comprenait pas un mot de ce qu'on lui disait.

Elle concevait les différents sons du diapason et semblait comprendre les mélodies et assurément le chant, mais ce n'était qu'exceptionellement qu'elle comprenait un mot isolé comme docteur; elle ne saisissait presque jamais une phrase. Elle écoutait peu les sons.

Elle vivait ainsi jusqu'à sa mort deux ans plus tard. Pendant deux ans elle fut observée journellement par moi et par d'autres docteurs.

L'autopsie montrait: *à l'hémisphère droit une destruction répandue* de l'île de Reil, de l'opercule frontal et *de la première et de la seconde et partiellement de la troisième circonvolution temporale.* La circonvolution transverse était ramollie; le ramollissement pénétra au fond, jusqu'à la capsule interne.

L'hémisphère gauche: la lésion est presque exclusivement corticale. La corticalité est *détruite* dans la moitié postérieure de la *première circonvolution* temporale et dans toute *la partie postérieure de la seconde circonvolution temporale* jusque dans le pli courbe. L'insula est bien conservée. La *circonvolution transverse* est restée intacte à l'exception d'une petite lésion superficielle à la limite de la première circonvolution temporale. Pas de lésion dans la moelle profonde.

De ce cas on peut tirer les *conclusions* suivantes:

1. La corticalité de la partie postérieure de T_1 et de T_2 ne transmet pas l'ouïe, car elle était bilatéralement détruite ou isolée, mais l'acuité auditive n'était pas altérée.

2. La circonvolution transverse du côté gauche, qui seule était conservée, aura transmis ici une ouïe aigue des deux oreilles, c. à. d. l'ouïe est bilatéralement innervée. La théorie Wernicke-Friedländer sur la localisation du centre auditif ne semble donc pas exacte.

3. La surdité verbale pure qui fut constatée complète, était causée par la destruction de la moitié postérieure de T_1 et de T_2 du côté gauche.

4. La surdité verbale pure n'est donc pas toujours causée par des lésions souscorticales, et la doctrine de Lichtheim n'est pas appuyée sur ce cas.

5. Le cas célèbre de M. Déjérine, où il y avait une atrophie des cellules des deux premières circonvolutions temporales, est en concordance avec mon cas.

6. Il est probable que la surface auditive est localement séparée de la surface perceptive des mots, à l'opposition de la théorie de Wernicke et d'autres, mais en accord de celle de M. Flechsig.

7. La destruction de la corticalité de la perception des mots ne provoque pas nécessairement un dérangement du langage interne.

8. La surdité verbale pure ne coincide avec la surdité corticale, comme le prétend M. Déjérine.

9. La théorie de M. Déjérine et d'autres sur l'aphasie

ne se laisse pas expliquer par ce cas, car ici il n'y avait jamais une lésion des autres formes du langage: la lecture, l'écriture et même la perception musicale étaient intactes.

10. La surdité verbale pure ne coincide pas avec la surdité labyrinthique, comme on l'a prétendu.

11. La surdité verbale pure et la surdité psychique ne se couvrent pas, car la femme en question entendait et comprenait d'autres sons comme ceux de la montre et des clefs etc.

12. La surdité verbale n'était pas nécessairement causée par une interruption dans le conduit médullaire, comme l'ont prétendu M. M. Lichtheim, Ziehl et d'autres.

13. L'innervation associative du langage et de l'écriture conservée, cette innervation doit provenir d'une autre corticalité psychique, qui paraît être d'une autre valeur, plus haute, que le centre auditif des mots. Il existe probablement un centre supérieur de la perception et de la mémoire du langage.

14. Le centre de la lecture était dans ce cas démontré indépendant du centre auditif des mots.

15. La doctrine actuelle de l'aphasie me semble donc renversée par ce cas et doit être révisée, et il faut un examen minutieux des lésions médullaires, c. à. d. des voies d'association — dans tous les cas d'aphasie.

Résumé.

De même qu'il existe pour la vision des centres séparés d'une valeur différente, il y en a aussi pour l'ouïe. Il y a une surface sensorielle pour la perception immédiate des sons, une autre pour la perception des mots, et celle-ci semble séparée de la corticalité des mémoires et de l'innervation des mots, c. à. d. il y a des centres psychiques d'une valeur différente.

Le cas démontré est donc d'une importance fondamentale au point de vue psychologique du cerveau.

A case of pure Word-deafness.

Clinical History.

Clara Nilsson, aged 54 years, maried, Stockholm 1902. Nr 141. Entered the Hospital $^4/_4$ 1902, died $^6/_6$ 1904.

Prehistory. No inheritance. Patient acquired syphilis 1883, by nursing a child of another family and got different signs of syphilis, as ulceration at anus, vulva and on her chest and mamilla. 1885 pharyngeal symptoms. Was treated 1884 and later antiluetically in 3—4 months.

Present disease. In May 1901, suddenly twitchings appeared in the right hand and foot and in the eyelids on the right side. The mouth was thrown obliquely. The twitchings ceased, but pat. had the sensation of paralysis in her right hand and foot. The superior right eyelid remained lower than on the left side, and pat. talked unclearly, and it was difficult to understand her. The food (and soup) ran out from the mouth. Consciousness was not lost, but her memory was from this time always weakened.

The latest period of her disease. On the 31st of March, when she was occupied cutting wood and keeping a match in her hand, the match dropped from her hand. She found in the darkness her left hand, which was quite cold, but did'nt recognize the hand as her own and began to bend the articulations. Soon afterwards the whole body began to tremble, and she fell to the left on her left paralysed hand, without losing consciousness. She stood up with some difficulty, went to her chamber and told her daughter what had arrived, and could then perform her duties.

On the 3rd of April the twitchings began again from time to time in her left arm and her left leg, and she went to bed feeling sick. She never lost her consciousness. There remained a certain weakness in the left leg and the twitchings in left arm and leg came and went. She remarked no anæstesia nor sensation of cool, since the 31st of March. There was no motility in the left arm, the left knee or foot. Entered the hospital $^4/_4$ 1902.

Status præsens $^9/_4$ 1902. Pat. is robust and fat. Sleeping and appetite are good. A great radiant scar on the 2nd right rib, a consequence of the open ulceration of 1896—1901. A little scar on the left fibula,

Pat. is in bed, has a little fever. No paræsthesia or hallucinations, headache or giddiness.

Psyche is clear, her comprehension is good, she is interested in the surrounding people and events, Her memory is weak, she is low-spirited and irritable. *No aphasia.*

Cranial nerves. I. Sense of smell good.

II. Sense of sight, on the right eye 0.3, on the left eye 0.2. Sense of colour good. The camp of sight smaller than normal, the left one a little smaller than the right. No hemianopsia.

III, IV, VI. No Strabism or ptosis. The left pupil larger than the right.

V. Sensibility on both sides normal. Sense of taste good.

VII. A ligt paresis of the left facialis.

VIII. Hearing good on both sides.

IX, X, XI, XII are normal.

Motility of the left shoulder a little limited, in the left arm, knee and foot completely disappeared, on the right side good.

Sensibility on both sides good, normal.

Reflexes on the left side stronger than on the right.

Heart. Sounds rather normal. Arteries: some rigidity.

Urine. No sugar, a little albumen.

Developement of the disease.

$^{12}/_{4}$. At 6 hours p. m. pat. suddenly became agitated and confused, talked unclearly and didn't lie quiet in the bed. Language sometimes clear, sometimes incomprehensible, could hardly speak a word. She appeared irritated, and supposed she would go mad. She was *word-deaf,* conscious.

$^{13}/_{4}$ Irritable and confused.

Word-comprehension. She did not understand what was said to her, even by regarding the mouth of the speaker. Her hearing was not lost. She did not answer questions.

Hearing. She hears a "Hallo", but does not attend much to the tic tac of a watch.

Language is sometimes very fluent; sometimes difficulty in pronouncing some words or sentences. She appears to have forgotten the name of her husband: "What is the name of my husband?" she askes.

Amnestic Aphasia. She recognizes the objects, but cannot name them.

Comprehension of objects is very good; when they give her a whatch, she regards it, gives it back saying she has no use of it, tells the names of her friends.

Reading: She appears not to understand reading, turns the newspaper upside down, does not understand its contents. She does not follow written orders.

Writing. She cannot be impelled to write, and refuses to take the pen.

Motility and sensibility are not changed (s. status.) No albumen.

$^{15}/_{4}$. She asked for drinkwater: "some cold in the mouth. I do not remember anything, I am not clear."

$^{16}/_{4}$. Pat. has read a newspaper and has partially understood what she has read. Pointed at a place in the journal, and said: "What a knave, how can he misbehave so?"

$^{17}/_{4}$. Pat. has read aloud to-day what was written on a paper, and has understood what she read.

$^{18}/_{4}$. Could tell the time by the watch.

$^{4}/_{5}$. Pat. is more animated and interested in the surroundings than formerly. She comprehends more of what she reads and what people say to her: at times she understands what they say to her, at times she does not comprehend anything. Generally she does not take care of the conversation, but only speaks for herself. She is chatty and speaks about the events in the hospital. She comprehends better what she is reading and comprehends clearly written orders. She often reads the newspapers and likes to have a chat about the contents of the journals.

$^{16}/_{5}$—$^{1}/_{6}$. Motility in the left side better.

$^{14}/_{6}$. Pat. still comprehends printed and written matter better.

$^{25}/_{7}$. Progressive amelioration, writes her name rather correctly. She understands what you write, if the sentences are short, by thinking for herself; she appears to be deaf when you addresse her.

She speaks very much, sometimes the words are failing, sometimes she does not remember the names of her friends, she is paraphasic, by repeating words; she remembers immediately the right word.

She is able to read, and tells correctly what she has read. Reads also written words and comprehends them.

She writes spontaneously or from dictation, although paragraphic.

$^{19}/_{12}$. *Clinical demonstration.* Examination.

Question: How old are you? Answer: Convulsions.

Question: What is your age? Answer: 55 years.

Question: Where have you got your nose? Answer: Don't understand.

Question: What is the name of the Doctor? Answer: On the right side.

Question: Have you any children? Answer: 2 in life, 2 are dead.

She distinguishes clearly high and low tones.

1903. Februar and March. Pat. animated and very much interested in speaking and tells everything fluently and humorously. The memory is good as also the ability of observation. She is interested in everything that happens in the surroundings. She speaks about the beginning and development of her disease. Reads with interest the newspapers and makes humoral remarks. It is often difficult to interrupt her speech.

She speaks correctly, reads correctly and comprehends printed or written words. Her word-hearing is variable, but is generally

defective. Noises and sounds are easily comprehended, as well as tones of different height.

Her state as before.

1904. A thorough examination was made in April.

State. General state as in April 1902.

Examination on *Cranial nerves:* in general as in April 1902. Hearing good on both ears, but she can't understand words.

Body: the left side and limbs are paretic.

Psyche: Perception good, recognizes objects, can name everything correctly and has correct ideas. Memory little weakened. Pat. talks very much, but often paraphasic.

$^{12}/_{8}$. It seems sometimes as if pat. could comprehend what is said to her. Speech is tolerably correct.

$^{15}/_{11}$. Pat. is chatty, humorous and likes to tell stories. She comprehends written words and sentences very well. Memory good. She comprehends different sounds for ex.: of keys, whistles etc., recognizes different voices. Being occupied with her own thoughts, she does not give attention to voices or talks of any kind.

She is not completely *word-deaf* to a certain kind of words. Speaks without hindrance, but a little paraphasic. Names objects correctly.

No *alexia,* she reads printed and written matter correctly and loudly, and tells what she has read.

Agraphia. She writes by order her name and her thoughts. Small errors, neglected letters, or syllables were not attented to. Some words were not written.

Calculation. She can perform simple counting by heart or by writing.

Amusie. Pat. could formerly sing, now no more; she hears the songs. but does not catch the words. Whether she is able to distinguish different melodies from one another, can not be decided.

Otological examination (Doktor Stangenberg). Showed normal state, but it was not possible to decide if she could distinguish between different tones, but she made a distinction between tones from different tuning-forks.

$^{15}/_{12}$ The tic-tac of a watch was heard on the right ear at a distance of 20 cm., on the left ear at 30 cm. ***She hears on both ears all tones, from the highest to the lowest.***

She can repeat words quickly without any delay, and not only consonants but also vowels, without regarding the mouth of the speaker.

Pat. can distinguish tones of different musical instruments (as of tuning-forks, voices etc.) and also tones of different height; she can tell which tone is higher or lower, if the difference is greater than a fifth. When there are small differences, she often makes mistakes.

She remembers common melodies, and tells the names of the songs, and can also repeat them by singing, even if she makes small mistakes. She has *great difficulty in understanding what is said to her*, and it is necessary to repeat it many times in order to be understood.

Language. Pat. likes to speak, her speach is clear, and she finds the correct words. No paraphasia.

Reads her journal and understands what she reads. Her word-deafness is very much marked, very seldom she is able to comprehend any word.

Writing. She does not like to write, she copies correctly, and writes spontaneously common words.

1904. April. Her general state is worse. In May very bad. $^{6}/_{6}$. Dead.

2. Macro- and microscopical description of the brain.

Macroscopical post-mortem. Dura and Pia normal. Nearly symmetrical lesions are present in *both* hemispheres.

Left hemisphere. Fig. 1—9. The softening forms a contiguous field, beginning in the occipital lobe and distending forwards as a narrow field over the posterior portion of T^1, T^2, and the lower part of $T^{tr.}$ (gyrus transversus) and the boundaries of 0^2 0^3, T^3 and A. The following cortical surfaces are destroyed: a small surface between 0^2 and 0^3, about 2—3 cm. before the 0- pole, T^2 (posterior part) with its white substance. T^1, (posterior half, between 7.5—10.5 cm. before the 0- pole) and the ventral margin between 5—7.5 cm. Of this portion only the cortex on the inferior surface is destroyed. A communication of the cortex of the extension 10.5—6.5 cm. with more central part is not possible, but a communication of the more frontal T^1- cortex with frontal parts of the brain may remain (Details in the orig.) T^{tr} in its lateral part at the union with T^1 is destroyed to an extent- of 1 cm.^{2}, (both the cortex and the marrow). The fig. will show how deep the destruction is extended in the white substance.

Right hemisphere. Fig. 10—21. Resulting from a thrombosis of the art. media, the softening has destroyed a considerable part of the middle brain.

This destruction is extended over the posterior part of the white substance of T^3, the operculum of F^3, C^a (= F^a, ascending frontal convolution) C^p (= P^a, ascending parietal convolution), the whole Insula, the greatest part of T^1 and T^2, and extends on both sides of t^l (sulcus parallelus); T^{tr} is totally destroyed, T^2 only in its frontal parts and at the T-pole. The destruction extends into the white

substance of Insula, the lenticular nucleus, downwards into the marrow of T^3—T^4, and has reached frontal portions of the internal capsule. The optical system is conserved, as well as the Thalamus, and partially the Globus pallidus. Corresponding to the frontal destruction, this touches the wall of the frontal ventricle.

b. *Microscopical examination.* (s. Original). A. *Left hemisphere.* The changes of the white substance may be studied on the figures. The most important changes concern T', T'', and T^{tr} (Fig. 1—9).

T^1: the softening has destroyed on frontal sections only the dorsal cortex, in middle sections the total convolution, on more occipital sections only the ventral surface, and extends to the white substance of T^{tr}.

T^2 is softened on frontal sections only on its dorsal surface, on more occipital sections nearly totally the softening penetrates into the occipital marrow, where stripes of new bundles are degenerated as far as the venticular wall.

T^{tr} is totally destroyed only in its frontal portion: on more occipital sections only the marrow of the root of T^{tr} is softened, in variable extension as fig. 7—9 show.

Secondary degeneration is to be seen in deeper marrow, corresponding to the auditory bundle.

The occipital optical bundles are preserved.

B. *Right hemisphere.* Corresponding to the great lateral destruction, the white substance is destroyed as far as fig. 10—21 show.

F^3: the marrow is resorbed as far as to the ventricular wall.

C^a an C^p: the ventral surface is softened.

Insula is totally softened as deep as to the globus pallidus (Fig. 12).

T^1 and T^2 nearly quite softened (Fig. 12—18).

T^{tr} totally disappeared.

Putamen disappeared.

Some foci of softening in the dorsal-anterior part of the internal capsule.

Secondary degenerations in Türck's bundle in pes, and in the frontal pes-bundle. (details, s. orig.)

Optical way essentially unaffected.

Résumé of the clinical observations.

Pat. aged 54 years was insons infected by lues 1883, manifesting itself by large chronic ulcerations in the skin of the chest, and by symptoms from anus and vulva. In May 1901 the first symptoms of her disease appeared in the form of twitchings in

right hand and foot and the eyelids; she had also a sensation of paresis in these parts.

In March and April 1902 repeated attacks with paresis of the left side, without aphasia but consecutive hemiplegia sinistra.

In April 1902 an attack of word-deafness in transitory and incomplete form. From this time the word-deafness persisted with its consequences until her death.

Present state in April 1902. Patients *psychical state* was, as to the comprehension of the surrounding world, normal, she conceived everything quickly and in a normal manner, recognized all objects, was vivacious and interested, but she soon became tired. Her ideas are normal, no hallucinations, except at the attacks. Memory in general good. Irritable.

Sensibility on both sides normal, as to all qualities.

Smell, taste good. Power of *sight* diminished on the right eye to 0.2—0.3, on the left eye also to 0.2. The sight-fields a little narrowed. *Hearing* on both ears sharp. Dr. Stangenberg could not detect any changes or defects. She perceives all tones, without any partial loss of tones.

Comprehension of sounds in every respect good, *with exception of words.* She could distinguish the voice of a man from that of a woman, and recognized the voices of different persons, but could not comprehend the meaning or sentence of the words they said. In general she was unaffected by voices.

Music-comprehension. Pat. can distinguish different instruments and tones of different height when the interval is greater than a fifth, can even distinguish between tones with a difference of half a degree.

Singing. Pat. could formerly sing, and even now recognizes the melodies of common songs, and is able to sing, although not quite correctly. She comprehends if the song is false.

Conclusion: The power of hearing is good, the comprehension of sounds and tones and melodies is good.

Aphasia. After the first attack there was no aphasia, and the comprehension of speech and words was unaffected. After the second attack ($^{12}/_{4}$ 1902) a marked word-deafness was observed. This symptom was constant, remained for 2 years, until her death and was stationary, but *not absolute.*

Comprehension of letters was not always absolutely absent. Sometimes she was able to comprehend short syllables and words, but she was nearly completely word-deaf to common conversation or address, and she said: there is something wrong in my brain-office.

The repeating of single letters is sometimes possible, but only exceptionally of words and never of sentences. There was also

sometimes a mechanical echo-repeating, but no psychical comprehension of the words.

No paraphasia in general, only exceptionally.

Alexia was not to be observed with exception of the first time after the attack. Pat. reads correctly, loudly and quickly, printed and written matter and comprehends everything. She tells correctly the contents of a journal.

Agraphia. Spontaneously she could write her name, but not from dictation, and simple letters when regarding the mouth of the speaker. In copying she was successful.

Motoric aphasia. Immediately after the attack of $^{12}/_{4}$ 1902 her speech was unclear and confused, but already the following day fluent, although she had some difficulty in finding certain words and sentences. Shortly afterwards she was always able to find the correct words, and spoke clearly and intelligently, often humorously, and named the objects exactly.

Amnestic aphasia. The second day after the attack and some days later, she could not remember the name of her husband, she recognized her parents and their names.

Soon afterwards her speech became correct and good, and she could speak of everything she liked, up to her death.

Muscles of the eyes. The pupils narrow, the right narrower than the left. Reaction persisted. No strabismus or ptosis.

Facialis: a slight paresis on the left side.

Motility. Very much marked paresis on the left side, especially in the leg.

Reflexes to be found on both sides; vasomotoric troubles at the end of life.

Internal organs. Arteriosclerosis.

Heart 1904: Dilatation, Myocarditis, Stenosis ostii mitralis. Kidneys: induration, infarcts.

The clinical diagnosis of a myocarditis and arteriosclerosis on luetical basis was verified by the post-mortem. The mitral stenosis was latent.

III. Clinical Remarks. Word-deafness.

The present case is without any doubt a case of "*pure word-deafness*" or "*surdité verbale pure*". Those both terms seem not quite exact, the term "pure" being only negative. The term of Lichtheim "subcortical" is not to be recommended, the anatomical localisation not being always in the white matter as in the case of *Déjérine-Sérieux* with

exactly cortical changes, and in the case of Pick with cortico-medullar changes.

The name of Lichtheim is based on theoretical ground without sufficient supporting section.

The pure word-deafness as being based on clinical observations ought to have a clinical name and ought to bear the name of "perceptive word-deafness". The character of this form of word-deafness is that only the comprehension of words is wanting and of course also the power of repeating words or writing from dictation, but the pat. is able to speak, to read with psychical comprehension and to write spontaneously and to copy.

All these characters were to be found in my case, and the word-deafness of the pat. was nearly absolute, constant and stationary. No repetition of words was possible; no paraphasia. Pat. could read the journals and write tolerably. Of such cases only a few with a postmortem are known, and only few of these are typical. Such cases are the following:

1) case of *Déjérine-Sérieux* with a progressive deafness until complete deafness, but incomplete word-deafness and repetition.

2) cases of Pick, not typical (Liepmann).

3) case of Veraguth (unclear case, Liepmann);

4) case of van Gehuchten and Goris, case of abscess, without satisfactory anatomical examination, as operated.

The remaining case 5 of Liepmann (Gorstelle) seems to speak in favour of Lichtheim's theory, an hemmorrhage being met with in the white substance. My case is of a nature to elucidate this form of aphasia.

All forms of pure word-deafness were at first considered as cerebral cases, but since Freund had drawn the attention to the fact that a labyrinthical deafness simulates a real word-deafness, this form has played a certain role in the history of pure word-deafness. Monakow has arrived at the conviction: "dass es keinem Zweifel unterliegt, dass der reinen Worttaubheit stets ein ausgedehnter bis in den Markkörper dringender Herd oder Massenatrophie in der Reg. temp. (Heschls Windung, hinteres $^1/_3$ von T^1, nicht

selten doppelseitig) entspricht. Unaufgeklärt ist nur ob es nicht stets um eine mit einem peripheren Ohrenleiden (Labyrinth-Erkrankung oder einer primären Degeneration des N. acusticus, eigene Beobacht.) notwendig kombinierte Herdläsion oder örtliche Atrophie in der Regio temp. handelt."

In some cases (of Déjérine-Sérieux, and Freund, Liepmann's case Hendschel), a disease of the labyrinth on one side was to be found. Monakow makes the conclusion, that the perceptive word-deafness is essentially of cerebral and not of pure labyrinthical origin, but Freund considers Liepmann's case not as a cerebral case (against Liepmann). Liepmann also opposes against the meaning of Freund, and is convinced of the existence of cases of word-deafness of really cerebral origin (case Gorstelle). After Bezold's theory that partial tone-deafness (of the sexte h_1—g'') is characteristic of labyrinthic form, it will always be necessary to examine the cases in that respect, before an exacte diagnosis can be made. From this point of view also the present case is worthy of attention. By repeated observations I could state that no partial tone-deafness was present, as Bezold supposes, but notwithstanding a word-deafness was observed. There is consequently no cause to assume a labyrinthical change as a cause of the present word-deafness.

Here are also the necessary guarantees, that the case of Clara Nilsson is a *case of pure word-deafness.*

My case is in opposition to the meaning of Monakow, that this form of word-deafness reposes on a labyrinthical base, and it shows that *cerebral forms of pure word-deafness do exist.*

As to the *anatomical changes,* it is to be observed that in this case the power of hearing sharply was unaffected on both ears (in opposition to the preceeding cases). The lesions were bilateral, and the case shows that in spite of a bilateral destruction of T^1, the hearing can exist without diminution of its sharpness, and that the destruction of one

temporal lobe does not diminish the power of hearing on the opposite ear, as I have already shown many years ago in cases published in my Pathologie des Gehirns.

Larionow and Edinger have also proved this.

The theory of Lichtheim does not find any support in my case, but the conception of the anatomy of the pure word-deafness will be cleared up and extended by my case. Liepmann found a medullary (subcortical) lesion and assumed his own case as supporting Lichtheim's theory, but the present case shows that *in such cases also the cortex can be softened.* The supposed subcortical nature of the pure word-deafness is not exact or exhausting the anatomy of this form of word-deafness.

As to the narrower localisation of the subcortical word-deafness, Monakow supposes as basis "einen ausgedehnten bis tief in das Mark dringenden Herd oder Massenatrophie in der Reg. temporalis (Heschl'sche Windung, hinteres Drittel von T^1, nicht selten doppelseitig). This assumption is not verified in my case, the convolution of Heschl being destroyed only in its more frontal portion, the posterior part being conserved, but a bilateral destruction of T^1 and T^2 and of the right T^{tr} being found. I conclude that the posterior portion of T^{tr} does suffice to a good hearing. *This case is of course fundamental as to the localisation of pure word-deafness.* The destruction of T^{tr} is of course not the condition necessary for the appearence of the pure word-deafness, as certain authors presumed. I conclude that *T^{tr} (Q) is the auditory centre.* Consequently I do accept the meaning of Monakow and Quensel.

But Quensel says: "Die Querwindung bildet eine Grenz- und Übergangsstation. Alle durch einen tiefer peripher gelegenen Herd bedingte Worttaubheit könnte man als rein perceptive bezeichnen, diese ist stets absolut, sie ist rein und — stabil. Umgekehrt ist jede durch einen zentraleren Herd hervorgegangene Worttaubheit nothwendig partiell, kompliziert und meist auch rückbildungsfähig. Rein associative form."

I do not accept this conclusion. T^{tr} being the real primary

hearing centre, every destruction of T^{tr} or of the more peripheral hearing conduction ought to bring on a cortical deafness or a conduction-deafness. (Leitungstaubheit). Every destruction, if bilateral, or a destruction of the corpus callosum, resp. the right hearing conduction and also of the connection with the right hearing centre, resp. this centre itself, is characterized through the acustic irritation not being forwarded to the wordhearing centre in the left T^1. Such changes being present, the pure word-deafness ought to be the necessary consequence. But there is in those cases no real aphasia, but only a labyrinthical, conductional or cortical deafness present, simulating a perceptive word-deafness. As a consequence of the destruction of the hearing-cortex or of the conduction, the power of repeating words or writing from dictation is impossible, but the person is able to speak, read and write as a deaf man, still he is not aphatic. The cortex too, or the communication between T^1 and T^{tr} being destroyed, the present form of word-deafness in my case will show all those characters of a pure word-deafness. *Consequently T^1 mediates the passage of the sounds of words to the association centre, where the comprehension of the words takes place.*

By *clinical observation the existence of two forms of word-deafness, the word-sound-deafness and the word-comprehension-deafness is proved.* Consequently there exist two centres: 1) of word-sound 2) of word-comprehension.

In consequence of this theory, we ought to acceept 3 forms of word-deafness.

1) A *pseudo-word-deafness* essentially only a form of deafness with 3 subforms:

a) of labyrinthical, b) of conductional, c) of cerebral (cortical) origin.

To these classes belong many cases of pseudo-word-deafness, as the subcortical word-deafness (Liepmann's case) or cortical deafness, by destruction of T^{tr} (some cases of Quensel) or labyrinthical deafness (Freund).

2) *Perceptive word-deafness* is a consequence of the destruction of the centre for word-sounds in T^1 or of the

conduction between T^{tr} and T^1 or of the conduction between T^1 and the centre for comprehension of words. This form is characterized by a loss of the comprehension of words, of the power of repeating words or writing from dictation, but conservation of the spontaneous speech, writing and reading. To this form belongs my case.

3) Associative word-deafness with troubles of the internal word, also of the spontaneous speech as a consequence of the destruction of the centre of word-comprehension, which is probably situated in (T^2 and) T^3.

The confusion about this matter — the real nature of word-deafness — is very remarkable, and difficult to understand. This confusion is, after my opinion, a consequence of an erroneons localisation and limitation of the hearing centre in relation to the word-centre, the authors localizing those to the same surface in the temporal lobe. In this respect Wernicke is at present a leader, supposing that those both centres coincide and that the hearing conduction of the sexte $h_1 - g^2$ ends in the word-centre in T^1, where only this sexte would be perceived. Wernicke has *by this assumption destroyed the base of his own theory on word-deafness* and *his whole doctrine of aphasia*, and *disapproved* the *specificity of the word-deafness. Aphasia would of course be in concordance with this new theory of Wernicke no more a loss of one of the highest psychical faculties* and *functions*, viz. *to comprehend the speech, but only a form of partial deafness* to those tones, wich are necessary for understanding words — *of course a form of central deafness.* But, I think, it *will be necessary to distinguish between cortical deafness to sounds or tones and word-deafness.* The present case is of nature to clear up this matter, and it will be necessary to make a revision of the doctrine of aphasia in this respect.

Conclusions from the present case.

1) The cortex of the posterior parts of T^1 and T_2 does not belong to the hearing centre as being in both hemispheres destroyed, without any lowering of the acoustic power. (Reservation in respect to the part of T^1 in immediate vicinity to T^{tr}.)

2) T^{tr}, which was conserved on the left side in its posterior portion, may have received the acoustic irritation. This part only on the left side remaining, the ears (hearing) may be bilaterally innervated. The conservation of only the posterior part of T^{tr} on one side may suffice for a sharp hearing. The localisation of Wernicke-Friedländer of the hearing centre in T^1 is erroneous.

3) The "pure word-deafness" in this case was effected by the bilateral destruction of the posterior portion of T^1.

4) Lichtheim's theory as to the localisation of the subcortical word-deafness is not conclusive.

5) In the case of "pure word-deafness" cortical, corticomedullar, or only subcortical (medullar) changes are present. The name "subcortical deafness" is consequently not supportable.

6) The auditory centre is of course separated from the word-centre, in opposition to Wernicke's but in concordance with Flechsig's doctrine, an opinion supported by myself as early as 1887 at the Swedish medical Congress in Norrköping.

7) The destruction of the word-sound-centre in T^1 does not provoke any trouble of the "internal word" and the spontaneous speech.

8) The word-sound-centre and word-comprehension-centre are locally separated, and the destruction of these centres provoke different forms of aphasia.

9) The pure (perceptive) word-deafness does not coincide with the labyrinthic form and may exist without any lesion of the labyrinth (against Monakow and Freund).

10) The pure word-deafness does not coincide with the psychical deafness, as some authors think, the present pat. understanding the nature of every sound, except that of words.

11) The reading faculty is independent of word-deafness and has a special centre, separated from the word-hearing centre.

12) Also the writing is independent of wordhearing.

13) There exist in the temporal lobe at least 3 different centres above each other: the primary acoustic centre,

the word-sound-centre, the word-comprehension-centre. With those the music-clang-centre and music-comprehension-centre are coordinated, the localisation of which is not known. Also the limitation of the word-comprehension-centre is not yet sufficiently known. The word-clang-centre is localized to T^1.

14) The doctrine of word-deafness must be revised.

The most important conclusion is that there exist in the temporal lobe different psychic centres lying above each other, of different value and different localisation.

IV. Pathologico-anatomical remarks.

Observations in case Clara Nilsson.

1. A. *Left hemisphere.*

Secondary degenerations from the great softening in the temporal lobe.

1) In the occipital optical radiation atrophical bundles from the softened focus perforate the white substance as far as to the ventricular wall (Fig. 3—7).

2) 2 white stripes emanate from the degenerated focus below the hilus of T^{tr} and upwards into the white marrow (Fig. 7—9), one parallel to the insular surface (association fibres).

3) The other more ventral stripe takes its course inwards to the internal capsule, on the dorsal side of the external geniculate body and disappears here in the retrolenticular capsule (in the direction of Türck's bundle or the acoustic conduction) (s. original).

B. *Right hemisphere.* Primary focus (s. orig.).

Secondary degenerations:

1) The acoustic conduction must be destroyed or atrophical. The internal geniculate body is completely degenerated; the tissue is nearly destroyed, the nerves degenerated. A degeneration in the lemniscus is not to be seen, but in the pes and the brachium of colliculus posterior. The degeneration of the internal geniculate body seems to depend upon the destruction of the T^{tr} (s. orig.).

2) A second degeneration has a greater interest. First is to remark that in spite of the great destruction of the T-lobe, since 2 years, the lateral part of pes is very well colored. Consequently the lateral bundle exists, and it is possible to follow a degenerated field ventral from globus pallidus upwards and round the external geniculate body (Fig. 1—6 in Text) (Fig. 19) (s. orig.).

Conclusion: in the formation of Türck's bundle in pes. not only bundles from the temporal lobe contribute, but also bundles from other cortical surfaces.

2. Türck's *bundle.* Very different opinions exist about the origin of this bundle. The controverse concerns not so much the course of the bundle as rather its origin, its situation in pes, and the physiological function of the nerves of this name. And probably very different bundles bear this name.

The following table shows how different the oponions are about the origin of Türck's bundle.

A. *Only in the occipital lobe.*

Meynert, Strickers Handbuch, S. 722, 1871.
Huguénin, (Déjérine, Soc. de Biologie, 30. XII; 1893).
Charcot, Localisations, 1876 p. 223.
Ballet, Brissaud: Thèses de Paris (1880, 1881).
Flechsig, Neurol. Centralblatt. 1904, p. 1065.
Probst, Arch. f. Psychiatr. Vol. 35. p. 39 (in Cat).

B. *Only in the temporal lobe.*

Ferrier and Turner (in monkey).
Marie and Guillain, Semaine med. 1903, p. 229.
Déjérine, Anatomie II, p. 141—146; Soc. de Biologie $^{30}/_{12}$ 1893.
van Gehuchten, Anatomie II, p. 470.
Flechsig, Neurol. Centralblatt. 1908 p. 57. (Complete degeneration enters only by destruction of Flechsig's hearing sphere; Tü. has no connection with T^3).
Kam, cit. by Bechterew, p. 506.
Obersteiner, Anleit z. Stud. d. Centralnervensystems. 1901, p. 398.
Mingazzini, Anatomia clinica. 1908, p. 181—184. Tü. from T^1, T^2 (and T^3).
Probst, Kais. Akad. zu Wien 1906, März, p. 54, 55; Tü. from T^1.
Pusateri, Rivist di Fren. Tü. from T-pole.
Anton, Arch. f. Psych. Bd 32, p. 112.
Kattwinkel and Neumayer, Deutsche Zeitschr. f. Nervenheilk. Vol. 39. Neurol. Centralbl. 1911, p. 489. Tü. only from T^3 (T^1, T^2).
Brodmann, Physiol. d. Gehirns, 1914. p. 130.

Mills and Spiller, No degener. after destruction of T^1 & T^2 (cit. by Marie & Guillain).

C. *In the temporal and occipital lobes.*

Bechterew, Leitungsbahnen, p. 506; also Kreuser.
Gerwer, cit. by Bechterew.
Zacher, Arch. f. Psychiatr. Vol. 22, p. 670.
Edinger, Vorlesungen, 1911. S. 497. Principally from T^2, T^3 and the lateral O-lobe.
Hösel, Arch. f. Psychiatr. 1902.
Schütz, Monakow. Neurol. Cblt. 1902.
Monakow, Gehirn. Pathol. 1897, p. 39, 44. Fig. 26, 27: 1905, p. 52, 54, 82, 115, 117, 120, 410, 426. Fig. 84.

D. *Only from the parietal lobe.*

Winkler, cit. in Neurol. Cblt. Jelgersma, ibid.
Brero, cit. by Bechterew.

E. *From the parietal and temporal lobes.*

Sioli, Zeitschr. f. Psychiatr. 1889. Bd 45, p. 429.
Rossolimo and Frylinsk, Cit. by Bechterew.

F. *From the temporal and occipital and probably also (?) parietal lobes.*

Monakow, Gehirn Pathologie. 1905.
Henschen, (S. Orig.)

G. *From the great late-myelinised caudo-ventral cortical surface.*

C. Vogt, Études sur la myélinisation des hémisph. cérébraux, Paris 1900.
C. and O. Vogt insist (Bruxelles 1911) against Flechsig that a destruction of the anterior gyr. trasversus does not cause any degeneration of Tü.

In the literature there are a great many facts to be found about the course and origin of this bundle. I bring up for discussion here only a few of these facts, and especially those published by Déjérine and Marie (s. orig.); and I pick up from my earlier observations in "Pathologie des Gehirns" 6 observations (s. orig. p. 375—380) of interest for the solution of the problem of the origin of Türck's bundle.

Conclusions: as to the origin from the O-lobe.

1. The calcarine cortex has no relation to Türck's bundle.

2. The cortex of the lateral O-surface has intimate relations to the ventro-lateral portion of the Türck's pes-bundle.

3. The cortex of the O-T-convolution (gyrus fusiformis, g. occipito-temporalis) has probably some relation to the dorso-lateral portion of Türck's bundle. Those fibres are mixed up in Türck's pes-bundle with fibres derivating from T^2 and T^3 (no fibres from T^{tr} or T^1).

These conclusions from my own preparations are in good concordance with the observations of some other authors (s. orig. 364—375). I may consequently be allowed to assume that the fibres of Türck's bundle emanate from the cortical viz. psychical surfaces of the representations, and not from the primary sensory surfaces. The function of Türck's bundles is to mediate the influence of the sensory representations (of sight and hearing) on the cerebellum and also to govern the organ of equilibrium.

V. The clinical and anatomical limitation of the hearing centre.

It is commonly admitted that the precise situation and limitation of the acoustic centre in the brain is not yet exactly known. The oldest observation about the acoustic centre was made by Ambroise Paré 1538, when he observed a case of deafness after a trauma of the parietal bone (s. Jules Soury, Système nerveux central. Paris 1899. I, p. 360). The renowned anatomist Varoli assumed already the bilateral innervation of the ears.

1. *Experimental researches.*

First through the renowned experimental researches of Ferrier and his successors the situation of the hearing centre was precised. Through his extirpation of T^1, the hearing faculty was lost on the opposite ear. But this observation was contradicted by various authors. Ferrier

had limited this centre to T^1, but Luciani-Seppilli assumed that the centre was extended also to the parietal and frontal, and even to the hippocampal lobes.

Munk's opinion differed considerably from those of the above mentioned. Through extirpation of the posterior portion of T^1 and T^2 cortical deafness appeared, and through the extirpation of a certain surface in the second Sylvian fissure, psychical deafness — a term introduced by Munk, which afterwards played an important role in the physiology and pathology of the brain. Munk was also able to provoke a deafness for certain tones — tone-deafness — and Larionow could also in Bechterew's laboratorium verify Munk's conclusion as to the existence of partial tone-deafness, provoked by extirpation of certain portions of the temporal surface, but arrived at a contrary localisation of those surfaces for the different tones.

Ferrier's results were contradicted by Schäfer and Horsley. Monakow provoked, through extirpation of both temporal lobes, only a temporary deafness and assumed that the acoustic centre extended widely over the limits of the temporal lobes.

He supposed also "dass das Gebiet im Cortex, in welchem die Schallreize zu Gehörseindrücken verarbeitet und als solche erweckt werden, weit über die sogenannte Hörsphäre hinausgehe und dass es durch den operativen Eingriff nur im Sinne der Diaschisis geschädigt, d. h. temporär gehemmt werde" (Die Lokalisation im Grosshirn, 1914. s. 803) — But the term of diaschisis cannot clear up the contradictory experiments of the authors, and Monakow admits him-self, that "diese wiedersprechenden Angaben der Autoren sich zum Teil durch die Schwierigkeiten erklären, Hunde auf den Umfang der Gehörstörung exakt zu prüfen."

There is no doubt that also it is difficult through experiment to limit the action of the extirpation.

According to my opinion, we meet the same difficulties here as to the limitation of the exstirpation and the explication of the experiments on the sight centre. As to these

I uttered as early as 1892: "In der Tat ist während der letzten 20 Jahre so viel Scharfsinn, so viel Mühe, so viel Umsichtigkeit und Gewissenhaftigkeit auf diese Frage konzentriert worden, dass wir auf dem eingeschlagenen Wege kaum hoffen dürfen ans Ziel zu gelangen." (Pathol. d. Gehirns II, s. 262). The starting-point of the experiments is a wrong one. The further and more detailed history of the experiments on the hearing centre may be found in Bechterew's paper (Über die Gehörcentren der Hirnrinde, Arch. f. Anat. und Physiol. Physiol. Abth. Supplem. Band 1899) as well as in Brodmann's and Monakow's works 1914.

2. *Myelogenetic Methods.*

Flechsig was the first one who threw light upon the question of the situation and limitation of the acoustic centre, viz. through his fundamental myelogenetic researches.

In his first papers, he localised this centre to the "most posterior part of the temporal convolution", but already 1895, after new observations, he localised it essentially to the *Gyri transversi*, especially to the anterior of them.

This convolution is hidden in the depth of the Sylvian fissure 1908. Flechsig explained still more clearly his conclusions in this respect by uttering: "Die erste Schläfenwindung gehört, mit Ausnahme dieses kleines 1—2 cm grossen Stücks nicht zur Hörsphäre. Hierin weiche ich von allen neuen Forschern ganz erheblich ab. Weder bei Monakow noch bei Wernicke u. a. findet man einen Hinweis darauf, dass lediglich die temporale Querwindung die Hörsphäre darstellt — ausschliesslich die Myeologenese gibt einen Fingerzeig" (Neurol. Cbltt. 1908, s. 50 ff.).

Afterwards Monakow did verify this result, and other authors, as Quensel and Siemerling arrived at the same result.

Flechsig assumed further that the acoustic conduction was limited to the anterior Gyrus transversus. Against

this meaning opposed 1910 C. and O. Vogt (Nouvelle contribution à l'étude de la myéloarchitecture. 20:e Congrès des méd. alién. etc. de France, Bruxelles — Liège 1910) and admitted that the conduction from the internal geniculate body finishes not only in the (anterior) T^{tr}, that the myelogenesis entered sooner in certain parts of T^1 than in the Gyrus tranversus, and that the cortex of T^{tr} contained different myeloarchitectonic fields.

The method of degeneration verified and completed the results from the myelogenetic method. Forel had observed that the external geniculate body and colliculi anteriores are atrophied in Talpa europæ, but the internal genic. body and the posterior collculi ware very well developed in this animal, Monakow happened provoke a degeneration of the internal geniculate body by extirpation of the surface B of Munk, and from this fact infers that the temporal lobe is of a great importance to the hearing act.

3. *Clinical localisation of the acoustic centre in man.*

As regards human beings, very late hearing troubles were known as consequence of lesions of the brain. 1879 Nothnagel could not find any cases of real deafness as a consequence to lesions in the centrum ovale or in the cerebral surface, although already Ambroise Paré 1538 had observed deafness after trauma; the discovery of the hearing centre is ascribed to Wernicke, as having published a case of central deafness in consequence of bilateral growths in both temporal lobes, but this case is not of a nature to precise the localisation of the acoustic centre. Also other authors, as Schiess-Gemuseus and Hutin have described cases of central deafness through lesions in the temporal lobes. In 1890, Ferrier complains not to have found any decisive case as to the localisation of the hearing centre. 1891, Mills arrived at the following result. "Destruction of the auditory areas of the two upper temporal convolutions (T^1, T^2) of both hemispheres is necessary to compleat brain-deafness. The *retro-insular convolutions*

are anatomically and functionally closely related to subdivisions of T^1, the most posterior of these retroinsular convolutions with the superior $^2/_3$ of T^{1a}. Mills supposed to have found the precise localisation of the acoustic centre, but was not, as I think, authorized to draw such a conclusion from his case.

Bechterew localised the centre to T^1 and T^2 and to the posterior part of Insula. Déjérine says: "Il est aujourd' hui bien démontré que la partie postérieure du lobe temporal n'a rien a faire avec la perception des sons proprement dite".

Monakow accepted only 1914 the doctrine of Flechsig. Already 1887, I myself had at the congress in Norrköping (Sweden) uttered: "The rough sensation (Empfindung) is formed at one place of the temporal lobe, the posterior part of T^1 is educated to receive the clangs of the language. Here the representations about the psychical explanation of these clangs are formed, and here are the recollections deposed. Our representations are not localized to the same surfaces of the cortex as the immediate perception or sensation, (Empfindung).

At the Congress in Rome 1905, I localized, basing on my clinical case, the acoustic centre to the posterior portion of the gyrus temporalis transversus.

At the same time Campbell arrived at quite the same conclusion, basing it on his own fundamental microscopical researches, and later Brodmann has arrived at the same view, basing on his cytoarchitectonic researches.

1914 he expressed his opinion in the following manner: "Die klinischen Erfahrungen scheinen im allgemeinen die anatomischen und experimentellen zu bestätigen. Eine genauere Abgrenzung des — — eigentlichen Hörcentrums, ist jedoch bis heute noch nicht möglich. Es besteht aber die grösste Wahrscheinlichkeit, dass die clinische Hörsphäre etwa den mittleren und hinteren Teilen der T^1 entspricht, also annähernd der structurellen regio supratemporalis (Brodmann, Physiologie Fig. 78). Ob und inwie weit beim Menschen eine räumliche Trennung der eigentlichen Hör-

sphäre von der sensorichen Sprachzone durchzuführen ist, bleibt noch ganz unentschieden."

Anton. Pick and Strohmayer seem to accept this meaning. Very late Monakow joined Flechsig's view; Probst had already 1906 proved that the central acoustic path "durch den ventralen Abschnitt der inneren Kapsel zur temporalen Querwindung zieht, wo die Fasern endigen". "In der Rinde der T^1, T^2 und T^3 strahlt keine degenerierte Faser ein", and he arrived at the conclusion that "die eigentliche Hörrinde sich über die ganze Querwindung erstreckt."

As results of his clinical experience, Monakow expressed 1907 his conclusions in the following words that "die Hörsphäre des Menschen in die hintere Hälfte von T^1 (auch gyrus subangularis), in den G. supramarginalis, angularis, T^2, T^3 und die hintere Inselrinde zu verlegen ist." In later works by Monakow he takes different positions as to this matter. After an analysis of 16 cases of central deafness with postmortem he found that in all these T^{tr} and the posterior $^1/_3$ of T^1 were deeply changed or had disappeared; but later in his work he says: "sicher ist indessen, dass in sämtlichen Fällen die hintere Partie von T^1 und der Insel sowie der G. angularis, dann auch die unteren Temporalwindungen beiderseits zerstört waren". And then on the base of these facts he arrives at the conclusion: "In der Temporalrinde (beim Menschen Pars post. von T^1 und beide Heschl'schen Windungen) muss zweifellos die Haupteingangspforte für die Schallreize gesucht werden" — of course a much narrower limitation of the acoustic centre.

4. *Changes in the temporal lobes in deaf and dumb.*

Various authors have found in the secondary changes of the brain in deaf and dumb some facts of importance in respect to the localisation of the acoustic centre. But already the starting point of these observations seems to

be very uncertain. As I remarked already 1890, as to the secondary changes in blind people, not only the primary sight-centre in the brain will be degenerated or atrophied, but also all the cortical surface, being in some connection with the sight-centre, as well as the surroundings of this centre. So it was in a case of blindness of 50 years, published by me 1890. The atrophy extended to the entire occipital lobe, but was most developed in the calcarine fissure. (Pathologie d. Gehirns I. case 1.).

In the same manner it ought to be as to the cortical acoustic centre. Further it is proved that hearing residues may remain in deaf and dumb, and that no absolute atrophy enters in the acoustic conduction here, the acoustic nerves being often very little atrophied. Consequently it will be very difficult to determine through microscopical examination the situation and extension of the primary acoustic centre.

The present researches have also not arrived at a decisive result. Most authors had also the prejudice that the hearing centre was localized to T^1 and consequently neglected the examination of the surrounding parts, especially T^{tr}. Most examinations touch only T^1 and are only macroscopical.

Macroscopical examinations have been made by Fr. Schultze, Oppikofer and Politzer (7 cases), who found the temporal lobe normal; Donaldson found it small and the cortex thin, especially on the left side. Already Broadbent localized the atrophy in T^1, Mills in T^1 and T^2, Zancla found the T^{tr} small, but the architecture normal.

Only a few *microscopical* accounts are accessible. Drooglever, Foortuyn saw in T^{tr} no giant cells in many places, and in Brodmann's field 22 (T^1) a considerable diminution of the granular layer.

Strohmayer, who was quite convinced of the localisation of the acoustic centre in T^1 (perhaps also in T^2), found T^1 on both sides atrophied, also both insulæ, especially on the left side, atrophied, but T^2, T^3 and Uncus and G. Hippocampi normale, and in T^1 all layers, especially the

4:*th*, changed, where Strohmayer finds the end-station of acousticus. Strohmayer concludes also: The acoustic centre is in T^1; T^2 is associated with T^1. A narrower localisation is not allowed.

Brouwer found, trough an examination both T^{tr} considerably changed. The internal molecular layer was smaller and had lost many cells, the subgranular layer had completely disappeared, the polymorphe layer was not much changed. Brouwer says he could not explain the changes satisfactorily.

It is also clear that in deaf and dumb atrophy sometimes occurs in T^1, sometimes in T^{tr}, but it is not possible to see from the accounts of the anatomical examinations whether this atrophy is more exquisite in the one or the other part. All examinations agree in that respect that the most importent changes are present in the IV layer of T^1 and T^{tr}.

As to the sight centrum, the most exquisite changes also were to be found in the IV layer of the calcarine cortex, as I could prove by microscopical examination 1890—1892. Of course it is probable that this layer of the T^{tr} cortex receives the acoustic irritations, as the same layer of the calcarine cortex receives the optical impressions.

Campbell seems to be the only one who has made (1905) a rather satisfactory examination. He found in T^{tr} "exquisite changes clearly concentrated in the Gyri of Heschl, although distributed to a certain degree and extent over the psychical field of cortex and the posterior insula."

Campbell has also verified my observation of 1890 that by the loss of a peripheral sensory organ an atrophy appears not only in the corresponding brain-cortex, but also widely in the neigbourhood of its centre.

Conclusions. As a summary of all clinical observations on the situation, limitation and function of the hearing sphere, it may be said that there are *two different theories* in this respects. Anatomical and physiological facts

localize the hearing sphere in the temporal lobes. But as to the preciser localisation, two different views are to be found. Ferrier, the English experimentator, Déjérine, the French clinical scientist, Luciani and Seppilli, the renowned Italien physiologist, and Bechterew, the Russian, all these as well as the German physiologists Munk and Hitzig, Brodmann and Wernicke, locate the hearing centre in the posterior part of T^1 and T^2.

On the other side, Flechsig limits this centre to T^{tr} (and perhaps to a small spot of T^1 in the vicinity of T^{tr}). Quensel, Mills, Mott and Berger, agree in that respect. I myself have already 1887 postulated a special hearing centre and 1905 I located it in T^{tr}.

Monakow takes an intermediate place; formerly he accepted the first view, later he has accepted the view of Flechsig, but with a certain reservation.

VI. The functions of the temporal lobe and espec. of the Gyrus transversus.

If we accept the theory that the hearing centrum is limited to T^{tr}, the next question will be; what is the function of the other surface of the T-lobe in relation to that of T^{tr}?. In consideration of the contradictory experimental results, it is clear that this problem can be solved only with the help of clinico-anatomical facts in man.

First it will be suitable to analyse: How is a sensory (hearing) centre to be understood? I think it will be necessary to distinguish between the two words, hearing sphere and hearing centre, which are now used promiscue. I think sphere has a wider notion than centre.

By *hearing centre* I mean a cortex surface:

1. *Anatomically,* where the hearing conduction finishes in the temporal lobe;

2. *Physiologically* where the primary station for receiving the acoustic irritations is localized; the destruction of which causes a loss of the hearing power;

3. *Pathologico-anatomically,* where a degeneration occurs after the destruction of the hearing conduction; or the destruction of which provokes a descending degeneration of the hearing way;

4. *Whose* integrity is necessary for hearing. The clinical definition coincides with the physiological.

By *hearing sphere* I mean the wider territorium, which in some manner participates in the hearing act, consequently not only the hearing centre, but also where the sensory impressions are elaborated to higher psychical products, and where also probably the psychical acoustic recollections are deposed. The destruction of this wide field may probably not provoke any degeneration of the acoustic path, but a loss of higher psychical faculties or psychical symptoms.

Now the question arises: *is it possible to hear with an isolated hearing centre?* This question might seem superfluous, but the hearing centre being only a very limited surface, it is rather not correct to call it a psychical surface, where every form of psychical hearing elaborates and where our whole acoustic world and all acoustic recollections of every kind are deposed. The hearing act is a most complicated function, that necessary includes both a physiological-acoustic and a subjective act. However, the following questions must be answered: 1. What does take place in the T^{tr} after the arriving of the acoustic irritation by the acoustic path? 2. How are the sounds received and comprehended in T^{tr}? 3. The same question may be asked as to the Wernicke's centre and the rest of the greater hearing sphere.

First we must observe that every hearing ear is bilaterally innervated: the accoustic paths are also incompletely cruized. That is both anatomically and clinically proved, and the present case of Clara Nilsson is a verification of this fact.

As has already been announced, there are different theories about the seat of the hearing centre, one of them localising it essentially in T^{tr}, the other in T^1 and T^2.

To solve his problem *clinically*, it is necessary exclusively to examine cases with lesions limited to T^{tr}, but in consideration of the bilateral innervation, cases with unilateral lesions are not fit to solve the problem.

Only bilateral cases give a sure guarantee of a satisfactory resolution of this problem. Many authors, as William Boyd, supposed to have solved the question by using unilateral cases, but failed. The same faulty conclusion Niessl von Mayendorf made in the explanation of his case (Neurol. Cbltt, 1914. p., 308; s. orig.).

Only through bilateral lesions of the hearing centre, complete deafness may be provoked. But such cases are rare. It is a great pity that much of these rare and valuable cases are not sufficiently described, and in the whole literature there exists no systematical summary of those cases which are adapted to solve the above problem. This fact has caused me here to make a registration of them. (s. p. 398. in the orig.).

1. Cases with *remaining Gyri transversi* (in extract).

Case 1. Luciani-Seppilli. Great cortical softening of the cortex of both T-lobes. Pat. able to hear. Word-deafness.
„ 2. Fürstner. Aphasic. Bilateral cortical destruction.
„ 3. Kahler and Pick. Able to hear, considered deaf. Bilateral destruction of the temporal cortex.
„ 4. Pick. Woman, 27 years old. Reacts on sounds. Bilateral lesions of T-lobe.
„ 5. Heilbronner. Case II. Reacts only little on sounds. T^1 and T^2 on both sides destroyed.
„ 6. Freund. Is not deaf. Softening of both T-lobes.
„ 7. Anton (24 years old). Insula intact. Left T^{tr} intact. Both T-lobes destroyed.
„ 8. Déjérine-Sérieux. Progressive deafness. Bilateral cortical atrophy.
„ 9. Bischoff. I. Hearing intact. Bilateral atrophy of temporal lobes.
„ 10. Veraguth. Temporary word-deafness, hearing not lost. Light changes in T^1. (atrophy).
„ 11. Seppilli. Woman, 51 years; Hearing conserved, bilateral destruction of T^1, T^2; T^{tr} on right side seems conserved.

Case 12. Quensel-Blosen. Hearing conserved. Bilateral destruction of T-lobes (T^1). T^{tr} partially intact on both sides.
„ 13. Henschen. Above; hearing good. Bilateral destruction of T^1 (partially); the left T^{tr} partially conserved.
„ 14. Strohmayer. Hearing good: bilateral cortical destruction of temporal lobes.

2. *Analysis of the cases with remaining G. transversus.*

A thorough critical analysis of these 14 cases (details s. original, p. 404—409) will give the result that in all cases the *posterior part of T^1 was softened on both sides on a greater or smaller surface, or in the interior, generally symetrically, but the hearing power was conserved. The positive proof that the hearing centre is not in T^1 is consequently given,* especially as to the Wernicke's centre, which in all cases was softened, in spite of the hearing power being conserved.

However, a reservation is made as to the small surface of T^1 that lays in the immediate neigbourhood of T^{tr}, the description of which in the above cases is often neglected.

The conclusion that Wernicke's centre does not belong to the hearing centre is fully proved.

Another question is whether other parts of the T-lobe are able to receive acoustic irritations. In a great many of those cases, the T-pole was unaffected, as being nourished by other arterial branches than the posterior parts, as in the cases 12 and 13, but in the cases of Kahler-Pick, Luciani-Seppilli and Pick (case 27 years), the entire cortex on both sides seems to have been destroyed.

Of course neither the middle part of T^1, as Déjérine supposes, nor the T-pole, nor the posterior $^1/_3$ belong to the hearing centre.

T^2. As to T^2 we find that in 6 cases it was destroyed in the left lobe and in 11 cases in the right, in many cases on symetrical surfaces. Consequently T^2 does'nt belong to the acoustic centre.

T^3 is not so often affected as T^1 and T^2, but in 4 cases it also was bilaterally affected; of course it does not belong to the hearing centrum.

We arrive consequently viâ exclusionis at the probable conclusion that *the hearing is mediated through the* T^{tr}, as the anatomy seems to indicate. It must be regretted that the descriptions as to T^{tr} are not so precise or complete that it is possible in all cases to judge whether they are affected or intact. That derives from the circumstance that only little attention was attached to these convolutions, a consequence of the opinion that the hearing centre was localized in T^1.

T^{tr} was partially unaffected, *in my case,* n:o 13, on the left side, (but destroyed on the right side). Pat. had a good hearing. In this case (s. above) T^1 and T^2 were to a great extent destroyed on both sides. Also in case 12 of Quensel, T^{tr} was unaffected on both sides, the two T^1 and T^2 being on symetrical places bilaterally destroyed.

In the other cases the descriptions of T^{tr} are very defect, and only a supposition may be uttered about the condition of T^{tr}, but the essential part of T-lobe decidedly being destroyed, how will you explain the conserved hearing, if T^{tr} were not partially conserved?

In Pick's case n:o 7, probably the left T^{tr}, and in Seppilli's case n:o 11, probably the right T^{tr} was intact. All these facts speak in favour of the view that *T^{tr} receives the acoustic impressions, and that these of course are the hearing centres.*

3. *Cases with destroyed Gyri transversi.*

The next task will be to show *positively* that a destruction of T^{tr} is always connected with complete deafness. Fortunately there are cases described in which T^{tr} is totally destroyed, and with complete deafness. Besides these, T^{tr} are in some cases of deafness *probably* destroyed.

Case 1. Marchand. Hearing lost; bilateral destructions of T^1, T^2 on the left side "à sa partie postérieure", probably also T^{tr}; on the right side T^1, T^2 + la partie postérieure de la substance grise de l'insula".

Case 2. Shaw. Perfectly blind and deaf. Bilateral destructions of T^1.

„ 3. Heilbronner. No reaction by acoustic irritation. Extensive bilateral destructions.

„ 4. Wernicke-Friedländer. Deaf: bilateral gummata in the posterior parts of T-lobes.

„ 5. Anton. Did not react on sounds; T^1 + T^2 and their central connections bilaterally destroyed.

„ 6. Mills. Her deafness to sounds was complete. Bilateral destruction: of T^1 and T^2 on right side, also the retroinsular gyri, on left side the posterior atrophied.

„ 7. Hans Berger. Without reaction on sounds. Destruction of T^1 + T^2 + the convolutions of Heschl.

„ 8. Mott. Absolute deafness due to the destruction of the cortex on both sides, corresponding to the posterior $^1/_3$ of T^1, the transverse gyri of Heschl and the posterior part of T^2.

„ 9. Pick. (Woman 58 years). Complete deafness; both Insulæ destroyed and on left side T^1, P^2, A; on right side Insula + C^a + C^p.

On examing the above cases you will find that in cases 1—4 no detailed description of T^{tr} is given by the authors, but anyhow their destruction is probable. In cases 5—9 they are described as destroyed. In all cases the deafness was complete.

4. *Summary, hearing centre, word-deafness.*

Above it is proved that a destruction of the temporal gyri does not provoke deafness, if the T^{tr} are totally or partially intact; the cases show also that a complete deafness enters if also the T^{tr} are affected. But yet the experimentum crucis is wanting — the positive proof that an isolated destruction of the transvese gyri provokes an absolute deafness, it being possible that the T^{tr} only in connection with the temporal lobes provoke a complete deafness, and that the T^{tr} of course only by means of the T-lobe are able to mediate a normal hearing. Perhaps such cases may be found in the literature, but none is known to me. In consideration of the ramifying of the art. cerebri media, it is possible that only the artery that

nourishes the T^{tr} may be trombosed. Probably such cases are to be found among the aphatic ones. For this purpose it will be necessary to make the sections more detailed and precise than has been common hitherto.

VII. Gyri transversi (T^{tr}; Q).

These forming the hearing centres, the next question will be to ask wether both T^{tr} are necessary for a sharp hearing, or how much can be destroyed without loss of hearing. This question is really answered already, because in my case n:o 13 only the left T^{tr} was left, and besides only the posterior parts of it, viz. about $^2/_3$.

Many cases prove that one T-lobe can be missing without loss of the hearing. So in my case Malm, and in the cases of Pick (n:o 4 & 7) and of Seppilli (n:o 11), where only the T^{tr} on one side was intact.

Further it is indifferent if the remained T^{tr} belongs to the left or the right hemisphere. The surface of T^{tr} is not larger than about 2 cm^2, and one might think this small surface would be needed undiminished to receive all the millions of acoustic impressions that every day enter a human brain. But the above cases prove that this is not so. Not only my case will prove it, but also the case of Pick (n:o 4), where the right T^{tr} was destroyed and also the left seems to have been partially affected.

Quensel found in 2 cases a partial degeneration of the hearing conduction without a complete word-deafness existing. He says in reference to these cases: "Es genügt ein Bruchteil der zentralen Hörleitung, um die Perception der Wortlaute in ausreichender Weise aufrecht zu erhalten. Eine funktionelle Verschiedenheit Ihrer Elemente — — — lässt sich wohl kaum annehmen".

However, the theory of the specific conduction and localisation of the specific sensory impressions and of the fixed connection of the peripheral nerves with homologous cortical cells requires that such a fixed arrangement of the hearing fibres exist also in the acoustic conduction, as I have proved existing in the optical ways, a fact verified

by many authors. At present, every prove of such an arrangement is missing as to the hearing path. And we do not hope to find conclusive proofs in this respect, the conditions for such proofs in consideration of the bilateral innervation not being favorable. (s. orig. p. 424).

There is no hope that all these conditions will be fullfilled in a single case.

If the theory requires a systematic arrangement of the acoustic nerves in the acoustic path, a homologous arrangement must exist in the cortical surface of the hearing centre. However, partial destructions of this surface are not rare, and consequently we may expect once to find a case of partial tone-deafness with a corresponding lesion in the cortex of one gyrus transversus with total destruction of the other. Such cases might solve the problem of a projection of the organ of Corti on the cortical surface of the hearing centre.

The exact limitation of the hearing centre cannot be reached on only clinico-anatomical way. The necessary facts for that purpose are failing. Consequently it will be necessary to recur to anatomical facts and ask if the T^{tr} cortex has a specific architecture and if its limitation relatively to the neighbouring cortex is sharp enough to determine the extension of its surface.

a. *Anatomical remarks on Gyri transversi.*

Very late Heschl drew the attention to gyri transversi, already described by Barkow and Burdach, Flechsig being the first one who has determined their physiological function. The Heschl's convolutions are generally two, the anterior being the most constant.

As to their microscopical construction, Flechsig, Siemerling, Campbell, Ramòn y Cajal, O. Vogt, Brodmann and Rosenberg have made them an object of researches which coincide in the capital points, but differ in some other respects.

Macroscopically they are characterized by their covered situation, their form, smallness and constance. They differ

in form on both sides. The cortex in new-born children is characterized by its thickness, depending on the lower layers. Elliot Smith says: the two gyri of Heschl represent a sharply defined anatomical area of the cortex. occupied by two very dense bands. The area surrounding it on the surface of T^1 is composed of thicker cortex with less dense bands. (Journal of anat. and physiol. Vol. 41 p. 252).

Microscopically, cells. Flechsig did not find specific new cells, as Ramòn y Cajal, who admitted specific giant cells. However, Campbell utters: I must direct special attention to the condition of the external layer of *large pyramidal cells.* — which constitute the chief distinguishing feature of this area. (Campbells Fig., p. 154).

1893, by Hammarberg in my laboratory, and later, 1905, by Campbell (p. 155), the tickness was determined.

As to the *fibres*, Campbell says (p. 149) "The fibre-endowment here is unquestionable great; the distinctive features are the presence in the radiary form of numerous large fibres, the existence of a line of Kaës, and the general wealth of fibres in all layers."

These facts are also verified by myself, and are very conspicuous.

Myelinisation. According to Flechsig the anterior transverse gyrus receives myelinic fibres later than all sensory spheres. In a fœtus of 50 cm. length myelinic fibres may be seen coming from the interal capsule resp. the lenticular nucleus and entering the anterior T^{tr} in its interior $^2/_3$; only a few enter T^1. Consequently only that part is to be determined as a hearing centre; the hearing conduction is always limited to the anterior gyrus. The observation of Flechsig was verified by Cecile Vogt and extended to animals. "Nous sommes d'accord avec Flechsig que la partie du lobe temporal qui se myélinise le plus tôt est formé par les gyrus transverseaux et la partie postérieure de T^1. Celle-ci représente le centre de Wernicke." (Cfr. Étude de la myélinisation, Paris 1900. p. 64).

Brodmann assumes that the hearing cortex (Regio supratemporalis) is corresponding to the posterior part of $T^1 + T^{tr}$ and is more extended than Flechsig supposes. As to the structure, Brodmann utters: "Die Hörrinde (umfasst) etwa die hintere Hälfte der oberen Schläfewindung mit den angrenzenden, nach der Insel zugewandten Querwindungen Heschls und entspricht demnach im groben annähernd dem Bezirk, den man klinisch als Hörsphäre abzugrensen pflegt, nur dass unsere structurelle Region viel ausgedehnter ist als jener Bezirk. Die Zone stellt — ein funktionell einheitliches Gebiet" (S. 108).

These anatomical facts show evidently that the T^{tr} must be considered as a specific organ.

b. *Clinico-physiological remarks on T^{tr}.*

The clinical analysis has arrived at quite the same conclusion, although many of the clinical observations are not of a quite decisive character. The anatomical structure seems to give a good limitation of the hearing centre and limit it to the gyri transversi. What I asked (1887) on the Congress in Norrköping is of course evidently proved now, viz. that the hearing centre is separated from the word-centre. The function of the primary sensory surfaces in the brain has only been little discussed and has not been the object of special researches. As to the calcarine sight-centre, I already 1900 uttered the meaning that it receives the light-irritation and transmisses it immediately to an other surface. It mediates of course the transmission of the optical impressions. However, the question whether we see with the sight-cortex or hear with the hearing cortex requires an answer. In this respect it is interesting to analyse the above 14 cases. (Cfr. original). The cases 12 (Henschen) an 13 (Quensel-Blosen) prove that the sharp hearing is conserved when T^{tr} is intact. Of course this convolution mediates the hearing, even if T^1 and T^2 are destroyed. Nor is the subjective hearing

dependent on the conservation of the posterior part of T^1 and T^2, but ought to be received or mediated by other parts of the T-lobe, no extra-temporal convolution being able to mediate the hearing.

The cases 8 of Déjérine-Sérieux and 9 of Bischoff seem to be in favour of the conclusion that an extensive atrophy of the temporal lobes may be accompanied by a diminution of the intelligence. However, it is difficult to decide in what degree other changes of the brain may have contributed to this trouble.

Seppilli's and Pick's case 7 seem to show that T^2 and T^3 are enough for a subjective hearing, (T^{tr} being conserved) but that the softening of T^1 may have contributed to the diminished intelligence.

In case 1 the function of an isolated gyrus transversus may possibly have been observed, although no guarantee exists. Also in Pick's case 4 we may have a case of probably isolated T^{tr}. The pat. was not hearing in the common sense, but did react on acoustic irritations, and the case Kahler-Pick has an analogous character. Pat. did not react on exterior influences, but on acoustic irritaments.

From all these clinic-anatomical facts (s. orig.) may be concluded, as to the function of T^{tr}, that in case of integrity of T^{tr} only on one or on both sides, but destruction of the other T-convolutions, a person is not completely deaf, or insensible to sounds; he does react on sounds, turns his head, makes faces and nodds, but he has not the least comprehension of the contents, the importance or the nature of the acoustic irritation.

Such a man reminds us of the dog without a cerebrum of Goltz, who was irritated by sharp sounds, but did not understand the nature of the acoustic irritament.

Under these circumstances, is it allowed to speak of a subjective hearing? Now, that depends upon what one means by hearing. It ought to be the simplest form of hearing without understanding — a kind of reflex, a form of psychical deafness. It will of course be necessary to

make a distinction between hearing with comprehension and an acoustic reflex, without understanding.

Against this conclusion it may be objected, that the cases quoted are in consequence of the psychical state of the pat. not observed with necessary precision or exactness and of course not demonstrative. I cannot deny that such an argument is justified. The above conclusions are, indeed, hypothetical and not decisive.

With such a conception of the function of T^{tr}, the question arises if it is exact from a linguistic point of view to name T^{tr} a hearing centre that not hears, but only mediates the hearing; the hearing taking place in another part of the temporal lobe or in the whole brain. It seems more adequate to name the gyrus transverse "a hearing central", in analogy with telephone central or telegraph central or an "acoustic receiving station", an acoustic door, where the sounds enter the brain. The function of this surface may be to transform the sounds in such a manner, that they may be adapted to be transmissed to stations of higher psychical value, where they may be deposed as acoustic representations and recollections.

Certainly the sounds are already in the peripheral organ and intermediary stations (gangl. genic. int.) transformed into adapted forms of energy, before entering the cortex.

c. *Earlier conceptions on the function of gyri transversi.*

1. *Monakow's theory.*

The conception above proposed on the function of T^{tr} does not differ considerably from the new doctrine of Monakow of 1914, but was originally performed by myself, before knowing the opinion of this author. Monakow's conception has provoked a certain astonishment, as not being in concordance with his own earlier, since years defended doctrine about the psychical function of the brain, and involving quite a new opinion of this author on this matter. However, this theory is only a consequence of my views on the organisation of the primary sensory

surfaces of the brain, which I have defended since more than 20 years in respect of the sight centre. This doctrine of mine has been attacked by Monakow since years (1890) in his many great valuable books about the Pathology of the Brain, and in many pamphlets written by him and his pupils.

I do not here enter into a detailed discussion about Monakow's two different positions at different times; I only remark that this prominent author even in his latest work "Localisation im Grosshirn" has a very unclear position as to the limitation, organisation and function of sensory surfaces or centres, viz. especially the optical and acoustic centres. As to the criticism of these different standpoints I refer to my original paper (p. 434—436). Here I will only remember that one of them coincides with my always pronounced doctrine on the function of the visual centre, the other with a quite opposed view, that Monakow earlier defended against myself, up to 1914. According to one of these theories there do not exist very much circumscribed "insular" sensory centres, according to the other, defended by myself, the primary sensory centres are very much circumscribed, specifically organized small surfaces, where the peripheral sensory surface — retina — has a fixed projection, and where the resp. visual or acoustic impression enters, but only transitorily, the irritation being immediately removed to a higher psychical surface.

Consequently the primary sensory centre — viz. the calcarine visual surface is only a reception station that mediates the transmission of the visual or acoustic irritations and probably transforms or adepts them to a higher psychical function.

This later view Monakow seems to have accepted *now* on the hearing centre, at the same time combating the analogous principle as to the visual centre. However, Monakows position in respect of the hearing centre is a double one and very unclear, sometimes accepting and sometimes rejecting the doctrine of sharply limited sensory

centres and a projection on them (Cfr. orig.). Probably Monakow has made his conclusions on the base of his experiments on animals and further applicated the results as conclusive even on man.

2. *Wernicke's centre.*

It will be necessary in the first place to get a common conception about the functions of the temporal lobe. Since Wernicke's discovery of the localisation of the sensory aphasia, the neurologists were so occupied with this matter that no time remained to explore the other functions of the T-lobe. As has been already remarked, Wernicke localized his word-centre in the posterior portion of T^1 and T^2, and at the same place also the hearing centre. This view dominates even now many authors as Anton, Pick. Heilbronner and Strohmayer.

However, Wernicke of late years has essentially modified his earlier theory, in concordance with Bezold's doctrine that "der grösste Teil der Acusticusfaserung für das Verständniss der Sprachlaute ohne Belang ist" and "dass von der ganzen Tonscala nichts weiter als die kleine Strecke b_1—g_2 inklusive also eine grosse Sexte, übrig bleibt, deren Perzeption sich als unumgängliche Nothwendigkeit für das Verständniss der Sprache erweist". — "Das menschliche Hörvermögen umfasst aber eine Tonstrecke von über acht Oktaven. Wenn davon nur höchstens zwei erforderlich sind, um das Sprachverständniss zu ermöglichen, — — so gelangen wir zu der Vorstellung, dass nur etwa der vierte bis fünfte Teil der Projektionsfaserung des Acusticus in dem sensorischen Sprachzentrum sein centrales Ende zu finden braucht. — — Dieses erstreckt sich — nur über das hintere $^1/_3$ oder die hintere $^1/_2$ der T^1 und den angrenzenden Bezirk der T^2. Dabei ist an der Tatsache, dass auch der übrige Schläfenlappen zum grossen Teil als Endstätte des Nervus acusticus dient, aus anatomischen und physiologischen Gründen ein Zweifel nicht gestattet. Ich sehe mich also auf Grund dieser neuen Ermittelungen zu der Annahme gedrängt, dass das sensorische Sprach-

zentrum mit der Endstätte derjenigen Projectionsfaserung zusammenfällt, welche die Tonhöhe von $b_1 - g_2$ enhält. Daraus ergibt sich, dass der linke Schläfenlappen im Falle subkortikaler sensorischer Aphasie auch wirklich immer taub für die genannten Tonhöhen sein müsste." — —

Of course Wernicke considers — and Bonvicini has accepted this view — his word-centre as the field of projection of the sixth $b_1 - g_2$, whose acoustic fibres finish in the posterior $^1/_3$ of $T^1 + T^2$. *Such a conception of word-deafness includes quite a new view of the entire doctrine of aphasia.* Sensory aphasia is no more an inability to comprehend the psychical meaning of words and sentences, but only a central partial deafness of the tones $b_1 - g_2$, which have no cells more after the destruction of that part of $T^1 + T^2$. And consequently a high Soprano who speaks in a higher octavo will not be understood by those who surround her, but considered as aphatic or mad.

However, it is an anatomical mistake of Wernicke that the conduction of tones finishes in the first temporal gyrus. And that is the base and starting point of Wernicke's new theory. It will be clear from this account that an exact knowledge of the situation, limitation and organisation of the hearing centre has the greatest importance for the right conception of words and is a fundament of the anatomico-physiological brain psychology, and also the first condition for the solution of the problem of word-deafness, and in general of functions of the entire brain.

I think that the false view on the situation and function of the hearing centre has also misled the entire doctrine of aphasia. This is also the opinion of Flechsig. Strange enough, Wernicke and his school have mixed cortical deafness and cases of peripheral or cortical deafness (subcortical deafness), which are considered as cases of real word-deafness. As far as I am able to conclude, Wernicke *has overthrown his own entire doctrine of word-deafness by accepting his new view about the nature of word-deafness.* It will of course be necessary to except from the real word-deafness all those cases in which the word-

deafness depends on a peripheral subcortical or cortical deafness.

An analysis of my case n:o 13 will clear up this matter. Here the right temporal lobe was nearlly totally destroyed, Wernicke's centre on the left side was softened and of course only a portion of the left T^{tr} intact. Pat. was able to hear quite keenly and had no partial tone deafness, but was entirely word-deaf. Pat. was also able to perceive every form of sounds and music, but not of words. It is clear that the destroyed W. (= Wernicke's centre) was not able to mediate these perceptions.

Through the researches of my collegue Edgren, it is proved that musical perception does not coincide with word perception. *Amusie* is also observed without word-deafness and vice versa. Of course there ought to be at least two different centres in the temporal lobe. Further it is proved that word-deafness is not always combined with a loss of word-representations or word-memory.

Trough the destruction of W. one looses the perception of words, through the destruction of M (= musical surface) one looses the perception of musical sounds. But hitherto cases of deafness to common sounds (Geräusche), combined with intact perception of words or music are, as far I know, not described. But the problem of a special receiving station for perception and "gnosis" of common sounds, separated from those of words and musical sounds, is of great interest. The child screams from his first day, and also perceives screaming earlier than other forms of sounds. Later on the perception of music and melodies enters; at last the perception of words enters by education. The same evolution seems to enter also phylogenetically. Lower animals comprehend common sounds, some, as birds and certain serpents, sometimes also musical sounds, only the highest and most intelligent animals, as educated dogs, are able to perceive some words, and there is no doubt that monkeys have a kind of language, although very little developed.

This analysis will make it very probable that *isolated*

centres of different forms of sounds may exist. In consideration of the immense mass of different sounds of quite different nature and sense, which are used for various purposes, it seems probable that the corresponding surface, where all those sounds are received, is a very extensive one. This brain-surface may receive all those sounds entering in T^{tr} and there elaborate the psychical perception of them. But our knowledge of this matter is very defective. A clinical case of Kehrer is of great interest in this respect, and may be quoted here.

Kehrers 68-jähr. Pat. zeigte sich orientirt und litt an Totalaphasie. Vestibularuntersuchung ergab negativen Befund. Akustische Leitungsstörung ausgeschlossen. Bei tadelloser Gnosie auf anderen Sinnesgebieten war eine völlige Aufhebung der Reaktionsfähigkeit auf nicht sprachliche akustische Reizo der verschiedensten Art auffällig, Die Wortstummheit war eine temporale, aber bildete sich zurück; dagegen *blieb die aussersprachliche Taubheit bestehend.* Pat. litt an *Seelentaubheit nur* für Geräusche, trotz dem die Funktionsprüfung wohl durchführbar war und er durch Sprachlaute erweckbar war.

This very interesting observation seems to prove that an *isolated psychical sound-deafness may exceptionally exist without any cortical deafness sensu strictiori and without any word-deafness.* Theoretically also a special cortical surface in the temporal lobe for the psychical perception of sounds may be postulated. But its localisation is hitherto a secret. Only hypothetically we can look for its place. There is no cause to look for it outside the temporal lobe. T^{tr} being the common hearing centre, W. the word-perception-centre, M (at the T-pole?), only (T^2) and T^3 and possibly OT remain as a possible seat of the psychical perception of sound. Sound-deafness being so seldom observed, it is probable that a bilateral destruction of the sound centre is necessary to provoke a psychical sound-deafness.

As Brodmann has shown, the cortex of T^2, T^3 and OT have specific structures and different cyto-architecture (Brodmann's chart). The field of T^3 as well laterally as ventrally takes up a considerable surface, and yet Brod-

mann's fields 36 and 37 are remaining, the function of which is not yet known.

A case of Stauffenberg speaks in favour ot these views (from F. Müller's clinic and Monakow's laboratory).

Frau B., 61 Jahre alt, litt an Seelenblindheit, aber die Sprache war nicht an sich gestört. Gehör gut. Pat. reagirte auf leisestes Geräusch, nur das *Erkennen durch das Gehör war anfangs etwas gestört.* Bekannte Melodien sang sie richtig mit. Später erkannte sie schon bald am Geräusch fliessendes Wasser. Diktatschreiben gelingt gut. Nachsprechen ungestört.

Here we have a case with permanent good hearing power and good perception of words and musical sounds. but from the beginning the conception of sounds failed. The lesion was bilateral; on right side the almost entire T-lobe with T^{tr} was destroyed, and also on left side, with exception of T^{1} and T^{tr}—a most demonstrative case. (Cfr. the orig., fig. 14, 24, 25). The poles were intact as well as the music-perception.

We now arrive at the question: What is the function of Wernicke's centrum? If the word recollections are not lost through the destruction of this centre, they cannot be conserved or deposed in this centre, and it being probable that word-clang-images and word-clang-memories are closely combined with each other, it may be concluded that the word-clang-images are not deposed in W., and that of course the *psychical perception of words has not its seat in W.* However, the integrity of W. is necessary to the subjective perception of words.

Of course it will be necessary to assume that *W. as well as T^{tr} is a transmission station for words,* where a certain transformation of the word-clangs takes place, for the purpose of making these word-clangs adapted to a higher psychical perception. Such a theory is not in concordance with the common view that the acoustic word-images are deposed in W. This new view being accepted, word-deafness ought to be characterized as a state of inability to transmiss the word-clang to the station of psy-

chical word-registering or word-memory, where also the psychical word-images are deposed.

The above argumentation seems consequently to lead to the conclusion *that several centres or stations for transformation and transmission of the acoustic energies or irritaments are to be found in the temporal lobe, one above the other.* The first is the gyrus transversus, the second W. for words, and further a higher psychical acoustic centre for deposition of word-images. From this centre the energy may emanate in some form of energy to be combined with other optical and tactile energies, to give rise to complicated forms of psychical unities of energies of the subjective form of representations of mixed optical-acoustic-tactile representations, which form, after new transformations, the higher conceptions or ideas. The musical acoustic energies seem to go to the T-pole, and common acoustic sounds (energies) may be received and deposed in the remaining portion of the temporal lobes.

How is it possible now that some sounds are in such a manner elected and directed to different parts of the temporal lobes? I think we have in the phenomena of the air-telegraph some analogous facts. The electrical energy, sent out from the telegraph-office, is spread in various directions, but received only at a station of similar vibration. In the same manner I imagine that only certain cell-groups are able to receive acoustic energies of a certain nature or length of their waves. That may depend upon inherited changes of certain groups, educated in the length of times for those purposes, in concordance with certain hitherto unknown laws of attraction or repulsion or laws of conduction.

The theory I present here is for the present only a ***working hypothesis*** to elucidate the clinico-anatomical facts and has need of new supporting facts.

3. Limitation of Wernicke's field. (W.)

The limitation of W can be determined in an anatomical or combined clinico-anatomical way.

Anatomical limitation. It is commonly accepted that word-deafness enters through destruction of the posterior part of T^1 and T^2. The anterior limit is determined by different authors in a different manner. Déjérine and Monakow localise W in the posterior $^1/_3$ of T^1 and T^2, and Dèjérine localizes the hearing centre in almost the same place. Monakow accepts a wider surface and extends the centre to the gyrus supramarginalis, but gives at the same time a very unclear description or definition of the aphasic localisation, in the following words.

"Eine gewisse Lokalisation der Sprache ist selbst in Kortex zunächst theoretisch zweifellos vorhanden; diese Lokalisation zeigt aber in der Wirklichkeit eine von Grund aus andere Gestalt als wie sie bisher von den Klinikern (psychologische Sprachzentren im Sinn von Wernicke und seinen Schülern) angenommen wurde. Sie ist keine inselförmige, sondern eine "anatomisch-tektonisch *unentwirrbare.*" Ihr Character ist, wie schon derjenige einer Lokalisation für die Fertigkeitsbewegungen und die Sinneseindrücke, ein polymorpher und lässt sich nur aus der Entwickelungsgeschichte einigermassen verstehen und ableiten. Innerhalb dieser chronogenen Lokalisation ragen aber bestimmte Punkte des Zentrennetzes empor, und diese sind keine anderen als die Fociaggregate und deren perifokale Zonen im Operaclum-gebiete: der eigentliche ***Mutterboden für die articulierte Sprache*** (v. Monakow, Lokalisation in Grosshirn (p. 872).

By these words, Monakow, the leader in the physiology of the brain, has involved himself in an impermeable cloud, quite as God in old times when he led the Israelites from Egypt. But his oracular words do not guide the investigators in their sphere, as little as his doctrine of *diachisis*, which can quite arbitrarily transform every fact into the reverse. Monakow also embraces two opposite views as to the localisation of the speach; one of these postulates an insular localisation, the other not. (s. original, p. 445.) Wernicke seems to accept Déjérine's limitation of W. However, Brodmann has shown that the two posterior

thirds of T^1 have the same architecture, and the T-pole another. It is of course probable that W., the field of word-deafness. is *extended over the middle and posterior* $^1/_3$ *of* T^1. At this conclusion also Starr and Naunyn arrived by *clinico-anatomical* analysis of cases of word-deafness, before Brodmann and Flechsig had published their results about the architectural limits of the field of T^1. We are of course very much inclined to determine the *posterior* $^2/_3$ of T^1 as the field of Wernicke's word-deafness in concordance with the rule that *every surface of a specific structure has a special function* (s. orig., p. 444—450), or, as Brodmann says, "jede Area von specifischem histologischem Bau hat eine specifische Dignität."

T^2, does it belong to the wordhearing centre? Starr thinks so, and with him most authors, but not Déjérine, Monakow and Naunyn. T^2 has a structure different from that of T^1. Only bilateral cases are of a nature to decide this question, — but upp till now only a few such cases are known. Pick has published such a case with bilateral destruction of the marrow of T^2. The man showed a form of agrammatismus, but no word-deafness (s. orig., p. 450).

The different structure speaks against the supposition that T^2 belongs to the word-hearing field.

Amusie. In his remarkable paper on amusie, Edgren arrives at the view that a special form of deafness is characterized by deafness to music, and he locates it on a surface, anterior to W. Through a critical review of the matter Probst could prove that "in allen jenen Fällen, wo das Musikverständniss verloren gegangen war, auch eine Läsion der vorderen Abschnitte der T-Windungen zu konstatieren war. Dem gegenüber stehen nun alle jene 22 Fälle, wo bei erhaltenem Musikverständniss auch die vorderen Abschnitte der Schlafenwindung erhalten waren. Bisweilen lokalisiert sich der Musiksinn in der rechten Hemisphäre, doch ist ein Vikarieren nicht nachweisbar".

This surface, the T-pole, has also a special structure.

Auerbach's opposed view is not decisive, and his assumption of the localisation of the musical faculty in the posterior parts of T^1 and T^2 do not base on decisive facts; his conclusions (s. orig.) may also be erroneous.

I tried above to prove that a bilateral destruction of the entire cortex of T-lobe is accompanied by deep psychical trouble. And indeed, the cases above throw light on this field. Anton too, in his interesting paper, has shown that such a pat. has no more any acoustic memory. Further he has lost every perception of his loss, and he has no want of any acoustic memory. The acoustic world is dead to him. If T^{tr} remains, pat. still reacts on sharp sounds, but probably only by reflex way, without any psychical perception. The destruction of the entire temporal cortex has of course a special character, and is attended with grave consequences. Pat. has lost a part of his intellect, the acoustic portion, that resides in the temporal lobe. We are generally inclined to consider the intellect as an indivisible unity. But the facts above do not seem to be in favour of such a view. These facts are also opposite to Monakow's theory: "Die Annahme, dass die höheren seelischen Funktionen vorwiegend in besonders abgetrennten Rindeninseln — sich abspielen, ist nicht haltbar, man muss sich vielmehr die der geistigen Arbeit dienenden Elemente über die ganze Rinde ausgebreitet denken." However, Monakow is not very consequent, as he says: „Es giebt aber sicher für die geistige Arbeit nothwendige anatomische und architektonische Vorbedingungen, deren Strukturen bald in diesen bald in jenen Windungen dominieren". (s. Orig., p 453).

VIII. Common psycho-physiological remarks.

Amongst the innumerous brain problems, the question of the mechanism of thinking is certainly the highest, most complicated and most interesting. Our thinking emanates from the experience, collected by our senses and

elaborated by the brain. "Nil est in intellectu quod non antea fuerit in sensu" — this old sentence is even now-a-days recognized as correct and well grounded. Our present knowledge of the brain allows us to require an idea about the starting-point of the thinking and the development and formation of our sensory representations, and makes it also possible to localize certain of the psychical processes necessary for thinking. The thinking is a very complicated internal process that consists of a series of more elementar psychical moments, inseparable as to time, but yet consecutive. Consequently, on analysing the thinking, it will be necessary to dissolve it in more simple unities or elementar processes to clear up the function of our psyche. The most elementar psychical process is the primary perception (Empfindung) in the sensory cortical surfaces, where the impressions from the peripheral senses are received (Wundt).

Consequently it will be of great importance to get an exact knowledge of the localisation, limitation, organisation and physiological function of these sensory surfaces — and this knowledge will be the fundamental and first condition of a scientific psychology. The progress of our ideas about thinking will be dependent of our knowledge of the cortical sensory cortex, where the peripheral irritations enter the brain. Every thought requires a collaboration and association of the sensory perceptions, which are transformed, further connected and elaborated into thoughts.

But the peripheral and primary sensory perceptions of different senses are not able to collaborate, being different forms of energy, optical, acoustic, tactile etc.; of course, it will be necessary that all these different perceptions are transformed into similar forms of energy. I think that is the purpose and the necessary function of the primary cortical stations or centres. The optical centre receives optical energies, and transforms them into a new form of energy, in the same manner that the hearing centre receives and transforms the acoustic energies into similar form. Only after this transformation, these new forms of energy

are able to collaborate with one another, or unite them and form a higher psychical form of energy. These transformations will only be possible through the rather similar forms of the cortical cells, although these in the different sensory centres, are only to a certain degree of the same nature and not absolutely of a uniform construction. Through this collaboration of the cells of the sensory centres complicated representations, containing elements from different senses, will be formed, and on this basis the more complicated thoughts arrise. These contain, of course, a complicated mass of more elementar moments, arranged in different thoughts in a very different manner, and consequently forming different combinations in different thoughts.

To an adequate reception of the peripheral irritations in the brain cortex, organs of a physiological construction, analogous to the peripheral senses, ought to exist, i. e. a retina corticalis, a cochlea (corti) corticalis etc. ought to receive impressions, adequate to the peripheral as to intensity, form, colour and localisation of acoustic vibrations in the external world. The central retina, the cells of the hearing centre etc. ought to be connected with the peripheral sensory surface through a *fixed* arrangement in such a manner that every peripheral nervous cell corresponds to a central nervous element (brain cell). By this arrangement, the sensory peripheral irritaments conserve their local signs (Lokalzeichen) even after their transmission into the brain cells. This theory requires with necessity a projection of the peripheral sensory surfaces on the cortical surfaces in the brain, as well as a fixed connection between the peripheral and central organs.

The daily experience teaches us that we are able to localize every sight perception in the sight-field. Already from this experience it is allowed to conclude that a fixed connection exists betwen the retina and the corresponding point in the sight centre If that was not the case, an irritaton of the inferior part of the retina could be perceived in the brain as coming from a more dorsal

point of the retina, and every localisation of the sight impression would be impossible. This arrangement is absolutely necessary for a normal orientation in the sight-field.

A fixed and homologous arrangement of the peripheral and central sensory cells is denied by Monakow. His hypothesis of a change (Umschaltung) or intermixing of the optical irritations in corp. geniculatum externum abolishes every possibility of receiving adequate irritations as to the intensity, quality and localisation of the peripheral sensations. His hypothesis is of course absolutely contrary to anatomico-clinical facts, already proved, and to the daily experience. For example, an irritation of a finger will always be perceived in the brain as coming from the finger, not from the foot etc. It is here a question of a fundamental arrangement, and not only an anatomical, but also and especially a physiological organisation of the greatest importance for the brain functions, the nourrishment and the power of defense of animals. And without such an organisation every psychical perception of the outer world would be impossible.

The doctrine postulated by Wilbrand and myself, which has afterwards been proved through clinico-anatomical facts — the theory of the projection of the ocular retina on the *calcarine cortex, is of course a basis and fundament of the doctrine about thinking, and also the starting point of consecutive psychological researches.*

As to hearing, it is probable that the cells of Cortis organ correspond to tones of different height (vibrations), and that also every peripheral cell corresponds to a certain cell in the hearing centre. Without this fixed arrangement, it would be possible that a high tone, received by the ear, produced another tone in the hearing centre. Such an arrangement would make every musical perception impossible, and in the animal world make it impossible to the beasts to find their food or catch game.

But hitherto no decisive proof exists in respect of such an arrangement of the hearing centre, but it seems pro-

bable that the hearing apparatus has an analogous anatomical structure and physiological arrangement as the optical. The experiments of Munk and Larionow will show that the extirpation of certain places in the hearing centre produces a partial deafness to certain tones. However, their experiments do not concord as to the localisation of the high and low tones. As to the senses of smell and taste, the question does not so much concern a localisation in the centre of the corresponding sensation, but the quality and intensity of the irritation that is a necessary condition for the animals when looking for and collecting their food. It is likely that those central surfaces are arranged — mutatis mutandis — in a manner analogous to the others.

Consequently, the investigation of the sensory surfaces, their localisation and organisation, their centrifugal and centripetal connections are the next fundamental problems. Only after the solution of these problems, we can hope for a further progress in the way to find out the mechanism of thought.

The central and periferal sensory surfaces, retina, Corti, etc., ought to work in analogous manner. Retina having received an optical impression, immediately transmisses this irritation to the nerves, and becomes ready to receive new impressions; in the same manner, the cells of the central retina in the calcarine fissure ought to work. The impression received will be carried out and transmissed to a higher psychical surface on the lateral occipital surface, the place of visual representations, and probably the recollections. Without such a mechanism, the very small central visual surface would be overfull of the immense visual impressions, overstrained and inable to receive new impressions from the eye.

Already a priori, Niessl von Mayendorf's hypothesis that the primary sensory centres are not only a receiving apparatus, but also places for further elaborations of higher psychical perceptions and magazines of recollections, does not seem to be acceptable, and is in opposition to well

known and proved facts. The surfaces of the representations are placed in the neigbourhood of the sensory centres; the optical fields are localized on the lateral occipital surface, the acoustic probably in T^2+ T^3, the tactile probably behind the gyrus centralis posterior (parietalis ascendens) in the superior and inferior parietal convolutions (P^1, P^2).

Our representations and memories are composed of sensory elements or energies generally from two or more senses, which are transformed in order to built up new psychical elements. By association of these higher psychical elements, new psychical forms arrise, with the purpose of forming thoughts. The elements of the primary perceptions and of the sensory representations are locally formed, fixed and deposed in form of recollections.

Through new transformations and associations of elementar representations, higher forms of complicated representations are built up, and these enter as psychical moments in the forming of thoughts. How many such transformation or association places exist is not hitherto known, Nor has the place of this welding of the more simple psychical elements to thoughts been known hitherto. But it seems probable that the frontal lobe is the focus of welding, where the different psychical elements from all sensory centres are collected in transformed and associated forms, and where the highest elaboration of thought takes its place.

We think in form of sensory representations or words, the painter in visual, the musical man in acoustic representations, the mathematician in mathematical formules, figures or letters, etc.

And even moral conceptions and sensations are often dressed in optical, acoustic or other sensory dresses and forms.

The realisation of these very complicated psychical operations presumes the presence of a series of psychical instances or mechanisms, one above the other, in which the primary sensory impressions or irritations are elaborated and transformed by degrees until they can enter as higher

psychical elements and form higher and higher psychical compositions.

Of course the theory requires psychical centres of different height and localisation, where the psychical energies undergo different transformations, quite as the iron-mineral, only by a series of transformations, becomes the finest instruments, which is suitable for the finest purposes.

Such a conception of the brain-mechanism is in full concordance with the development and organisation of the nervous system in the animal kingdom. In the lower animals there exist only reflex mechanisms. The higher the animals are, the more complicated mechanisms are developed, one over the other, until the great brain, cerebrum, rules the inferior mechanisms.

From these conceptions it will be clear that the discovery of the organisation of the primary sensory centres is of very great importance. Flechsig, for instance, says 1905: "die Auffindung der Sehsphäre bedeutet einen eminenten Fortschritt ... Ihre Verhältnisse sind thatsächlich massgebend für die Gesammtauffassung der Sinnessphären, ja der Rinde überhaupt".

However, Monakow, the eminent authority about brain knowledge, has denied this conception of the elementar functions of the brain, as well as the necessity of a cortical sensory apparatus in the brain, supposing that the brain immediately, without intermediation of sensory centres, receives the sensory impressions on large surfaces, Nor he is inclined to accept any projection of the senses, uttering in his latest great book on the localisation in the brain: "Die von Wilbrand und Henschen gelehrte Lokalisation, es müsse in Occipitallappen ein anatomisch scharf abgegrenztes optisches Wahrnehmungsfeld (in der Regio calcarina) und ein optisches Erinnerungsfeld (laterale Occipitalwindungen) unterschieden werden, kann nur ein klinisch-diagnostisches Interesse beanspruchen, physiologisch ist sie völlig unhaltbar". (s. 438).

As to the criticism of Monakow's doctrine, I refer to

the original text, (p. 462, and to my last paper (Über das Sehcentrum, Neurol. Centralblatt 1917, Dec.)

Here I allow myself to remark that Monakow in different places of his books takes very different positions to these important questions. His contradictions are so many and often repeated, that there is no more any meaning to combat or criticise them.

As to the hearing centre he has in his last book taken up an idea of mine from my earliest works, and especially from a paper, read in Rome 1894 (Revue ophthalmologique 1894), and afterwards published in Lewandowsky's Handbuch 1910, 1912. I have, since more than 20 years, defended the conception that the visual centre only transitorily receives the visual impressions in order to transmiss them immediately to other surfaces. Now I see that Monakow has applicated this idea on the hearing centre, and nearly used my words of 1894, and however he assumes that my doctrine is "völlig unhaltbar", adding: "Leider reichten aber bei derartigen Versuchen weder die Vorstellungskraft noch die biologisch-physiologischen Kenntnisse der Autoren (Henschen, Flechsig, Ramòn y Cajal) aus, um diese (Monakow's) Lokalisalionsweise durch physiologische, anatomische oder pathologische Thatsachen näher zu begründen und zu vertiefen (S. 438).

After my view, the memories and conceptions (Begriffe) are formed by an association of more elementary psychial perceptions and sensory representations. According to Jendrassik, "die akustischen Erinnerungsbilder sind demnach als in Zellgruppen successiv aufgenommene Eindrücke aufzufassen", und "die ganzen Worte sind lokalisiert, Teile der Sprache, nicht des Wortes.„ Every cell of course receives one unity. This hypothesis also localizes every sensation, every representation, every conception as a unity in one cell or cell-group. But the thoughts and sensations are immense as to number and quality, but the number of cells is limited. Consequently I think it is probable that the accomplished thoughts and sensations are not deposed in certain cells, but only certain psychical

elements, through the association of which the thoughts, sensations, etc. are formed, in the same manner as we can form every expression in speech by the association of a very limited number of the 30 letters or sounds af the alphabeth.

But I admit that here we move on a hypothetical ground.

Summary.

From this analysis it may be allowed to conclude that every psychical function presumes some primary local psychical processes in the sensory centres. The primary sensory impressions will be transformed and transmitted to higher psychical surfaces, where the sensory representations are formed, after which they are associated with representations from other senses. These representations or elementary memories co-operate as elements or articuli, though in a higher, transformed form, to the building up of thoughts.

In this theory the presence of different centres of different value are presumed, one higher than the other, of different structure and localisation, adapted to receive and conserve the psychical elements transformed by degrees.

In the temporal lobe there may be found a) a primary receiving centre in T^{tr}, b) a second word-clang-centre, c) a word-comprehending-centre, and coordinated with these, musical and common-sounds centres. Above those centres we have the great association centres, probably in the front brain.

Already 1887, I had expressed ideas essentially of these contents.

An appendix contains a criticism of Niessl von Mayendorf's doctrine as to the nature of the sensory centres in relation to the speech. (p. 468—472).

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Explication of the Figures.

1 – 9. Left hemisphere Nat size.
2. „ „ Sagittal Section of the middle part. I = Insula; $T^{tr.}$ = Gyrus transversus (of Heschl); Operc. = Operculum. Nat. size.
3. Frontal Section, 5 cm, frontal from the occipital pole. Nat. size.
4. „ „ 8 „ „ „ „ „ „ „
5. „ „ 8—9 „ „ „ „ „ „ „
6. „ „ 5 „ „ „ „ „ „ Microscopical Section, Double size.
7, 8. Frontal Section, 7—8 cm, frontal from the occipital pole. Microscopical Sections, Double size.
9. Frontal Section, 8—9 cm, frontal from the occipital pole. Microscopical Section, Double size.
10. Fig. Right hemisphere. Nat. size.
11. Frontal Section, 6—7 cm, frontal from the occipital pole. Nat. size.
12. „ „ 9 „ „ „ „ „ „ „
13. „ „ 10 „ „ „ „ „ „ „
14. „ „ 7 – 8 „ „ „ „ „ „ Microscopical Section, Double size.
15. Frontal Section, 8 cm, frontal from the occipital pole. Microscopical Section, Double size.
16. Frontal Section, 8 – 9 cm, frontal from the occipital pole. Microscopical Section, Double size.
17. Frontal Section, 8—9 cm, frontal from the occipital pole. Microscopical Section, Double size.
18. Frontal Section, 9 cm, frontal from the occipital pole. Microscopical Section, Double size.
19. Frontal Section, 10 cm, frontal from the occipital pole. Microscopical Section, Double size.
20. Frontal Section, 12 cm, frontal from the occipital pole. Microscopical Section, Double size.
21. Frontal Section, 13 cm, frontal from the occipital pole. Microscopical Section, Double size.