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A CASE OF PURE WORD-DEAFNESS WITH AUTOPSY[1]

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The following study is of a case which presented clinically a nearly classical example of that disturbance of speech which, from the descriptions of Lichtheim and Liepmann has been called subcortical auditory aphasia or pure word-deafness. It was possible in this instance to obtain a thorough examination of the subject's speech capacity and later to study the brain by the method of serial sections. For the clinical notes I am indebted to the physicians of the Danvers Hospital.

The subject was a man named Taft, possessed of ordinary mental capacity, evidently sufficient for the conduct of a successful lumber business. He was right-handed, and until he was about 45 he had enjoyed good health. At that time, while walking on the street, he suddenly lost the use of his legs, and for a period following he was unable to walk. He later on was able to get about quite well, but there persisted a certain amount of ataxia in his leg movements. He continued at his business until he was 52 when he gave up work. For some years previous there had been developing an increasing crankiness in his disposition and he showed a number of eccentricities of conduct, such as doing little things to annoy his family—as shouting, whistling and slamming doors. In more recent years these peculiarities became more pronounced. There is a general agreement among his friends that up to within ten days of his admission to the hospital, he retained most of his mental capacity. His memory was

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impaired, but if humored in his whims he was agreeable and well informed regarding current events.

When he was 67 he had what was described as "some sort of a turn in which he became decidedly worse." A few days later he experienced another attack in which he lost the power of speech. Whether or not he was unconscious in either of these attacks is not known. What he spoke could not be understood, wrong words were used in his sentences and he could not understand what was said to him.

For many years previous to these attacks he was regarded as having been totally deaf in his right ear. He and his daughter declared that this followed the explosion of a gun. Hearing in his left ear was not impaired until the present attacks. While during the previous year a slight slurring of speech had been noticed, no such disturbance as that which now occurred had been present. He had always spoken so that he could be readily understood. Ten days following these attacks he came into the Danvers Insane Hospital.

The physical examination made at his admission demonstrated a certain increase in the size of the heart, accentuated second aortic sound and irregular rhythm.

The neurological abnormalities were: inability to stand or walk; marked ataxia in his leg movements; upper extremities quite well controlled; the pupils were small and unequal, the left being larger than the right; although the reactions were difficult to test they seemed to react slightly to direct light stimuli; the knee-jerks were absent; tests for sensibility so far as could be determined did not show any abnormalities, except that he complained much of sharp pains in his legs.

The disturbances of his speech had at all times since his admission been most striking. He was wholly unable to understand anything spoken to him. Communication was only possible through gestures and writing. A systematic examination of his speech capacity was not made until some time later, but the notes made from time to time gave much information regarding the aphasic disturbances. At his admission he had some difficulty in reading the written questions, but with continued efforts he understood them in about one-half the tests.

While he never comprehended spoken words, he reacted quickly to sounds. He knew where he was, but instead of the name Danvers, he said "Dabbers, Dibbers," and finally "Danvers." That he knew the location of the hospital is evidenced in his reply, "Yes, that is it right here up on the hill."

He appreciated his inability to understand what was said to him and his difficulty in expressing his own thoughts. He easily became exasperated at his failure to speak as he wished. His first attempts in spontaneous speech were usually more or less jargon, but repeated efforts showed a progressing improve-

ment and eventually he expressed himself quite intelligibly. With some difficulty in expression he gave the date correctly, recalled that he came to the hospital on the day before, and in telling of his experiences in coming, said, "No, they bribed me here without consulting me."

When shown the written question, "How long have you been here?" he replied, "Well, I have been troubled a good many years; had this ten weeks; had trouble with my debs, don't wear here." His home had formerly been in the vicinity of Danvers, and in speaking of his son-in-law, said, "Don't you know the Harry Debber, down to Dibber, his wife is my son." He spoke of his daughter as "Katheribed, Caribed." He became exasperated at his failures, which he recognized keenly, and said, "I am kinder blue eyed, mixed kind of wide." To the question asking him if he knew the building he was in, he replied, "Yes, it is Purrington, I don't know just how to pass it out just now. I could think of it yesterday. It just comes for a moment and then bids back just the name."

During the next two months the notes record that he showed no marked change. "The aphasic condition persists, and he is at all times irritable and fault-finding." He sat in an invalid chair, unable to walk and he complained much of pains in his legs.

February 1, 1902. Some edema of the legs. Word-deafness complete. In referring to his ear, said, "I wish some one would sear this ear out. I want it combed onced. There is a little waxing in the egg."

March 13, 1902. Considerable improvement in his ability to speak more correctly. He hears sounds but cannot understand any word spoken.

April 15, 1902. Further improvement in spontaneous speech. He talks quite clearly, and with very few mistakes. He is totally unable to understand any spoken words, but he reads accurately with perfect understanding.

July 31, 1902. He speaks with very few mistakes. He writes legibly, but continues totally word-deaf.

April 2, 1903. Severe pains in his legs. Marked delay in reaction to pin pricks in legs. His spontaneous speech is more correct than at any previous time. He wrote a brief note quite coherently.

November 6, 1903. SPEECH EXAMINATION.

On this date a detailed and systematic examination of his speech capacity was made. A careful examination of his peripheral hearing capacity was kindly made by Dr. de Wales.

Left Ear.—Drum membrane generally thickened. Short process prominent. Posterior fold prominent. No light reflex. Sound reactions were as follows: Single and double vibrations of tuing fork c″ (512) and c‴ (1024) were plainly heard. Rinne 14/6. With Weber test he gives no response, in one test he said

that he heard it in his head but did not seem to localize it. Pollitzer's acumeter test gives air conduction at one inch and bone conduction at one inch.

Right Ear.—Drum less generally thickened than the left, but has islands of thickening around the periphery. Tests with tuning forks vibrating between c″ (512) and c‴ (1024) and the Galton whistle showed total absence of tone hearing. Rinne's test o/o. Laryngoscopic examination of the vocal cords showed movements in phonation to be unimpaired. The mucosa was a little reddened.

Dr. de Wales's opinion was that there was a chronic catarrhal condition of the left ear with very good hearing of the higher tones but none for the lower tones. The right ear was completely tone-deaf by reason of labyrinth disturbance.

The determination of whether the inability to understand spoken words is the result of peripheral or central disturbance is of fundamental importance in this type of aphasia. From the investigations of Bezold it is known that if as much of the tone scale as lies between b′ and g″ is preserved, a sufficient range of tone exists for the understanding of spoken words. The examination in our case shows that although the right ear is completely tone-deaf from labyrinth disease, there is preserved in the left ear a tone perception continuous from c″ to the Galton whistle. For some reason there is no statement in the record of tone perceptions below c″ (512). Although the lowest tone necessary for understanding of auditory speech is given by Bezold as b′ (480), this is but one tone lower than c″, and as it was remarked in the notes of the examination that c″ was plainly heard, there seems to be evidence that there was preservation of the required tone range in the left ear, and that the condition was central rather than peripheral.

Reaction to Words and Sounds Heard.—Although totally unable to understand a single word spoken to him, he reacted to sounds. In the ordinary intercourse with him on the ward, his usual reply, when one spoke to him, was, "I don't know what you say. I can't tell." He always gave good attention and his attitude was one of perplexity at his inability to grasp what was said. In the examinations the questions were written or printed and always were read with understanding. His attention could be gained by noises made near him. When blindfolded he quickly gave attention to the whistling of some one near and spoke of it as a noise. Similar reaction followed the ringing of a bell. The rattling of keys he called "a crackling." The imitation of the cry of a cat he spoke of as "singing." The barking of a dog was called a "noise." Several tunes were whistled near his left ear; although he gave careful attention, none awakened any certain recognition. As in all attempts to recognize spoken words, his face had a troubled, puzzled look, and often he would shake

his head at his inability to grasp the interpretation of sounds he heard. On one occasion there seemed to be a question as to whether he did not recognize the whistling of Yankee Doodle.

Spontaneous Speech.—In response to the request that he give an account of his trouble, replied, "What do you mean, my head here (pointing to his head), or here (pointing to his knees)?" It being indicated that the request related to his difficulty of hearing, he replied that he could hear all right; that thirty years ago a gun was fired close to his ear and for the past two years he had been entirely deaf. In attempting to repeat the alphabet he did so correctly as far as g, he then hesitated and again began, getting as far as v, he then was puzzled and unable to proceed, he remarked, "That is funny, by George, I can't get it." With the third attempt he gave it readily without mistake from a to z. The days of the week and the names of the months were promptly and correctly given. He regarded the requests as foolish and occasionally appeared impatient and disgusted, as when asked to count from 1 to 20, he laughed and remarked, "Well, I ain't a fool." He then counted correctly but repeated 15 twice. He counted backward from 20 to 1 correctly.

Calculation Tests.—$8 \times 4 = 32$; $9 \times 7 = 63$; $12 \times 11 = 132$; $96 \div 8 = 12$; $32 \div 4 =$ he hesitated, looked in a puzzled way at the examiner and remarked, "I don't understand what you mean." On being urged he gave the answer as 23; again gave 23 and then remarked, "I don't know what you mean, you mean divide it? Why, 32." The requests to repeat spontaneously the Lord's Prayer, or some familiar verses, caused him to laugh and reply that he could give none as he had forgotten them.

His capacity for spelling was tested by requesting him to correct words which were written misspelled. He recognized and corrected the mistakes in the words "Massachusetts" and "received." When shown various objects and requested to spell their names, he gave the following reactions:

Objects.	*Spelling.*
Wallet	+
Cigarette	+
Pencil	Called it "pencil" but spelled it "pncil"; then three times he spelled it "pecil," each time pronouncing it as he had spelled; in fourth attempt it was correctly pronounced and spelled.
Dipper	+
Boat	+
Tongue	Pointing to the tongue, "Why, my table, why, my touble," and spells it "t-o-u-b-l-e"; at another attempt

	he pronounces and spells the word correctly.
Handkerchief	+
Pillow	+

In all the tests the name of the object was spelled as he pronounced it.

Reaction to Things Seen.—

These tests were made by showing him either actual objects or their reproduction in pictures and requesting that he name them. There was no difference in his recognition by either method:—

Objects.	*Named.*
Pencil	+
Wallet	+
Keys	+
Flowers	+
Cabbage	+
Tomato	+
Beans	+
Cat	+
Celery	+
Automobile	+
Brush	+
Scissors	+
Tree	+
Basket	+
Elephant	+
Deer	+
Fish	+
Snake	+
Windmill	"Millbury, millwill, wingmill, windmill."
Butterflies	"Huckleberries, butterflies."

Portraits of Lincoln, McKinley and Pope Leo were correctly identified and named.

The mistakes made in naming the last two objects were not recognized, but after several attempts were correctly given. The dissatisfaction of the examiner, at his calling butterflies "huckleberries" seemed to puzzle him. He repeated the same replies several times and then said, "I can't give the name, it ain't clear to me." On a fifth attempt he gave the correct name.

To all questions regarding the use of objects he gave correct information.

Reaction to Things Felt (Eyes Being Closed):

Right hand—

Objects.	*Named.*
Button	+
Pencil	"Paycil."
Watch	"Feels like a watch."
Chair	+
Toothpick	"I don't know what it is, it feels like a stick."
Coin	+
Pocketbook	"Is that anything to eat? A purse."
Bell	+
Paper	+

Left hand—

Pencil	+
Keys	+
Watch	+
Stethoscope	Feeling it with both hands, "Well, I don't know the name of it. I don't know what it is." After looking at it with his eyes open, it was evident that it was an object unfamiliar to him.

Letters and figures traced on the skin of his forehead were not recognized.

Reaction to Words Seen:

Reading.—He read both printed and written words. The written or printed requests in all tests were readily read, either aloud or silently. In reading aloud from books and paper, he read perhaps somewhat less rapidly than one would expect, from his previous habits, and paraphasic answers occasionally were given. These always resembled the correct word in sound and were quite similar to the mistakes made in the previous tests in naming objects and in the examples of spontaneous speech given in the foregoing notes. He was a constant reader of the daily paper and on request would tell what he had been reading. All tests showed that he read with understanding.

Understanding signs read:

Y. M. C. A.	"Young Men's," the latter part of his reply was paraphasic and not understood.
Y. M. C. U.	+
I. O. O. F.	+

A. O. U. W.	His first reply was paraphasic and not understood; he then remarked, "I have forgotten what it is."
F. & A. M.	"American."
N. H.	+
R. I.	+

A series of numerals were correctly read. When a series of arithmetical problems, in which the signs of +, ×, — and ÷ were used, he showed some difficulty in readily understanding what the problem meant, but on repeating the tests the correct results were usually obtained.

Writing (Spontaneous).—In response to a request that he write an account of his sickness, he produced the following:

"Nov. 6.

"Dr. M.—

"You asked for my case, nearly twenty years ago. I was taken by locomotor ataxia, I got around by a cane until the last three years, and then used a crutch. A little over two years I became deaf and my brain cloudy and was sent here in December 1901. My brain is now fairly clear. I never had a headache, since I was 14 years old with a fever. I have much pain in my feet and legs mostly at night. For the last three months my water has made me much trouble a passing much in my bed at night.

"Fred A. Taft."

Many of the characters in this letter were sprawling and the handwriting was quite characteristic of the ataxia of tabes. A few words are lacking to complete some of the sentences, and in one instance the article a was used in an incorrect relation, but it gives no evidence of any marked disturbance of an aphasic nature.

Writing from dictation was absolutely impossible.

Drawing.—Requested to draw a house, he started to draw a horse, then stopped, and after reading the request, laughed and made a very fair start to draw a house. A tree and a cat were drawn promptly.

Copying was done correctly.

Understanding of Music.—There is no certain evidence that he understood tunes. Any detailed tests with musical instruments or of his ability to sing were not made.

The Internal Language.—In drawing conclusions from the various tests in other fields there is no evidence that there was any serious defect in his internal language.

Apraxia.—He showed on all occasions a correct appreciation

of the proper use of a variety of objects, and used them without error excepting for the ataxia in his movements.

Understanding of Colors.—He was able to name colored yarns with but occasional errors. In a few instances, there was a slight paraphasia in his answers:

Color.	*Reaction.*
Red	+
Pink	+
Blue	+
Red	"Pink."
Brown	"Drab, brown."
Orange	+
Yellow	+
Purple	"Pupil."
Green	"Light green."
Black	"I don't know what color it is"; on a second attempt he calls it "brown."

SUMMARY

Reaction to Words and Sounds.—Total deafness to words spoken; but gives attention to sounds; no ability to recognize meaning of sounds heard; no ability to repeat words heard.

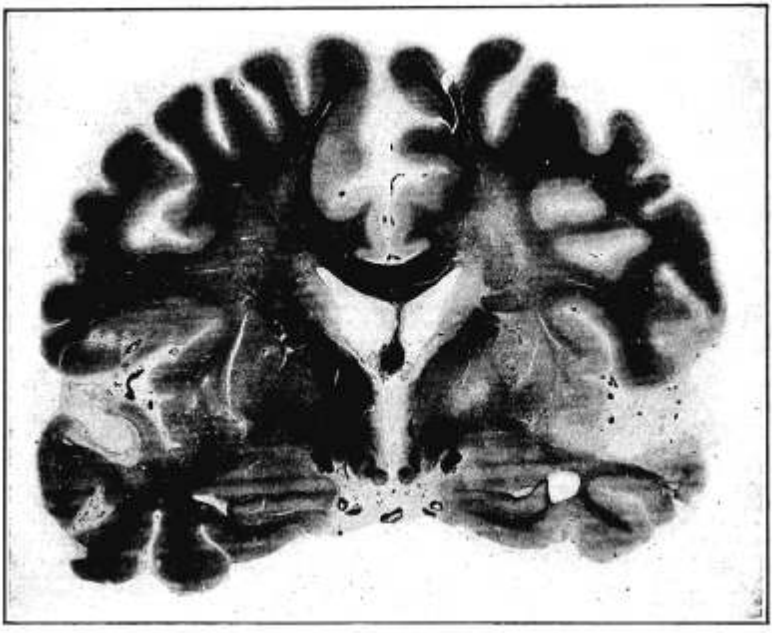

FIG. 1.

Spontaneous Speech.—Retained ability to speak spontaneously, with rare paraphasic utterances; occasional inability to speak readily the word desired, but later always giving the correct reaction; calculation fair; spelling good except for occasional paraphasia; spelling good for words pronounced.

Reaction to Things Seen.—Objects correctly recognized and named except for an occasional paraphasic reply; mistakes in pronunciation not recognized; correct color recognition.

Reaction to Things Felt.—Good for familiar objects; an occasional paraphasic reply.

Reaction to Words Seen.—Read printing and writing understandingly; unimpaired reading except for an occasional paraphasic reply; meaning of familiar signs recognized; slight difficulty in readily understanding meaning of arithmetical signs.

Writing.—Spontaneous writing and drawing ability retained; ataxia (tabetic) in writing movements; no ability to write from dictation.

Internal Language.—No evidence of impairment.

During the next six months his speech disturbance showed no change. He suffered much from cystitis and pains in his legs.

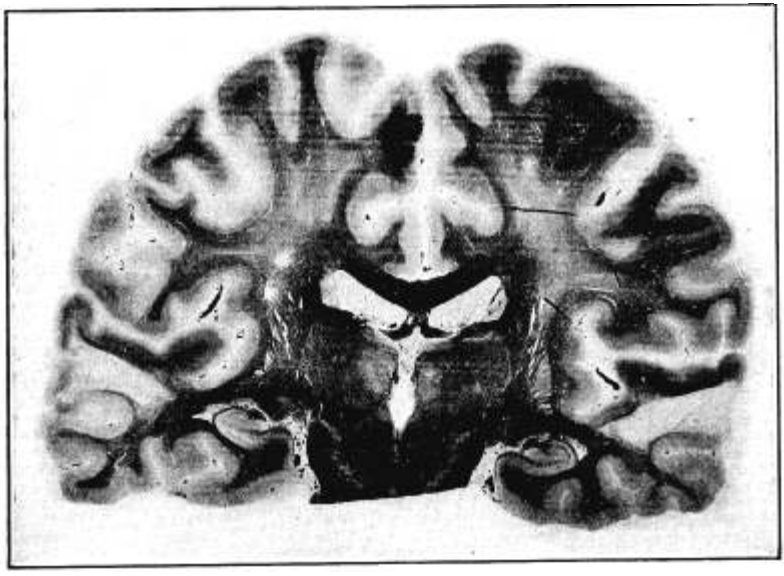

FIG. 2.

July 8, 1904. He continues totally word-deaf; he is able to read and has a good understanding of what he reads. His spontaneous speech exhibits rarely some paraphasia, a word being occasionally used in wrong relations. His memory for events from day to day is fairly good.

February 24, 1905. His physical health has failed much, but his mental condition remains much the same. He shows some difficulty in performing mathematical calculations requiring any considerable mental effort. All objects shown to him are correctly named. His written letters show grammatical correctness; his ideas are clear, but his vocabulary shows appreciable limitations. In reading aloud written directions he will rarely miscall a word, and in speaking spontaneously he sometimes makes a similar mistake.

On October 17, 1905, he fell dead suddenly, from the rupture of an aortic aneurism.

At the autopsy, held a few hours after death, the chief gross findings among the body organs were: Extensive recent hemor-

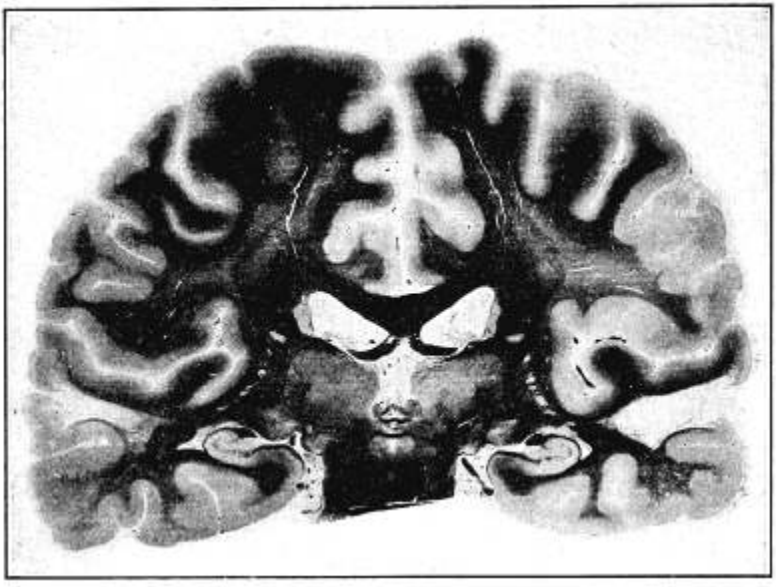

FIG. 3.

rhage into the right pleural cavity; a large aneurism of the thoracic aorta, with a recently eroded opening in its walls (the inner wall of this was covered with old and recently organized fibrin clots, and presented numerous calcareous plaques); mitral insufficiency; cardiac hypertrophy; chronic fibrous myocarditis; cirrhosis of the liver; cholelithiasis; purulent cholecystitis; chronic interstitial nephritis; purulent prostatitis; chronic cystitis.

The gross examination of the brain showed severe atheromatous degeneration of the arteries at the base of the brain. Both middle cerebral arteries showed scattered atheromatous patches.

The pia mater was transparent and delicate, excepting in the regions of both Sylvian fissures. There were residuals of old softening in both temporal lobes. In the fresh brain the regions of the right and left first temporal convolutions were sunken inward, and the pia intimately adherent to the softened areas. The limits and more exact localizing of these softenings was worked out from serial sections and will be described in another place.

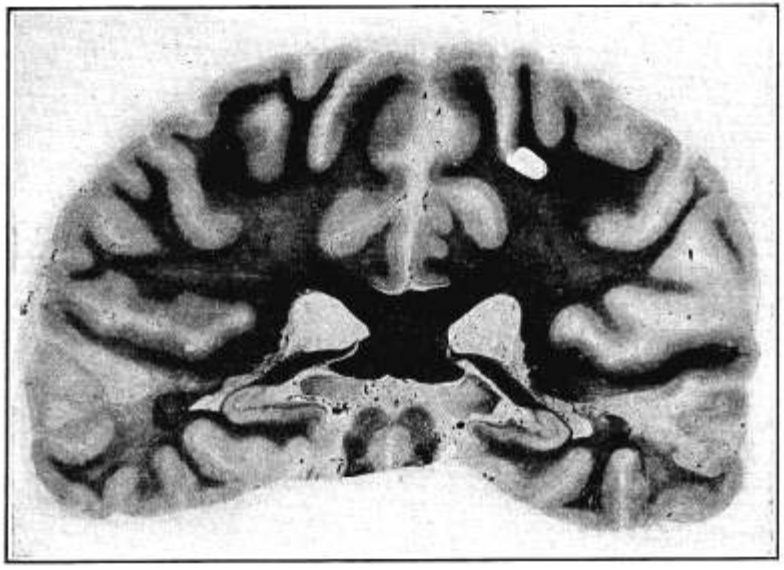

FIG. 4.

The brain was at once put into 10 per cent. formaline solution and after six weeks was divided by frontal cuts into a number of blocks, which later were cut into serial sections varying from 75 to 100 micra in thickness and stained by the Weigert-Pal method. These sections formed the basis for the descriptions following. The cranial nerves showed no gross abnormalities. The spinal cord presented a gray streak along the dorsal columns in the lower dorsal and lumbar regions. Subsequent histological study of the cord showed a characteristic tabetic posterior column degeneration.

The study of the serial sections demonstrated that only an incomplete idea of the extent of the lesions and their exact location was possible in the uncut brain. While both right and left temporal lobes were involved, it was found that on the left side the lesion was peculiarly limited to certain portions of the temporal convolutions, the relations of which were determined by subsequent reconstruction of the lesion in its entirety from the serial sections.

The relations of the lesions are well demonstrated in the photographs of the sections pictured in Figures 1–4. Fig. 1 is of a section passing a little posterior to the beginning of the involvement of the left T_1. On the left side the entire lower lip of T_1 is destroyed, including cortex and the larger part of the center of the convolution. The outer one-third of the dorsal surface is gone. All that is preserved is the inner two-thirds of the cortex with a narrow strip of underlying fibers.

The destruction further involves the cortex at the bottom of T_1 fissure and the greater part of the cortex of the dorsal surface of T_2. In both places the underlying fibers are more or less affected. On the right side the lesion extends farther forward than on the left and in this section all of T_1 is absent and the adjacent angle of the island shows a defect in the cortex and underlying fibers.

Fig. 2 is of a section passing through about the middle of the

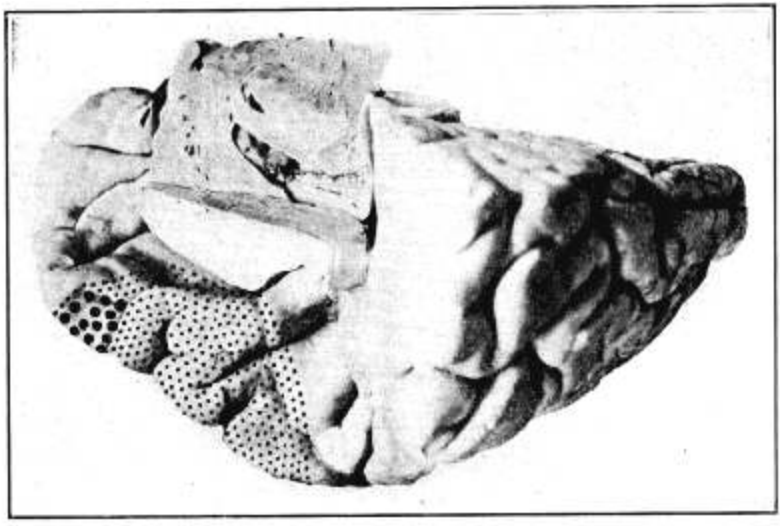

FIG. 5. Left temporal lobe, dorsal surface. In this and in Figs. 6, 7 and 8 the large dots cover those areas where the cortex and underlying fibers were destroyed. The smaller dots cover areas of cortex beneath which fibers in the center of the convolution were destroyed, but the cortex where dotted was intact.

transverse convolutions on the dorsal surface of the temporal lobe. The sections between Fig. 1 and Fig. 2 demonstrate that the lesions in their posterior continuation progressively receded from the dorsal and outer surfaces of T_1, both of which in this section, together with a considerable number of the fibers of the center of the convolution, are preserved. On the left side the

destruction of T_2 is more extensive than farther forward, and in this section all of the cortex of the dorsal surface and most of the center of the convolution is absent. There remains preserved only the cortex of the outer surface and all of the ventral surface with a narrow layer of adjacent fibers. In this and in all other sections the cortex which remains, even close to the margin of the defect, shows no disturbance in the number and arrange-

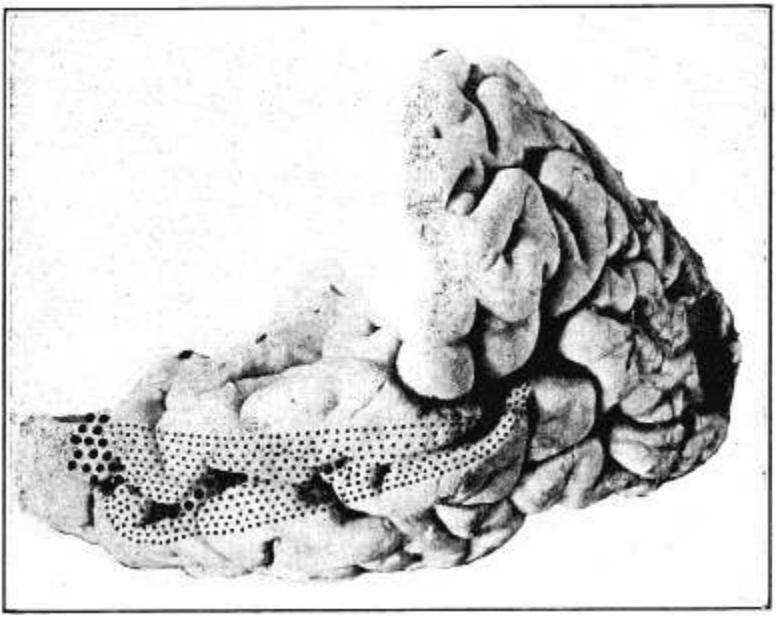

FIG. 6. Left temporal lobe, outer surface.

ment of nerve cells, and no certain loss of radial or tangential fibers. At the bottom of T_1 fissure, a triangular area extends inward; in this area fibers are greatly diminished in numbers. On the right side only the cortex of the ventral surface of T_1 is involved. T_2, as on the left side, shows the greater destruction and here is all gone except the cortex of its ventral surface and a thin layer of adjacent fibers. The deep fiber areas at the bottom of T_1 fissure show absence of many fibers. The involvement of the island terminated some little distance anterior to this section.

The section pictured in Fig. 3 passes through the extreme posterior end of the transverse convolutions and island and through the geniculate bodies. On the left side the cortex of the ventral surface of T_1 is defective. The cortex of the upper surface of T_2 and most of the fibers of the center of the convolu-

tion are absent. The deep fiber areas of the lobe show beneath the bottom of T_1 fissure great diminution in numbers and the degeneration continues inward as a thin streak traceable as far as the lenticular nucleus. This streak occupies the position of the fiber radiations between the internal geniculate body and the first temporal convolution.

The condition on the right side is much the same. The internal geniculate bodies on both sides give no evdience of degeneration changes. Their cells are of normal appearance and their fibers abundant and deeply stained.

Fig. 4 is of a section passing posterior to the island, and through the extreme posterior end of T_1. On the left side the lower surface of T_1 shows an absence of a narrow strip of cortex. The upper surface of T_2 is destroyed together with a considerable portion of the center of the convolution. The deep fiber area of the lobe is pale, showing great diminution in fibers, and a triangular area of fiber degeneration, in which are a few

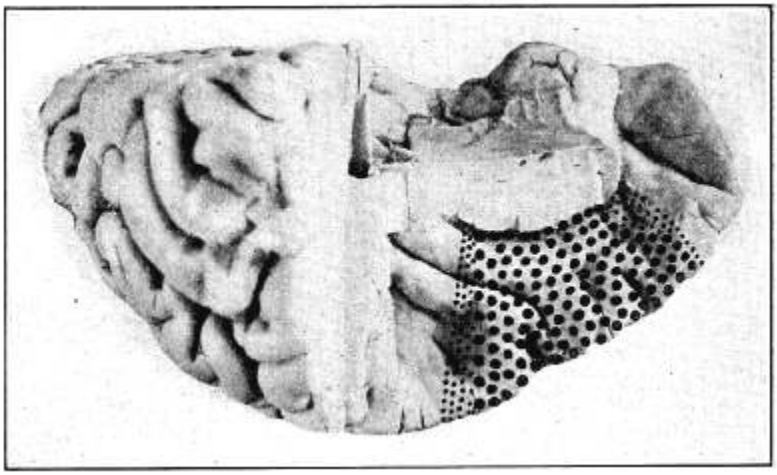

FIG. 7. Right temporal lobe, dorsal surface.

small cyst-like extensions of the softening, which pass inward, cutting through the sagittal strata near to the ventricle.

On the right side T_1 is involved only at the extreme inner portion of its ventral surface. T_2 has the cortex of the inner half of its dorsal surface destroyed and nearly all of the fibers of the center of the convolution. The degenerations in the deep fiber areas of the lobe are much the same as on the left side.

Posterior to this section the involvement of the cortex in both left and right temporal lobes is less. For a considerable distance there is marked degeneration in the deep fiber area of

the temporo-occipital lobes with small cavity extensions of the larger defects. The degeneration in the sagittal strata is present as far posterior as the strata are demonstrable. These degenerations are evidently the result of cutting the fibers by the softening rather than secondary degenerations of the fibers in their origin or termination. Nowhere can it be demonstrated that there are degenerations of the fibers of the corpus callosum.

The relations obtained from reconstruction of the lesion are shown in Figs. 5–8. These are from photographs of the dorsal and lateral aspects of the temporal lobes in which the areas, where the destruction involves the whole thickness of the convolution, have been diagrammed with large dots. The areas marked with the smaller dots indicate the parts of the convolution in which the cortex was preserved but undermined by fiber degenerations.

On the left side, Fig. 5, the cortex of nearly all of the dorsal

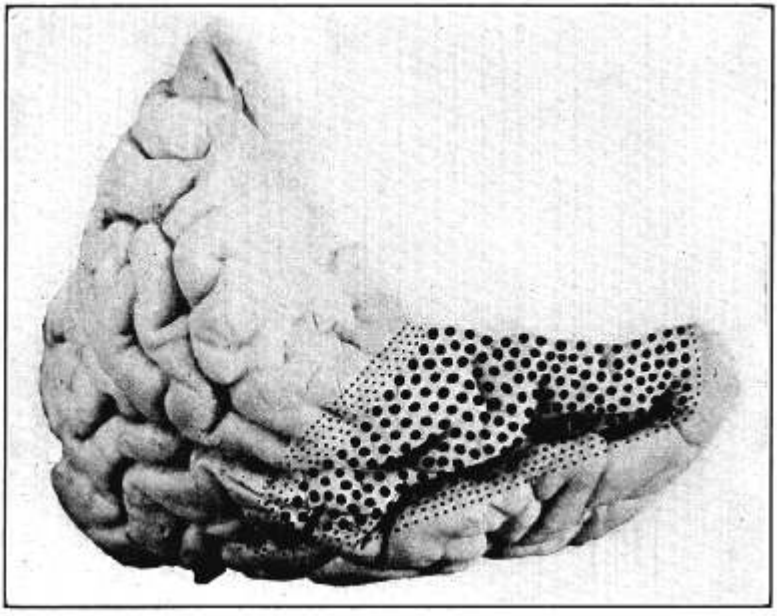

FIG. 8. Right temporal lobe, outer surface.

surface of T_1, including all of the transverse convolutions, is preserved. The sole defect is a small area not more than 1 sq. cm. at the outer edge of the convolution, immediately anterior to the transverse convolutions. Here the defect extends through the entire thickness of the convolution, showing on the lateral surface in Fig. 6. Posterior to this position the involvement of

T_1 is confined to the lower surface of the convolution. Anteriorly the whole of this surface is gone, together with the greater part of the central fibers of the convolution. In its posterior continuation the defect progressively recedes towards the bottom of the fissure leaving more and more of the central fibers preserved. The upper surface of T_2 is largely destroyed, together with most of the central fibers of the convolution, the defect being a part of that involving T_1. In its posterior extent it becomes more limited to the bottom of the fissure. About 1 cm. anterior to the termination of T_1 fissure, no more surface defect is present, the degeneration being confined to subcortical fiber areas.

On the right side T_1 is destroyed to the extent seen in Figs. 7 and 8. The view of the dorsal surface of the lobe, Fig. 7, shows complete destruction of T_1 from near the anterior limit of the island back to the middle of the transverse convolutions. Along the anterior half of the continuity with the island the lesion has cut out the cortex of the island and its subjacent fibers to a depth of 5 mm. The lateral view of the lobe shows complete destruction of T_1 back of the anterior end of T_1 fissure to about where the posterior and middle thirds of the convolution join. The lesion extends through the bottom of the fissure into the dorsal surface of T_2, widening in extent of surface and depth of invasion in its posterior direction. A little posterior to the middle of T_2 it involves the convolution in its entire thickness. Back of this the lesion leaves the cortex free but extends below the fissure into the deep fiber areas of the temporo-occipital regions, along the sagittal strata to near the tip of the posterior horn. On both sides the fibers of the center of T_1 and T_2 are involved for some distance away from the surface defects. In part this appears to be the direct result of the lesion and in part secondary degenerations. The extent of this undermining of the cortex is diagrammed in the figures.

The foregoing descriptions show that a clinically unobjectionable case of pure word-deafness may result from disturbances of the cortex and underlying adjacent fiber regions in parts of the first and second temporal convolutions in both hemispheres.

The number of autopsies reported on well observed and uncomplicated cases of pure word-deafness is not large. In eleven reports which are accessible to me are found three types of anatomical lesions:

1. Destruction of the fibers in the center of the left temporal lobe, in the course of the auditory radiations from the internal genticulate bodies: Liepmann's case Gorstelle (1); Wernicke's case Hendschel, the autopsy in which was recently reported by

Liepmann (2); and a case of abscess in the temporal lobe reported by Van Gehuchten and Goris (3).

2. Destruction of cortex and underlying fiber areas, in the first and second temporal convolutions of both hemispheres. Cases reported by Edgren (4), Ballet (5), and three by Peck (6, 7, 8).

3. Atrophies of the first and second temporal convolutions in both hemispheres. Cases of Dejerine and Sérieux (9), Strohmayer (10) and Veraguth (11).

It is thus evident that in the majority of cases of pure word-deafness the lesion is different in the position from that postulated by Lichtheim and Wernicke for this type of aphasia, viz., a true subcortical involvement. With these differences it is unfortunate that all of these cases were not studied in serial sections, in order that it could be determined what the exact relations of the lesion were to those parts of the temporal convolutions whose integrity is essential for the preservation of word-understanding. In none of the cases is it possible to determine from the descriptions whether or not the relations found to exist in our case are present. The reconstruction of the relations in the left temporal lobe demonstrated that in spite of destruction of a considerable area of cortex in the left first and second temporal convolutions, there is left preserved the area which abundant evidence justifies us in regarding as the receiving station for the auditory radiations. This area is the cortex of the transverse convolutions on the dorsal surface of the first temporal convolution.

The importance of this area has been fully analyzed recently by Niessl von Mayendorf (12) in a consideration of the relation of the temporal convolutions to word-deafness. In this it is shown very convincingly that the middle portion of the left first temporal convolution and more exactly the transverse convolutions on its dorsal surface are of special importance for the preservation of word-understanding. As evidence for this he states the facts: (1) The greater number of lesions associated with loss of acoustic word-memories involve the middle part of the left first temporal convolution and the anterior transverse convolutions. (2) There is a difference in the cortical architecture of this area and adjacent regions of the first temporal convolution. (3) The fibers of this area are myelinated at an

earlier date than others near by and at a time coincident with those of the auditory radiations.

In recent contributions of Meyer (13) and Quensel (14) is given further corroborative evidence of the importance of this area as the auditory receiving station.

In our case the cortex of the transverse convolutions in the left hemisphere was intact, but the convolution was undermined throughout nearly its entire length by the degenerations in the fibers of the center of the first temporal convolution, large numbers of which had been destroyed in the softening, and there also existed a very considerable streak of degeneration in the course of the radiations between the geniculate bodies and this convolution.

In view of this the lesion present may in its anatomical relations be regarded as subcortical, at least in the manner it affects the part of the temporal convolutions essential for word-understanding. In this case as in a number of others, there is also involvement of the right temporal convolutions. This fact of the occurrence of lesions in both temporal regions in conditions of pure word-deafness has influenced Pick and some others to find the explanation for this type of speech disturbance in the general diminution of central hearing capacity which would result from such extensive injury to the cortical relations of both auditory nerves. That the existence of lesions in both hemispheres is not essential for the production of pure word-deafness is evident from the clear cases of Liepmann, and Van Gehuchten and Goris, in which there was preservation of sufficient extent of tone range, for the understanding of spoken words, but a pure word-deafness existed with lesion of the left hemisphere alone.

This fact together with the known importance of the left hemisphere for speech function and the very extensive destruction of the transverse convolutions in the right hemisphere in our case of Taft, has seemed to us ample justification for regarding the involvement of the left auditory relations as the important one for the production of the pure word-deafness; and that the condition is the result of the isolation of the receiving station in the transverse convolutions in the left hemisphere by an anatomical lesion affecting its fiber relations with the internal geniculate body.

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