

A CASE OF APHASIA WITH LOSS OF MEMORY OF NOUNS,  
(SENSORY ANOMIA) WITH AUTOPSY.

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*Introduction.*—The expression “naming center” has been used in describing cases of aphasia with loss of memory for nouns. The term was first suggested by Broadbent in 1872. In evidence of the existence of such a center a case of tumor involving the under surface of the temporal lobe, and particularly the third temporal convolution, was reported by Dr. Mills.<sup>1</sup>

Collins<sup>2</sup> refers to optical aphasia, but criticizes the psychology of Broadbent's conception and attacks the value of Mills' case.

A somewhat similar form of trouble was described by Freund, in 1899, and called by him optic aphasia. In optic aphasia the patient, when an object is presented, can recognize it, knows its use and property, but can not give it the name. When, however, he touches, tastes, or smells it, he can pronounce the name. Starr<sup>3</sup> prefers to call conditions of this kind “intercortical sensory aphasia.” In such type of aphasia the patient is (*a*) unable to recall the name of a thing seen, or (*b*) to picture to the mind the appearance of a thing named, yet the name is recognized when heard and the object is recognized when seen. There is, theoretically in these cases a lesion of the association tract which connects the temporal and occipital lobes.

J. Vorster<sup>4</sup> reports a case of optic and tactile aphasia, and gives the history and literature of so-called optic aphasia. He collects also brief observations of eight cases that have been reported by others. A study of the cases shows that while bearing a general resemblance, in that things seen can not be named, there are a good many differences in the extent of the amnesia and in associated symptoms. Pure optic aphasia is certainly rare. There is often more than optic and tactile aphasia.

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<sup>1</sup>Mills: “The Nervous System and Its Diseases,” 1898 page 345.

<sup>2</sup>Collins: “The Faculty of Speech,” page 287.

<sup>3</sup>Starr: “Organic Nervous Diseases,” page 466.

<sup>4</sup>Vorster: Archiv. f. Psychiatrie, 1898, Vol. XXX, page 341.

In the case we are to report there was optic-tactile-gustatory-olfactory- and sound-aphasia, and, on the other hand, an object named gave rise to a visual image which the patient recognized. So that it was neither a pure optic, nor an intracortical optic aphasia.

*History of Case.*—Summary: Male, 48; syphilis fifteen years before, illness of three years; no paralysis, some mental dullness, no paresis, aphasia with inability to name objects seen, felt, heard, tasted or smelled; voluntary speech good, ability to understand good; softening of first temporal cortex and subcortex on left side.

Morris E., aged 48; pedlar; born in Russia.

*Family History.*—Father died at age of ninety-three, mother at 40; cause unknown. Had healthy sisters, and two healthy children. Three children died in infancy. No miscarriages.

*Personal History.*—Fifteen years ago had a tumor in the back of the neck which was removed three years ago. Is a moderate drinker and excessive smoker. Fifteen years ago had luetic infection, with secondaries. Treated with inunctions and internally.

*Present Illness.*—About three years ago and three weeks after the operation on his neck he noticed some pains in the neck and weakness in hands and feet, chiefly in the right hand and left leg. He had some difficulty in urination, some constipation, some headache and sleeplessness, but no paresthesias. A few months ago, owing to dysuria, sounds were passed, causing urethral fever. On admission he complained of weakness in right hand and left leg, constipation, some difficulty in urination, poor appetite, poor sleep, poor memory, inability to express himself correctly.

*Status.*—Patient is well built, well nourished, of healthy color. Gait shows a limp, the left leg being used like a wooden leg, that is without flexion at the knee. Mental condition is dull and apathetic, speech shows aphasia which will be described later.

Examination of motor system shows some nystagmus on looking to the right. Pupils slightly irregular, equal, moderately wide, direct reaction sluggish, consensual absent, accommodation and convergence reaction present. Left side of face somewhat better innervated than right. Movements of head painful, but not limited. Motor power of right arm diminished. No atrophy, Motor power of left leg diminished, also of the right to some extent. Reflexes of the arms are increased in left, diminished in right. Mechanical irritability shows some slowness in the left, and exaggeration in the right. The epigastric reflex is lively. Abdominal reflex diminished. Cremasteric reflex lively. Gluteal

and interscapular reflexes diminished. Knee-jerks lively, especially left. Achilles jerk increased. Plantar lively on both sides and flexor response. Easily exhausted ankle clonus. No tremor. Some ataxia of station is present.

*Sensation.*—No hyperesthesia. On the left lower thorax there is some hyperalgesia, and some on the right anterior surface of the trunk. The right arm is hypalgesic, so also are the extensor and flexor surfaces of the forearm, the dorsal surface of the hand and wrist and the first phalanges. From the interscapular space downward to the gluteal region there is analgesia of both sides. Deep sensibility, shows normal sense of position, normal imitation of position, normal pointing to direction of part examined.

No distinct limitation of visual field. Sense of smell impaired considerably on the left. Bone conduction slightly impaired on both sides. Palate and pharyngeal reflexes normal. Mentally the patient is dull and somewhat silly, but shows no distinct dementia. The most noticeable condition is his aphasia. This is characterized by the following points:

1. The patient can talk voluntarily and on being questioned.
2. He can repeat spoken words.
3. He can copy, but never could write well.
4. He has forgotten the names of some things seen, but can explain their use. Thus, looking at a clock he says it is something with which to tell the time. He cannot give the name of things felt, as for example, money, but he can tell what it is indirectly by paraphrase. He cannot tell the name of things smelled or tasted or heard. He recognizes the nature of all these objects.
5. He understands spoken words and tunes.
6. Can sing certain Hebrew prayers.
7. Can read printed words.
8. Can call to mind objects named.
9. Can read aloud and understand.
10. Can recall objects whose names he has seen.
11. Can repeat names of things mentioned.

Special questions show the condition better, for example:

Q. What is that (book)? A. "Holt." Q. What is that (cap)? A. What a man wears on his head. Q. What is that (paper)? Answer correct. Q. What is that (handkerchief)? Illustrates its use. Q. What is that (scissors)? A. What a man cuts with. Q. What is that (bottle)? A. What a man drinks with. Q. What is that (candle)? A. What one uses for light. Q. What is that (soap)? A. What one uses for washing. Q. What is that (knife)? Answer correct. Q. What is that (money)? Answer correct. Various other objects, such as a penny, bread, sugar, spoon, fork, apple, watch, he is unable to name, but can tell the use. Tested with salt on his tongue he cannot tell what it is; the same with sugar. When money is put in his hand he recognizes its nature, also that of knife and pencil.

In all the tests it was found that the patient was able to describe an object and its use, but was unable to find the name for it. Once in a while he showed some paraphasia. He understands all directions given to him.

To sum up, then, it would seem that the patient has a fairly good vocabulary, and can talk spontaneously, using a good many nouns, though he is sometimes at a loss for them. His speech is sometimes paraphasic, but there is no jargon aphasia. In asking him to name things, however, he is almost always at fault, and he cannot spontaneously or through the help of any sense except



Fig. 1.  
Showing areas of softening in frontal and temporal lobes.

hearing and sight (i. e., reading) name an object. He can, however, read the name aloud if written, and can repeat the name if he hears it spoken. He can sing. He understands what is said to him. He can read words, syllables and letters, also numbers. Thus because he cannot name an object if he tastes, smells or feels it, he can, however, name it if he hears the word (i. e., he can repeat). The capacity to write is present, but it is difficult to speak of it positively, because he could never write well. As far as he can write, it is voluntary, and he can also copy and write to dictation. The uses of things are recognized by him; he has no apraxia.

Up to the time of the death of patient the condition remained altogether stationary. The described disturbances of speech varied in intensity. The motor phenomena were quite bizarre at times—making one occasionally doubt the organic nature of the disease. The patient died suddenly from cardiac paralysis, the cause of which was found on post-mortem examination to have been due to syphilitic thrombosis of the papillary muscles, with acute dilatation.

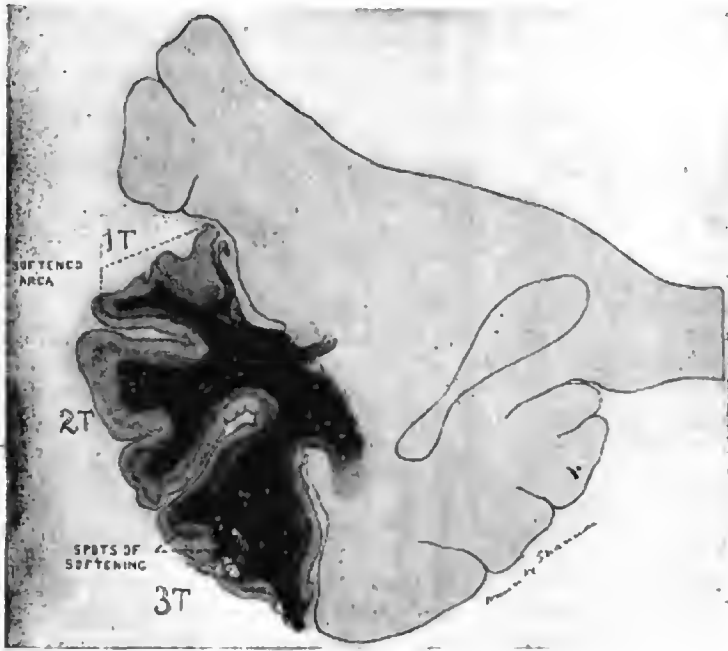


Fig. 2.

Transverse section through 1st, 2d, and 3d temporal convolutions, showing extensive superficial loss of 1st temporal, and small area in 3d temporal.

The *Record of Autopsy* by Dr. J. R. Hunt is as follows:

The anterior and middle cerebral arteries were atheromatous, and showed extensive sclerosis, but there was no thrombosis. Upon the internal surface of the left cerebral hemisphere there were to be seen three distinct patches of superficial softening. (Fig. 1.) One of these, and the largest, involved the posterior third of the first temporal convolution, extending into the lip of

the Sylvian fissure. The area of the softenings measured about two c.m. antero-posteriorly by two c.m. vertically. Measuring the total length of the first temporal convolution it was found to be 10 c.m., and the lesion lay between  $7\frac{1}{2}$  and  $9\frac{1}{2}$  c.m. from the tip; that is, in about the middle of the posterior third of the first temporal. The second superficial point of softening lies a little anteriorly to the middle third of the second frontal convolution. The third and smallest spot of softening lies on the inferior surface of the first frontal. These are all indicated in the photograph.

A section was made vertically directly through the center of the temporal lobe lesion, as shown in photograph (Fig. 2), and the extent of the softening could there be seen to be not very considerable. A block of the temporal convolution intersecting the whole of the softened part was removed and hardened in Müller's fluid and alcohol. Sections of it were prepared. These sections showed an extensive softening of the cortical layers of the convolution, this being more marked as we pass into the lip of the Sylvian fissure. The parts below the gray matter show the presence of connective tissue. In the third temporal convolution just below the level of the lesion in the first, there was found a small spot of subcortical softening about one c.m. in diameter. The microscopical examinations, so far as localization and determination of the pathological character are concerned, revealed simply the presence of a secondary softening due to obliterating endarteritis involving the cortex and some of the white matter of the areas as indicated in the figure.

The other two areas of softening were smaller and slighter in degree. The brain and medulla were normal and showed no trace of any central lesion, old or new.

*Anomic Aphasia and the question of a Naming Center.*

There has been a great deal of discussion, but I doubt if there is any very essential difference of opinion, regarding the nature of that type of sensory aphasia associated mainly with the inability to name things. The term, "naming center," it is agreed is rather an unfortunate one. The larger number of names, or nouns, used in speech, are not names of things that we see or feel with our hands. Thus, in reading a page of a medical article by a well-known neurologist, I find ten names of concrete objects and persons, and thirty-three names of things which have some quality, that is non-objective, such as area, paralysis, symptom, and so on. Now, the names of things we see are learned by repeatedly connecting the sight with a special sound, and we

finally never see an orange, for example, without being able to arouse a center in the auditory area which is connected with the center for articulatory speech. By feeling, smell, taste, we are also able to stimulate speech by this route. That is to say, the sensory stimulation goes, as a rule, first to the auditory center where the word was learned, and then through it to the articulatory center itself. Thus, in naming concrete things, we bring into play association tracts which reach from the optic or tactile area, olfactory or gustatory center to the auditory, and then to the speech centers.

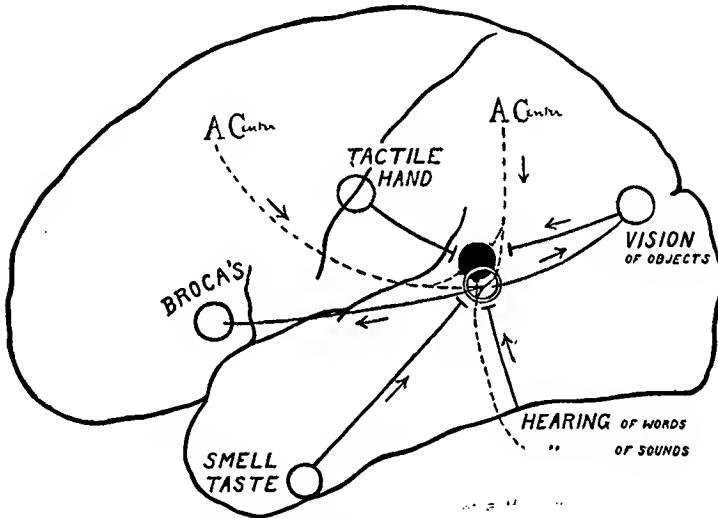


Fig. 3.  
Diagram to illustrate case of sensory anomia.

But perhaps two-thirds of the names we know, viz., the names of abstract things, are stored away among more or less complicated association tracts; when we wish to speak them, as when we say the word "symptom" or "justice," impulses from widely distributed association tracts, converge on the auditory center in persons not deaf or dumb; then are sent to the articulatory center. In ordinary normal persons the name of a concrete object no doubt brings into play mainly the optic and auditory areas first, but deaf and dumb persons would bring into play tracts which

pass from the visual areas to, perhaps the tactile areas, and thence to the gesture centers—a very different language mechanism than that of the normal man, for to the deaf-mute the name of an object is associated with muscular and tactile activities. The sight, for example, or odor, of an orange, in a deaf and dumb man, would arouse images first in the visual and gustatory centers and then pass to the Rolandic area where the gesture movements originate, and thence perhaps again to the center for the expression of the deaf and dumb alphabet. Thus, even for concrete names, there would not be any naming center common to all kinds of persons.

Even in learning the names of the abstract words of simple type, such as governor, king, business, money,—the localization and mechanism would be different from that involved in using terms more complicated, such as justice, right, anger, etc.; and it seems unlikely that there is a specially localized mechanism for such a process, except that the association activities from various areas go first to the auditory center and then to the articulatory.

The naming of concrete things, however, is a simpler and more specific process than that of the naming of abstract terms and probably involves a simpler and more localized machinery; hence it is that in ordinary aphasic lesions there is a loss of ability to name the concrete nouns more often than that of other nouns, since the machinery for the latter process is more diffused through the association centers. Thus, in an injury to the speech zone, we would expect that the capacity to name concrete things would be lost first, while we still remember the names of abstract qualities.

In the so-called sensory aphasia, with loss of ability to name concrete things, then we have not so much direct injury of a naming center as one of an associative mechanism connecting those parts of the visual and auditory areas, which are usually employed in normal individuals in naming these things.

Perhaps the term anomia is as good as any, however, to indicate all forms of sensory aphasia with inability to name concrete objects. When this inability is confined to objects seen, it is an optic anomia (or aphasia); and when in addition the patient can not name things felt or heard or smelled or tasted, it is a total sensory anomia. When it is confined to vision and touch, it is an



optic and tactile anomia, as is Vorster's case. These are clinical terms and are the safest to use. Our own case comes under the head of a total sensory anomia, because excitation from hearing noise, seeing, touching, tasting or smelling could not arouse in him impulses that enabled him to name the exciting object though he knew its nature. On the other hand excitations from the association areas in the processes of thought or of feeling did call up the name; and so did excitation of the auditory area when the name was pronounced. (He could repeat the word "bell," but he could not say the word "bell" when the bell was rung.)

We may infer from this that he had the idea of the word "bell" still stored away in his cortex; and speaking anatomically, we would call this a "subcortical aphasia."

All of these sensory anomias are probably usually subcortical, or intracortical, as these terms are commonly used; yet in our case the cortex as well as some of the subcortical tissue was destroyed. One of the writers (Dana) thinks that practically these terms are apt to be misleading and are difficult to apply in clinical work. It seems possible that often parts of certain cortical centers are destroyed and parts left intact. This view is illustrated in the diagram. That part of the auditory center concerned in receiving and transmitting impressions from visual, tactile and other special sense areas to the articulatory center is destroyed and the impulses to it blocked as indicated. On the other hand, conceptions of words from association centers (A.C.) and the sound of a word from the auditory apparatus arouses more easily the memory of the name and it is spoken. Voluntary naming in ordinary speech and the repeating of spoken words are simpler and easier mental processes, than the deliberate naming of an object seen or felt. The pathways and centers are perhaps more numerous and diffuse and not so easily blocked or destroyed. When one part is destroyed they can shift to another as indicated in the diagram.

Hence these anomias may be subcortical in their anatomical basis or "partially cortical."

There are various types of completeness of sensory anomia such as optic, optic and tactile, total, but the simple term sensory anomia will answer for the ordinary purposes of clinical description; nat-

urally some motor anomia goes with all severe forms of motor aphasia, and is only a part of it.

*Conclusions.—*

1. There is no special center for naming. There is an important station in the sensory mechanism for naming concrete things, and it lies in the first and second temporal convolutions, and this is a center for sensory anomia.

3. Sensory anomia is a form of aphasia characterized by inability to name things seen, and often things heard, felt, tasted, or smelled. Its especial character can be indicated in particular cases, by the terms optic, optic and tactile, etc.

4. The use of anatomical terms such as cortical and subcortical to indicate psychological processes is not a very practicable or rational procedure; and it would be better to drop the words cortical and subcortical in describing aphasias. In the present case there was what would be called a subcortical aphasia with a lesion mainly cortical.