one-sided spasm, or spasm beginning in one side, implies local change in the central nervous system as surely as one-sided palsy does.

Let us suppose that a square inch of convolution is diseased. If this part were destroyed, there need be no symptoms; but if it be not destroyed, but unstable, there must be symptoms—for it will discharge on muscles when its tension reaches to unstable equilibrium.

And even in those cases where we do find a lump in the brain, we do not discover the very changes on which the discharge depends. The lump does not discharge, but some ('softened') part of the brain near it—which part cannot be destroyed or it would not discharge at all, but which part must be diseased or it would not discharge so much, nor in so disorderly a manner, nor on slight provocation.

It is held by some that the coarse disease, although it lies in the cerebral hemispheres, is quite as much an eccentric cause of a fit, as is a worm in the duodenum; and that in both the medulla oblongata and pons are the centres which discharge. (1. Do not deny that grey matter in these parts is secondarily diseased.) When we consider that the hemiplegia left by the fits I describe is like that following destroying lesions in the Syphilis, it is not like that following destroying lesions in the pons or medulla oblongata, it becomes, I submit, infinitely more probable that the primary discharge is of grey matter in the region (cerebrum) in which the coarse disease is discovered.

2.—The functional nature of the change in nerve tissue.

If I am told that hemisepasm is "only a symptom," and may depend on "many causes," I admit it in the sense that various pathological processes may lead to that instability of nerve tissue which permits an occasional excessive discharge on muscles; but from the point of view of function there is but one cause of convulsion, viz., instability of nerve tissue. Of course there will be variety of range of convulsion, degrees of instability, degrees of quantity of nerve tissue unstable, and, more important than all, degrees of evolution of the nervous processes (near to and farther from the motor tract) which the pathological changes render possible.

3.—The pathological processes.

(a) Emolium.—It is not very uncommon to find when a patient has recovered of a recovering from hemisepasm, the result of emolium of the middle cerebral artery, or of some branch of this vessel, that he is attacked by convulsion beginning in some part of the paralysed region, almost always, I believe, the face or the hand. I have not, however, yet made a post-mortem examination of a patient whom I know to have had fits of this kind after supposed emolium. It will be safer, then, to say that such seizures occur in patients who have recovered partially, or seemingly entirely, from hemiplegia occurring with heart disease, or with the parietal state.

(b) Course Disease.—It is admitted that it may be of any kind, but it so happens that in nearly all the chronic cases on which I have had autopsies, the examination has revealed syphilitic disease of the hemisphere. The foreign body has been a syphilitic nodule.

4.—Circumstances which determine the paroxysm.

Many things may discharge nerve tissue. But I speak here only of chronic cases in which there is a persistent local lesion and an occasional discharge.

I think that there are two factors in the production of a paroxysm—1st. Permanent local instability; 2ndly. Something which determines the discharge of the part unstable.

The paroxysm "stores up" force, and when it reaches a certain degree of instability discharge, it is easily provoked. It may be that when continuous nutrition it has risen to a certain degree of instability— it explodes, either "spontaneously," or in some normal periodic change in the body, or in some abnormal disturbance, the result for instance of fright. It falls then to a state of stable equilibrium and order more by continuous nutrition rises to its former undue instability, when another explosion can occur. It is in short an exaltation of ordinary nutrition and function. I suppose that the provoking agents may be various—that many things will upset the equilibrium of the highly unstable nervous tissue.

All these general causes, I presume, act by altering the circulation in the head, during which alteration the equilibrium of the unstable patch is upset.

The usually accepted theory of the production of the paroxysm is that it is determined by contraction of arteries. Brown-Séquard.

I have advanced the speculation that, the liability to the convulsions which I have described in this paper—those at least beginning in the hand—is due to persistent changes in the region of the middle cerebral artery, and that the paroxysm itself is owing to a local vascular connection.

XXIX

Wernicke's Sensory Aphasia

IN THE Edwin Smith Surgical Papyrus, dating from the 17th century BC, an association was noted between temporal skull trauma and loss of speech. Nevertheless, before the work of Broca, the brain was generally thought to act as a whole, with no anatomical localization for different cerebral functions.

In 1861, Paul Broca presented two cases of dysphasia associated with lesions in the posterior portion of the second and third left frontal convolutions.

Although Broca underestimated the extent of the lesion in his first patient, his provocative presentations provided an early anatomical localization for the expressive aspects of speech.

In 1874, the German neuropathologist, Carl Wernicke (1848-1909), published a short monograph in which in he used simple anatomical diagrams to present a more comprehensive view of speech mechanisms.

Wernicke described five clinical syndromes that would be expected from lesions of (1) the afferent auditory pathways, (2) the speech reception center, (3) the association tracts between speech reception and speech expression centers, (4) the speech expression center, and (5) the efferent speech pathways. However, he provided little pathological documentation of these therapeutical possibilities. Of the ten cases he presented, only four had auditory examinations, and the lesions involved relatively large areas. Furthermore, the illustrations in Wernicke's monograph depicted the right side of the brain; he did not stress the importance of the left cerebral hemisphere, though all four of his patients examined postmortem had left-sided lesions.

Despite these inadequacies, some of Wernicke's deductions have proven correct. Subsequent studies have demonstrated that lesions in the posterior half of the left superior temporal gyrus and the adjacent part of the middle temporal gyrus (Wernicke's area) may give rise to a receptive dysphasia characterized by defective comprehension of spoken words, verbal but incoherent speech (jargon dysphasia), dysphasia, dyslexia.

The syndrome is now known as Wernicke's sensory aphasia, in honor of the man who first brought it to the attention of the medical world. Though the subject of aphasia is more complex than Wernicke envisioned, he shared Broca's distinction of providing the first anatomical framework for our present knowledge of aphasia.

References

THE APHASIS SYMPTOM-COMPLEX

A Psychological Study on an Anatomical Basis

by

Dr. C. Wernicke

... That destruction of Broca's area causes aphasia appears to be established beyond doubt through such cases as the striking one of Simon, which actually resembled an experiment. However, other conscientious and experienced observers are also correct in insisting that Broca's area is not the only speech center, and that circumscribed lesions in the region of the Sylvian fissure can produce aphasia.

We may now ask what lies near the Sylvian fissure, and we note a gyrus on the convex surface of the cerebrum, running in a curve directed posteriorly and superiorly, almost enclosing the Sylvian fissure. From the central sulcus it runs anteriorly in a distinct longitudinal tract, the first frontal gyrus. Its posterior pedicle is in the first temporal gyrus, just clearly discernible as the longitudinal tract. That the whole is to be considered as one gyrus is clear from comparison with the brains of animals, such as dogs. Comparative anatomy indicates that the gyrus describes a curve around the Sylvian fissure with the convexity toward the occiput and with two peduncles running more or less parallel to the Sylvian fissure in the frontal and temporal portions of the brain.

The whole area of the convolutions encircling the Sylvian fissure, in association with the cortex of the insula, serves as a speech center. The first frontal gyrus, being motor, is in the center for representation of movement, and the first temporal gyrus, being sensory, is the center for word-images. The fibers connecting them, which are the insular cortex, form the connecting psychic reflex arc. The first temporal gyrus consequently should be considered as the central end of the auditory nerve, and the first frontal gyrus (including Broca's area) as the central end of the nerves to the speech muscles.

[Figure 3]

Let F be the frontal, O the occipital, and T the temporal end of a schematically drawn brain. C is the central area around the Sylvian fissure (S) extends the first primitive connection. Within this convolutions, a1 is the central end of the acoustic nerve, a1 is the site of entry into the medulla oblongata; b designates the representation of movements governing sound production, and is connected with the preceding through the association fibers a2, a3 running in the cortex of the insula. From f the effector pathways of the sound-producing motor nerves run to the oblongata and exit there for the most part (the auditory and the phonic nerves extend still further caudally.

Aphasia can result from any interruption of the path a1 - a2 - b - f - a3. The clinical picture will depend upon the segment of the path involved.

II. A lesion may affect the center for word-images itself, a1. This locus is not identical with the central projection area of the acoustic nerve since aphasia with complete loss of word-images has been reported to occur in the presence of retained hearing on both sides. Destruction of a2, the center of the first temporopolar gyrus, leads to loss of memory for the sound-images of the names of objects, while the concept of an object, while the feeling and tactile images are essential to it. Impairments of the visual and tactile images (Finkelsburg's symptom) are thus not to be ascribed to a disturbance of speech but to disconnection of the concept, consequently of the intelligence.

Clearly, the preservation of the circuit a3 - b - f is worthless once the word-images are lost. The production of words is no longer initiated by word-images. Moreover, the path is cut which connects the sound that is heard with the remaining sensory image of an object (association fibers running from the first temporal gyrus to the other sensory areas of the occipital and temporal lobes). The patient, therefore, cannot repeat the spoken word—for that is indeed the particular function of the path a3 - b - f—nor can he understand the verbal expression by its word-images. Speech becomes a confused noise devoid of sense—or at best a strange language, the individual sounds of which he perceives and gradually learns to understand again.

There still remains a path by which speech movements can be initiated. The patient with the condition outlined has no disturbance of intelligence; he proves through his behavior and through his intelligent comprehension of signs and gestures that the sensory images of the objects surrounding him, as well as their concepts, remain intact.

The intensity of the symptoms varies with the severity and extent of the disease process affecting the first temporal gyri. In the severest form, involving loss of word-images of discrete objects and actions as well as of the conjunctions necessary to form sentences, etc., the diagnosis will depend on only two features; namely, the absence of spoken words and the defect in understanding what is heard. That such obvious abnormalities have not been observed till now, or at least have not been published, is attributable to the rarity of the cases and to the fact that experienced and intelligent physicians have considered this condition to be dementia.

There are moderate degrees of the ill, in which the important structural elements of sentences are retained and the general meaning of a question can be properly conceived. The presence...
of this defect must be established through suggestive questions. If the patient, for example, when asked: Is that a glass?, does not decide immediately but vacillates, considers, and finally utters a doubtful "yes" or "no", then he belongs in this category.

The following points deserve to be emphasized:

1. Partial lesions of the sensory speech center will limit the vocabulary to a definite vocabulary, both in speaking and in hearing. This vocabulary can be ascertainment through suggestive questions, but it requires a difficult and lengthy examination.

2. A large vocabulary is a prime feature of sensory aphasia. Conditions in which only a few simple words are retained always belong to the motor form of aphasia.

3. There is no trace of hemiplegia.

4. There is agraphia. Writing is a conscious movement, learned with the most intimate dependence on sound and always executed under the guidance of it. In cases of partial sensory aphasia, a partial agraphia may also be expected.

5. The situation is entirely different with respect to understanding written or printed characters. The undeciphered, slurred, irregular writing characterizes what is written only when he hears it spoken. The educated man, accustomed to reading from early childhood, skims over a page and understands its meaning without being conscious of the wording. The former will show symptoms of alexia in addition to aphasia, but the latter, in marked contrast to his inability to understand what is spoken, will grasp everything written. On the other hand, he will be just as aphasic in reading aloud as in spontaneous speech.

Agraphia as well as alexia are caused by disease in an entirely different region; namely, in the visual cortex, for the visual memory of the characters is indispensable for writing as for reading.

Case 2

Susanne Rother, 75 years old, wife of a porter, was admitted to All Saints Hospital on 7 October 1873. She showed signs of marked sensibility and advanced atherosclerosis of all accessible vessels, and she had an expression of suffering. She could walk only with assistance because of generalized weakness and a feeling of dizziness. She usually lay moaning in bed, wrapped up deep in the covers; she was incessant of feces and urine.

Her psychic state was considered at that time to be confusion, complicated by aphasia. She answered questions absurdly and failed to follow instructions, or else turned them around, thus giving the impression of apraxia.

In view of her failure to understand, the nurses believed she was deaf. She paid little attention to her surroundings and showed little need to communicate. Her spontaneous vocabulary seemed limited, but was still large enough that a motor aphasia could not be considered. The aphasia was recognizable in the confusions and distortions of the words she used. Thus, she very often said correctly: "I thank you very heartily" or "I thank you very much" etc. "I am very sick. Oh, I am so cold. You are a very good person," were often used expressions. The physician, whom she had just called a good gentleman, she then would call "my little daughter" or "my little son," both in the same sense.

Sensation appeared intact. The grasp was weak but equal on both sides. More exact examination of sensation and motor function was not made.

There was no improvement in the psychic or somatic symptoms.

Death came on 1 December 1874 after a protruded intestinal carcinoma, associated with vomiting and deep prostration during the last two days.

The history yielded that . . . the confused speech had appeared suddenly on 2 November 1873 .

Autopsy revealed edema of the pia and slight internal hydrocephalus. The convolutions of both hemispheres and both insular regions were shrunken and atrophic throughout. Furthermore, all cerebral arteries showed atheromatous degeneration to an extreme degree. "The branch of the artery of the left Sylvian fossa, running down into the inferior sulcus of Burdach, was occluded by a thrombus tightly adherent to the wall. The entire frontal temporal gyrus, including its junction with the second temporal gyrus and the origin of the latter from Bielof's inferior parietal lobule, were converted into a yellowish-white necrotic, to which opacified pia was tightly adherent. The connections of the temporal lobe into the insulums were for the most part destroyed in the softened places. The insula itself and the brain stem ganglia showed no changes. The focus of softening was not bordered by inflammatory hardening but passed over directly into normal consistency. . . .

XXX Wallenberg's Syndrome

The syndrome caused by infarction of the posterolateral portion of the medulla oblongata (the lateral medullary plate) is one of the most characteristic of the neurological syndromes following arterial occlusion. It was encountered occasionally by physicians over the years, but its existence was firmly established in 1895 by Adolf Wallenberg. He published a detailed clinical report of a single case. Wallenberg was a physician and neuroanatomist of Danzig who fled Nazi persecution and spent his last years in the United States. His expert knowledge of neuroanatomy, based in part on original research, permitted him to make sense of his patient's many symptoms and signs. He deduced the location of the infarct and postulated that it was caused by occlusion of the ipsilateral posterior inferior cerebellar artery. Furthermore, he reviewed several reports of similar cases and described the clinical picture common to all. His patient died in 1899, and Wallenberg verified the suspected arterial occlusion and medullary infarction at post-mortem examination.

Although Wallenberg thought the syndrome was due to embolization of the posterior inferior cerebellar artery, the most common cause is now known to be thrombosis of the vertebral artery. Furthermore, a preexisting ocular lesion in his patient prevented Wallenberg from noting the ipsilateral miosis that usually accompanies the syndrome. Nevertheless, our present delight in expounding the anatomy of the brain stem at the bedside of a patient with the lateral medullary syndrome is a tribute to the pioneering efforts of Adolf Wallenberg.

References