of this defect must be established through suggestive questions. If the patient, for example, 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 limit the patient to a definite vocabulary, both in speaking and in hearing. This vocabulary can be ascertained through suggestive questions, but it requires a difficult and lengthy examination.
2. A large vocabulary is a poor 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 aphasia. 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 aphasia may also be expected.
5. The situation is entirely different with respect to understanding written or printed characters. The uneducated, slightly literate man understands 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.

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

Case 2

Sussanne Rother, 75 years old, wife of a porter, was admitted to All-Saints' Hospital on 7 October 1873. She showed signs of marked senility and advanced arteriosclerosis of all available 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 incontinent 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 heartily" or "I thank you very much" etc. "I am very sick. Oh, I am so cold. You are a very good gentleman," 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 remained 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 protracted intestinal catarrh, 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.

XXX Wallenberg's Syndrome

The syndrome caused by infarction of the postrolateral 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 (1862 to 1949), who 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 in 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 expanding 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

Acute Bulbar Disturbance (Embolus of the Left Posterior Inferior Cerabellar Artery)

by Adolf Wallenberg

Danzig

This case report of an acute lesion in the medulla oblongata requires some justification since my observations thus far have been only clinical. However, the characteristic clinical picture, which can be easily recognized by a group of striking symptoms, will provide a certain interest in the following case history... [The detailed case history is presented, and then the following recapitulation:]

A 30-year-old man, with poor vision caused by a preexisting ocular condition (cataract on the left side, corneal scarring and anterior synechias on the right side...). Suffered an attack of vertigo without loss of consciousness. At the same time he developed pain and hyperesthesia on the left side of the face and body, hyperesthesia of the right half of the face, and loss of pain and temperature sensitivity in the right extremities and the right half of the torso, with retention of the sense of touch. There was paralysis of swallowing; impaired sensation on the mucosa of the mouth, throat and palate; discoloration of the soft palate (on the first day bilateral, later left-sided); total paralysis of the left recurrent laryngeal nerve, and paralysis of the left hypoglossal muscle. With this disturbance there is an innervation of the facial muscles. He also had ataxia of the left extremities without impairment of gross strength, and he fell to the left side... The pulse became slower (from 96 to 76 per minute). During the ensuing days the sensitivity of the right half of the face returned to normal. The hyperesthesia of the left half of the body disappeared, and that of the left trigeminal region changed to anesthesia primarily of pain and temperature (less for proprioceptive and cutaneous sensations), with suppression of the corneal and conjunctival reflexes... The pulse quickened again, but the other disturbances remained. On the eighth day an herpetic eruption appeared on some of the analgesic areas: the left face (including the nasal mucosa; the sensitivity of the mouth and throat had returned), right shoulder, and right inguinal region...

Two to three months after the attack, the patient's status was as follows:

a) Subjective symptoms
1. Vertigo and a sense of falling to the left.
2. Numbness on the left half of the face and the right half of the body.
3. Difficulty in swallowing (very slight).
4. Pain in the nape of the neck and occasionally in the left eye.
b) Objective signs
1. Unsteadiness of gait, with veering toward the left.
2. Ataxia of the left extremities.
3. Paralysis of the left half of the soft palate.
4. Paralysis of the left vocal cord; followed by paralysis, suggesting atrophy.
5. Greater volume of the left half of the tongue while resting in the mouth.
6. Disturbance of sensation in the first and, to a lesser degree, in the second divisions of the left trigeminal nerve, especially affecting the eyes, eyelids, bridge of the nose and nasal mucosa. The impairment mainly affects pain and temperature (directly; localization, electrocutaneous and pressure sensations are also involved to some extent.
7. Absence of the left corneal and conjunctival reflexes.
8. Diminution of pain and temperature sensitivity on the right side of the body...
9. Slight alteration of the other sensations (i.e., localization, faradocutaneous and pressure sensations)...

In the following weeks, the difficulty swallowing, the falling to the left, and the ataxia gradually disappeared. The other phenomena... remained unchanged...

The localization of the lesion in this case is not difficult, in my opinion... The diagnosis can be made on a secure foundation in view of certain pathological and experimental findings. I may add that this patient's ocular disorder presented an examination of the pupillary reflexes...

The paralysis of the vocal cord and the paresis of the tongue and palate on the left side strongly suggest the diagnosis of the left half of the medulla oblongata...

Only a few root fibers (or nuclear cells) of the hypoglossus may be involved, since the mobility of the tongue was completely unimpaired and the hypoglossus suffered only a loss of tone. The bulbar portion of the left accessory nucleus must be greatly affected in view of the paralysis of the vocal cord and the paresis of the palate. The extent to which the vagus and the glossopharyngeal are responsible for the weakness of the palate and the vocal cord is difficult to state, since the anatomical and physiological investigation of the function of the hypoglossal and pharyngeal functions... The region of the lateral medulla is incompletely invaded by the lesion, perhaps from the truncation of the degeneration of the pyramids to the emergence of the upper vagal or lower glossopharyngeal roots, and that the extent of the associated changes diminishes from below to above.

I have intentionally not mentioned the left trigeminal, because we can use its disturbance to establish not only the level of the lesion but also its location in the cross-section...

Beginning from the ventral surface of the medulla: the fibers of the pyramidal must be essentially intact, for all movements were normal in strength and directivity, but localized, electrocutaneous and pressure sensations are also involved to some extent. The predominate involvement of these sensory modalities on the right half of the body may help to localize the lesion. This assumption agrees fully with the extent of the lesion that we inferred earlier. I leave undecided whether the more marked involvement of pain and temperature... point to a particular area of the medulla in order not to lose sensitivity in our hypotheses...

The predominant involvement of these sensory modalities on the right half of the body may help to localize the lesion. This assumption agrees fully with the extent of the lesion that we inferred earlier. I leave undecided whether the more marked involvement of pain and temperature... point to a particular area of the medulla in order not to lose sensitivity in our hypotheses...

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TUBEROUS SCLEROSIS

A QUIRK of fate led Désiré Magloire Bourneville (1840–1909)1 to discover the disease that now bears his name. In 1879, while substituting for his teacher, L. J. F. Delasauve, Bourneville attended a child with psychomotor retardation, seizures, and an erosion over the nose and cheeks. After the patient’s death from pneumonia, Bourneville found peculiar firm enlargements in the cerebral convolutions, which led him to call the disease tuberous sclerosis.

Bourneville had procrastinated in reporting this case, his disease would not have been “new.” Simultaneously and independently, Hartdeng2 described the same condition in a newborn in Germany.

In addition to the cerebral lesions, Bourneville found several small renal tumors in his patient. However, only he and other physicians reported additional cases did it become apparent that tuberous sclerosis is a dysgenetic syndrome, frequently familial, involving many organ systems throughout the body.3 The development of radiology has facilitated the recognition of this disease, since scattered areas of calcification are often seen within the substance of the brain. Although Bourneville’s patient had normal ventricles, pneumoencephalography may demonstrate small nodules lining the ventricular walls, giving the characteristic appearance called “candle gutterings.”

Bourneville later studied cretinism and mongolism and became an authority on the subject of mental retardation in children. But he is best remembered for his initial chance encounter with tuberous sclerosis.

References

CONTRIBUTION TO THE STUDY OF IDIOCY* by BOURNEVILLE, Physician at the Bicêtre

.. . . . Case III

TUBEROUS SCLEROSIS OF CEREBRAL CONVOLUTIONS: Idiocy and Hemiplegic Epilepsy

Information given by her mother (March 31, 1879).—Father, 45, in good health. . . . No neurologic illness in his family. Mother, 40, never... Neither of her parents had neurologic diseases. No consanguinity.

Five children: 1. the patient; 2. and 3. two children who died while with a wet nurse (it is

3More accurately, the lateral columns—Translator.
4The lateral hypophysial tract—Translator.