CONCEPTS OF APHASIA: HISTORICAL AND CONTEMPORARY

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CONCEPTS OF APHASIA: HISTORICAL AND CONTEMPORARY

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PREFACE

Early in his studies of aphasia the student discovers controversy among the published authorities, as well as disagreement over terminology. The possibility of approaching the problem from anatomical, physiological and psychological viewpoints partly accounts for the existing confusion. The loose transference of terminology--as well as of conceptions themselves--from one frame of reference to another, does much to discourage the conscientious student.

There is no one existing source in which a resume of the conflicting theories, much less their consequent therapies, has been compiled and discussed. The student of this speech defect has to ferret out the divergent schools of thought in the few available books, in inaccesible medical journals, and psychological and speech publications.

It is the purpose of this particular study to gather and adequately describe the leading theories or concepts of aphasia.

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PART I

INTRODUCTION

CHAPTER I

APHASIA: DEFINITION AND ETIOLOGY OF

The earliest description of aphasia, as far as neurologist Henry R. Viets of Boston can ascertain, can be found in the medical works of C. Linnaeus[1] dated 1745. He set forth the classical symptom of knowing what one wishes to say, but being unable to say it. Linnaeus called this condition "forgetfulness."[2]

Immediately prior to Broca[3] there was only one type of aphasia recognized--the "alalia" of Lordat.[4] In 1861 Broca proposed the term "aphemia" to designate what we now understand by Broca's aphasia, or disorders of articulated language. The term "aphasia" was first applied in 1861 by Trousseau[5] to loss of speech due to a cerebral lesion exclusive of that due to paralysis of the muscles concerned with

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1. (Carolus Linnaeus, or Karl von Linné:--Swedish physician and naturalist, 1707-1778)

2. Henry R. Viets, "Aphasia as Described by Linnaeus and as Painted by Ribera," Bulletin of the History of Medicine, 13:3:328, March, 1943.

3. (Paul Broca:--Parisian anthropologist and surgeon, 1824-1880)

4. (Lordat:--A Frenchman who published a book on speech in 1823 and who later (1843) described his own condition of aphasia beginning in 1828)

5. (Armand Trousseau:--One of Broca's associates and fellow students of aphasia, 1801-1867)

speech and exclusive of dementia. This replaced the "aphemia" of Broca and was accepted as the preferable term. Later, suggestions were made to render the terminology more definite and suitable. McLane Hamilton (cited by Jackson, 1878) suggested the term "asemasia" (meaning the inability to communicate by signs or language), and Head[6] recommended "dysphasia" (disturbance of language). However, as Jackson[7] observed in 1878, it was already too late to displace the term "aphasia".

Kussmaul[8] noted, in a 300-page monograph on aphasia printed in 1885, that the word "aphasia" was then used to signify the entire symptom complex of disturbances of all types of man's communication--not merely speech disturbances alone. Déjerine[9], in 1914, defined "aphasia" as the loss of the memory of the signs by means of which civilized man exchanges his ideas with his peers. Here can be observed the flexible use of the word to include what we now term "agnosias" (loss of the function of recognition resulting from

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6. (Henry Head:--English Physician, 1861-1940. See Chapter V)

7. (Hughlings Jackson:--The founder of English neurology. See Chapter III)

8. (Adolf Kussmaul:--German physician, 1822-1902)

9. (Joseph Jules Déjerine:--Parisian Neurologist, 1849-1917)

an organic cerebral lesion and through one sense organ only[10]), and "apraxias" (loss of ability to perform as desired or as requested, through loss of memory of how to perform[11]) in addition to the "aphasias". However, Bastian[12] attempted to indicate motor speechlessness or pure word mutism in 1898. In this sense its application was greatly advanced by Henschen[13] in his monograph of 1922.

A good working definition of "aphasia" for contemporary use might be that of Michael Osnato[14] who defined it as

> loss of or encroachment upon the ability to change concepts into words, in spite of the integrity of the speech musculature, and a loss of the ability to understand things spoken despite the integrity of the sense of hearing.[15]

Another adequate explanation of the aphasic's condition is Russell Meyers':

> The term aphasia should be reserved for disturbances of speech which are manifested only as a disability of

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10. J. M. Nielsen, Agnosia, Apraxia, Aphasia. (New York: Paul B. Hoeber, Inc., 1948), p. 251.

11. Ibid., p. 268.

12. (H. Charlton Bastian:--British M.D. and Censor of the Royal College of Physicians of London at the end of the nineteenth century)

13. (Salamon Eberhard Henschen:--Swedish neurologist, See Chapter VI)

14. (Michael Osnato:--American neurologist)

15. Michael Osnato, "Aphasia and Associated Speech Problems," Neurological Bulletin, 1:414, Nov.-Dec., 1918.

> formulating and communicating the arbitrary social symbols of perceptual patterns. Aphasia implies that the speech dysfunction now in evidence does not rest upon a paralysis of the receptor, effector, or co-ordinating mechanisms involved in the language function. By simple clinical devices it should be readily possible to demonstrate that the patient can both see and hear and that he is able to produce non-language sounds co-ordinately and execute refined movements with his hands and fingers.[16]

Robert West defines "aphasias" as "failures of the faculty of forming, retaining, and reproducing mental concepts in association with purely arbitrary combinations of speech sounds."[17]

J. M. Nielsen[18] writes of aphasia as

> the term in common use for all disturbances of language, motor or sensory, due to a lesion of the brain but not due to faulty innervation of the musculature necessary for speech or to involvement of the sense organs themselves or to general mental defect.[19]

More briefly Nielsen suggests that "aphasia consists in organic disease of the cerebral memory engrams for

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16. Russell Meyers, "Aphasia: A Problem in Differential Diagnosis and Re-education," The Quarterly Journal of Speech, 23:364, October, 1937.

17. Robert West, "Aphasias and Related Alexias, Agraphias and Amentias," Brennemann's Practice of Pediatrics, p. 15.

18. (Johannes Maagaard Nielsen:--Los Angeles neurologist and psychiatrist, 1890-____)

19. Jeanette O. Anderson, "Aphasia From the Viewpoint of a Speech Pathologist," (unpublished Ph.D. Thesis, The University of Wisconsin, Madison, 1942), p. 180.

language."[20] Nielsen warns his readers to distinguish carefully between the object and its symbol, for "loss of recognition of the object or its symbol is agnosia for the object or the symbol. Loss of significance of the symbol is aphasia."[21]

We must realize that aphasia is simply a convenient name for a rather large group of disorders, the various types and classifications of which are described in Chapter XII.

The two main etiologic categories of aphasia are disease of and trauma to the brain. Disease can be thought of in general terms as infection, lack of nourishment (oxygen), and tumors. Trauma, a bodily injury produced by violence, or any thermal, chemical, etc., extrinsic agent, embraces cutting and tearing, as well as "strokes" and the formation of blood clots.

In cases of damage to the sensory nerve fibres, awareness of sensation is lost; damage to motor fibres results in loss of the power to move muscles; and damage to the association nerve fibres causes the loss, to some degree, of the power to correlate certain sensory and motor functions. When the association fibres having to do with the language function are damaged, the patient exhibits aphasia.[22]

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20. Nielsen, op. cit., p. 254.

21. Ibid., p. 255.

22. Ollie Backus, et. al., Aphasia in Adults (Ann Arbor, Michigan: University of Michigan Press, 1947)

Russell Meyers affords us a comprehensive listing of some of the most common etiologic factors underlying aphasia:

1. Brain trauma, at birth or subsequently during the life of the patient
2. Pathological process of the cerebral blood vessels
   a. Embolism, the sudden plugging of a vessel by a foreign body borne by the blood stream.
   b. Thrombosis, the slow occlusion of a vessel by a process of thickening of its wall or lining
   c. Hemorrhage, the rupture of a vessel leading to the extra-vasation of blood into surrounding tissues
   d. Mechanical occlusion by pressure from without the vessel
   e. Vasospasm, the transient narrowing of the vessel lumen under the influence of hormones and vaso-constrictor nerves
3. Brain tumors
   a. Primary neoplasms of the brain tissues
   b. Secondary neoplasma, representing metastaces or extensions
4. Infections of the brain
   a. Meningitides (epidemic and non-specific forms)
   b. Encephalitides (epidemic and non-specific forms)
   c. Abscesses
   d. Gromelomata (syphilis and tuberculosis)
   e. Parasitic cysts
   f. Arachnoiditis
5. Degenerative brain diseases
   Gower's "abiotrophic" processes, lipoid histiocytosis, tuberous sclerosis, etc.
6. Allergic cerebral edema, as in some migrainous manifestations
7. Pre- and post-convulsive seizure states[23]

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23. Meyers, op. cit., p. 365.

CHAPTER II

EARLY HISTORY

The beginning of any history of the function of speech and the theories built up around cerebral localization is apt to be clouded in mystery, doubt and confusion. During the Middle Ages the common belief was that the brain contained three ventricles, each of which was the dwelling place of one or more aspects of the psyche. The seat of the "sensus communis" was the anterior chamber which received the nerves of taste, smell, sight and hearing. The faculties of cogitation and reasoning resided in the middle ventricle. The posterior ventricle was the seat of memory.

At the beginning of the nineteenth century, the activities of the mind and theories of speech remained mixed with vague theological and metaphysical conceptions. Cerebral localization had not yet been discovered. The teaching current at that time was that all parts of the brain served the same function. In the event of injury or disease of one portion, any portion might take over. Physiologic--not anatomic--uniformity was being taught. Foremost of the men who emphasized the unified functioning of the cerebral lobes was Marie Jean Flourens.[1]

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1. (Jean Pierre Marie Flourens:--Parisian physiologist, 1794-1867)

The first real attempt to localize the faculty of articulate speech was made in 1808 by Franz Joseph Gall, an Austrian physician (1758-1828). He advanced the idea that the brain was a group of organs with individual functions, not a single organ. Correspondingly, each mental condition was attributed to the activity of one brain element.

Advancing such radical views, Gall encountered strong opposition. In a personal letter from the Kaiser, he was forbidden to continue his Vienna lectures (1796-1801) on the new theory of functions of the brain because "his views were considered so subversive of religion and morals."[2]

In 1808 Gall presented his memoir entitled "Introduction au Cours de Physiologie du Cerveau" to the Institute of France. The following year all his views appeared in a publication called "Recherches sur le systeme nerveux en general et sur celui du cerveau en particulier", written with Spurzheim as co-author. The faculty of speech Gall attributed to the anterior parts of the frontal lobes of the brain, "mainly on phrenological grounds."[3]

Today Gall's name is frequently associated with phrenology, the study of bumps on the head. However, "it is to this man that we are really indebted for the beginning of the

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2. Henry Head, "Aphasia: An Historical Review", Brain, 43:391, 1920.

3. Donald Core, "Some Modern Conceptions of Aphasia", The Medical Chronicle, 27(4th Series):309, April-August, 1914.

conceptions we now hold of the relations of the various portions of the nervous system to one another."[4]

Many of Gall's ideas have been accepted into the body of general knowledge.

> He was the first to insist that the brain should be examined anatomically from the spinal cord upwards; for he laid down that the functions of the brain could only be explained by considering their relations to those of the spinal cord. All nerve fibres ended in grey matter, and the white matter of the nervous system served for conduction only. He pointed out the analogy between the grey matter which covers the hemispheres of the brain and of the cerebellum, with that which forms the corpora quadrigemina and optic thalamus. All nerves, whether they belonged to the brain or to the spinal cord, took their origin in grey matter, and the substance of the cerebrum and cerebellum could not be considered to be in direct connection with the peripheral nerves.[5]

However, ideas such as these appeared interspersed with fantastic theories of the "moral" qualities.

Gall's greatest supporter and disciple, appointed Professor of Clinical Medicine at the "Charite" in 1831, was Jean Baptiste Bouillaud.[6] In 1825 he published a memoir called "Clinical Researches to Demonstrate That Loss of Speech Corresponds With Lesions of the Anterior Lobules of the Brain, and to Confirm Gall's Opinion on the Seat of the

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4. Head, op. cit., p. 391.

5. Ibid., p. 391-2.

6. (Jean Baptiste Bouillaud:--French physician, 1796-1881)

Organ of Articulate Language."[7] Apparently this was the same paper presented before the Royal Academy of Medicine under the name "Some Clinical Researches to Establish the Theory That Aphasia is Due to a Lesion of the Brain and to Confirm the Opinion of M. Gall as to the Site of the Lesion."[8] Bouillaud, the first man to point out that aphasia is correlated with a lesion in the anterior lobes of the brain, came to the following conclusions as a result of post mortems on a great many specimens:

1. Movements of the organs of speech are controlled by a cerebral centre, special, distinct and independent.
2. This centre is situated in the frontal lobes.
3. Loss of speech is due sometimes to loss of word memory and sometimes to a loss of power over the muscular apparatus that is responsible for articulation.
4. Loss of speech does not involve loss of movements of the tongue as far as mastication and deglutition are concerned.
5. Nerves that innervate the tongue associated with the production of speech either have their origin in the anterior lobes of the brain or are closely connected to that region by means of communicating branches.[9]

It is to be noted that Bouillaud did not specify laterality.

Working quietly at this same time was a Frenchman named Marc Dax (cited by Nielsen, 1947). He noticed that persons afflicted with hemiplegia, along with a loss of ability to express themselves, were nearly always afflicted

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7. Everett M. Ellison, "Sensory Aphasia", New York Medical Journal, 113:15:797, June 1, 1921.

8. Core, op. cit., p. 309.

9. Ibid., p. 309-10.

with a right and not a left hemiplegia. Presenting his views of cases gathered for thirty-six years, Dax published an article in 1836 in support of his conviction that "the anatomic structure on which speech was based was located in the left side of the brain."[10] This is the first time that one of the hemispheres was considered to be dominant. Hitherto, the two cerebral hemispheres were considered identical, not only anatomically but physiologically.[11]

Perhaps the greatest single influence on the entire history of aphasia was that of the French surgeon and brain specialist, Pierre Paul Broca (1824-1880). In 1861 he presented his report of 2 cases to the Anthropological Society of Paris. He contended that the faculty of language was at the base of the third frontal convolution of the left hemisphere (the so-called pars opercularis, or Broca's convolution). The clinical cases supporting his statement were three brains. (Inaccuracies exist in the literature as to whether it was 2 or 3 brains). It is difficult to ascertain why his conclusions were so violently supported since the lesions were large, and "it was only by deduction that Broca determined the lesion to have started in that area."[12] Making the acceptance of

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10. Nielsen, op. cit., p. 2.

11. J. M. Nielsen, "Function of the Minor (Usually Right) Cerebral Hemisphere in Language", Bulletin of the Los Angeles Neurological Society, 3:67, 1938.

12. J. M. Nielsen, Agnosia, Apraxia, Aphasia. (New York: Paul B. Hoeber, Inc., 1947), p. 2.

Broca's views appear even more implausible are the 1914 observations of Dr. Eugene Dupuy of Paris. He wrote:

> This localisation was founded on pathological observations, and the three brains which Broca showed are at present--where they have been preserved from the beginning--in the Museum of Pathological Anatomy in Paris; they are to be seen every day. They show that they never have been opened--i.e., cut into sections--so that it is impossible to say what other lesions exist, if any, beside those seen on the surface of those brains.[13]

Broca called the loss of function in his cases aphemie, or aphemia (absence of ability to speak). The lacking faculty which he thus designated was that of word articulation.

Broca's foremost champion was Trousseau who added the weight of his authority and invented the word "aphasia" to replace Broca's "aphemia".[14] By 1864 the French Academy recognized the term "aphasia" for all disturbances of phonetic speech.[15] The reason for this change in terminology is well explained by J. M. Nielsen. He relates how

> Trousseau learned from a well-educated Greek named Chrysaphis living in Paris that while a meant without and phemia meant speech, the term aphemia did not mean speechlessness but infamy. On the other hand, aphasia (which, according to Skwortzoff, had been used in 200 B.C.) meant speechlessness. Broca accepted Trousseau's correction, and since that time aphasia has withstood all attempts to

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13. Eugene Dupuy, "Localisation of Motor and Speech Centers in Definite Areas of the Cortex of the Brain", The Lancet, Vol II for 1914:207, July 25, 1914.

14. Head, op. cit., p. 395.

15. Arthur Ernest Davies, "Speech Reactions and the Phenomena of Aphasia", Psychological Review, 33:6:425, Nov., 1926.

> change it in spite of the obvious impropriety of the term since the enormous expansion of its meaning has been accepted.[16]

Trousseau, although at first accepting Broca's doctrine, soon voiced disagreement, with the discovery of several cases in which aphasic patients were found at autopsy to have lesions in the temporal and parietal lobes of the left side.

In all fairness to Broca, the little-known fact must be admitted that he too departed from his original stand of extreme localization. Several years later he insisted that "the insula and the lenticular nucleus are almost always involved together with the third frontal convolution."[17] However, Broca's followers were so carried away in their enthusiasm with his first publication that they overlooked his later cautions and instead accepted the third frontal convolution as the exclusive seat of motor aphasia.

> This exclusivism continued to exist in its entirety up to 1906, when Marie [See Chapter IV] first dared challenge the scientific accuracy of the generally adopted doctrine, which up to his time had been considered incontrovertible and therefore classical.[18]

Among the neurologists who agreed with Broca were Gustav Fritsch (1838-1897) and the man who in 1870 established

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16. Nielsen, op. cit., p. 2.

17. Alfred Gordon, "A Contribution to the Study of Aphasia", New York Medical Journal, 97:10, Jan-June, 1913.

18. Loc. cit.

the electric excitability of the brain, Edouard Hitzig (1838-1907).

As early as two or three years after Broca's publications, Charcot[19] published observations which would prohibit absolute acceptance of Broca's principle that speech depends upon Broca's convolution in the left hemisphere only for right-handed persons, and in the right hemisphere for left-handed persons. He stressed the difference among individuals in the importance to them of auditory, visual, and motor centers. He noted that "symptoms of lesions in certain cases differed from symptoms of identical lesions in others."[20] This he attributed to the relative development of the various centers during the acquisition of language. Accepting the dominance of one center over another in speech, he postulated visual-minded, auditory-minded, and motor types of individuals. Lesions in the guiding center would therefore cause greater aphasic damage than lesions in the minor center.

The foundation for the anatomico-physiologic concept of sensory aphasia was laid in 1866, according to J. M. Nielsen, by Wyllie's observation that "word ideas of associated motions which form the faculties of speech are supra-motory, while the situation of associated sensations which form the

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19. (Jean Martin Charcot:--French physician, 1825-1893)

20. Nielsen, op. cit., p. 5.

faculty of word comprehension is supra-sensory."[21] In other words, the loss of expressive ability was motor in character and the loss of receptive ability was sensory. Eight years later this theory received scientific confirmation by the cases of Wernicke of Breslau. Carl Wernicke (1848-1905), the 26 year old who established sensory aphasia on an anatomic basis[22], designated the auditory center to be the transverse gyri of the superior temporal convolution, and Broca's region as the conceptual center for articulated speech. He also "outlined the syndrome of what soon became known as Wernicke's aphasia; this was a fourfold syndrome including auditory and visual agnosias for symbols, alexia and agraphia."[23]

Wernicke described the subcortical fibers radiating from various sensory centers, converging into one point, a sensory speech center, and giving rise to a definite group of memory images. The classic explanation of sensory aphasia is "loss of memory images from a lesion in this center of the fibers radiating to it."[24] The area we now speak of as

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21. Ibid., p. 2-3.

22. Nielsen, op. cit., p. 4.

23. Anderson, op. cit., p. 11-12.

24. Lauretta Bender, "Disturbances in Visuomotor Gestalt Function in Organic Brain Disease Associated With Sensory Aphasia", Archives of Neurology and Psychiatry, 30:3:518, Sept., 1933.

Wernicke's area is the posterior half of the first temporal and closely adjacent second temporal convolution of the left hemisphere. The type of aphasia resulting from degeneration of the third frontal convolution of the left hemisphere (Broca's area) is designated motor aphasia. Wernicke's aphasia represented loss of comprehension of spoken language and of written or printed matter, inability to write, and paraphasia.

Contemporary with these developments, the little-known efforts of an Englishman named Moxon (cited by Nielsen, 1947) resulted in the recognition that the right hemisphere had a governing influence over the left in the performance of voluntary acts. It remained until 1900 for H. Liepmann to uncover this "foundation for the most important principle of modern apraxia."[25]

In the period immediately ensuing, a great many publications appeared. This period has been aptly termed the age of the "Diagram Makers". These men tried to map out the various language centers and their interconnections.

Soon after the publication of Wernicke's article, Kussmaul recommended that his term sensory aphasia be changed to word deafness. The term "agraphia" was introduced in 1867 by William Ogle (cited by Nielsen, 1947) to characterize

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25. Nielsen, op. cit., p. 3-4.

inability to write. Exner[26] in 1881 described a writing center at the foot of the second frontal convolution on the left sid e. Dejerine, in 1881, postulated a visual speech center in the left angular gyrus. Deterioration of this center would result in word-meaning blindness.

While these research workers were discovering "centers" and studying cases, synthetic thinkers were correlating these discoveries. "The first actual diagram was published by Baginsky in 1871 . . . From this time onwards the rage for diagrams became a veritable mania."[27]

Shortly after the appearance of Wernicke's work, Lichtheim[28] began to make diagrams of Wernicke's centers. Lichtheim's, of all the various diagrams, attained the greatest popularity.

> It was definite and precise; every form of aphasia was accounted for by a lesion of some hypothetical centre or purely imaginary path. Teachers of medicine could assume an easy dogmatism at the bedside and candidates for examination rejoiced in so perfect a clue to all their difficulties. But serious students could not fit these conceptions of aphasia with the clinical manifestations; incredulous of such scholastic interpretations, they lost interest in a problem of so little practical importance.[29]

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26. (Siegmund Exner:--Viennese physiologist, 1846-1926)

27. Head, op. cit., p. 396.

28. (Ludwig Lichtheim:--German physician, 1845-1915)

29. Head, opcit., p. 397.

In his diagramming of Wernicke's theory, Lichtheim added a new link with his concept center connected to the motor and acoustic centers. Apparently he intended this center to bridge the gap between the anatomical and psychological views of aphasia. But "these schematic structures were built of flimsy, theoretical material, on scanty clinico-anatomical foundation, and it is not surprising that a radical departure from the existing views on aphasia was soon to follow."[30]

It was H. Charlton Bastian[31] that Henry Head accuses of opening "the catastrophic road to schemas and diagrams."[32] Bastian's papers, first appearing in 1869 and undergoing subsequent revisions as time passed, included a scheme involving four centers: glossokinesthetic (tongue), cheirokinesthetic (hand), visual word, and auditory word centers. He called these the organic seats of physiological types of memory, and designated Broca's region--"the posterior part or foot of the third frontal and the inferior part of the ascending frontal convolutions"[33]--as the part of the brain which he called the "glosso-kinaesthetic" centre.

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30. William J. German, "Aphasia", Yale Journal of Biology and Medicine, 1:365- 1928-29.

31. (Henry Charlton Bastian:--English neurologist, 1837-1915)

32. Head, op. cit., p. 296.

33. H. Charlton Bastian, "Some Problems in Connexion With Aphasia and Other Speech Defects", The Lancet, Vol I for 1897:935, April 3, 1897.

In further clarification of his concept of a center Bastian states

> Although I am not a believer in the complete topographical distinctness of the several sensory centres in the cerebral hemisphere, I consider it clear that there must be certain sets of structurally related cell and fibre mechanisms in the cortex, whose activity is associated with one or with another of the several kinds of sensory endowment. Such diffuse but functionally unified nervous networks may differ altogether from the common conception of a neatly defined "center", and yet for the sake of brevity it is convenient to retain this word and refer to such networks as so many "centers".[34]

Bastian contended that we think in words, and words are remembered sounds revived in one or more of the word-centres. Association fibres would be involved in addition to the afferent fibres of the organic seats of the different kinds of word memory.

Bastian utilized Charcot's division of "auditives", "visuals", "motors", and "indifferents" for speech. ("There may be a revival of sounds of words as we hear them in ordinary speech; there may be a revival of visual impressions of words as we have seen them in written or printed characters; and, lastly, there may be a revival of the feelings of muscular contractions concerned in the pronunciation of words.")[35] He upheld Jackson's theory that the auditory centre was the primary reviver of words in the majority of

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34. Ibid., p. 934.

35. Ibid., p. 935.

persons. After revival of words in the auditory centre, he contended that through commisural fibres the corresponding glosso-kinaesthetic elements were aroused to effect pronunciation. He also allowed for a degree of concurrent activity during speech in related portions of the visual word centre.

To explain more completely why he does not limit cerebral activity to narrowly localised centres, but rather widespread activity in varied regions, Bastian rationalizes on our acquisition of speech:

> Words that are heard are first of all associated in the mind of the child with external objects, so that such auditory impressions become linked, by means of associational fibres, with the organic seats of the several sensor impressions. . .
>
> The sight of the mother recalls the name "mamma", just as the sound of this word would revive the corresponding visual, tactile, and other images. After a time the auditory representative of the name becomes reinforced by glosso-kinaesthetic impressions as soon as the child learns to utter the word; later by visual impressions, when it learns to read, and by cheiro-kinaesthetic impressions when it has learned to write.[36]

Thus Bastian denied the existence of a "naming" or "concept" centre as proposed by Broadbent[37], Kussmaul, Charcot, and Lichtheim. Instead he sought to explain speech defects arising, not from a lesion in a localised centre for concepts, but from lesions situated in or near the sensory inlets:

> A destructive lesion of the portions of the cortex which are most remotely connected with the sensory inlets would

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36. Ibid., p. 938.

37. (Sir William Henry Broadbent:--London physician, 1835-1907)

> destroy the capacity of the patient for highly abstract reasoning, and would no doubt inflict considerable damage on the language in which abstract thought is embodied, but this condition would not be recognized as an aphasia; and even the intermediate portions of the cortex in which conceptive thought is carried on might be seriously damaged without giving use to a special speech disorder, inasmuch as any impairment of speech which might be present would only be regarded as a part of a general decay of the reasoning faculties. When, however, the lesion is situated in or near to the sensory inlets a disorder of language results which is out of all proportion to the general impairment of the reasoning faculties.[38]

He believed that the "annexes" of the sensory centers are thrown into functional activity more or less simultaneously with the combined sensory area. This inseparability makes distinct localization impossible; the anatomical substrata for perception, conception, and revival of linguistic symbols occupies "a very considerable extent of the cortex of both hemispheres."[39]

Up to about the year 1889, with the exception of the little-heeded Jackson, the trend of thought remained in the direction of localization. However, M. Allen Starr[40], one of the first Americans to become interested in aphasia, announced in 1889 that he had found no case to substantiate

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38. Bastian, op. cit., p. 942.

39. Ibid., p. 941.

40. (Moses Allen Starr:--American neurologist, 1854-1932)

Broca's pronouncement.[41] Then Freud[42], two years later, suggested that there was no such thing as a center.[43] Dejerine also declared against the autonomy of the so-called centers.[44]

In 1897, Lautzenberg (cited by Ellison, 1921) summarized the situation regarding the disorders of speech as follows: "The Outstanding characteristic of aphasia is that every observer thinks it necessary to formulate a theory of his own before recording the facts that he has noticed."[45]

To sum up the situation,

> Up to 1906 four centres of speech were considered; one centre of motor aphasia located in the third frontal convolution and three centres called centres of sensory aphasia: first, temporal convolution for word hearing; second, angular gyrus for word seeing, and third, second frontal convolution for the faculty of writing.[46]

The stage was now set for Marie's[47] dramatic entrance. The history from this turning point to the present has been treated in Part II of this study through detailed analyses of

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41. Anderson, op. cit., p. 11.

42. (Sigmund Freud:--Viennese neurologist, 1856-1939)

43. Nielsen, op. cit., p. 7.

44. Ibid.

45. Ellison, op. cit., p. 797.

46. Alfred Gordon, "Diagnostic Consideration of the Older and Later Views on Aphasia Based on the Anatomical Findings of a Case of Typical Motor Aphasia", Medical Journal and Record, 128:11:564, Dec. 5, 1928.

47. (Pierre Marie:--Parisian neurologist, 1853-1929)

the viewpoints of certain outstanding thinkers on the subject. However, to complete the historical picture, a cursory sketch of this period is herewith set forth.

Marie's 1906 pronouncements which so startled the neurological world were that (1) Broca's convolution takes no part in the producing of aphasia, and (2) aphasia is dependent upon intellectual loss or deficit.

The aftermath of the keen controversy caused by these statements revealed that Marie had simply redefined aphasia, recognizing only one type--sensory, or Wernicke's aphasia. Other types he dismissed as anarthria. Another service rendered the study of aphasia by Marie was the interest he restimulated in this speech defect.

The next major trend of thought was given impetus by the publication of Henry Head's psychological views. He argued that aphasia was not so much a defect in general intelligence, but rather, difficulty in abstract symbolization. It is not the intelligence which is primarily affected, Head argued, but behavior which suffered.

In his recognition of aphasia as the disturbance of a highly organized act, he anticipated today's organismic approach advocated by Kurt Goldstein.[48]

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48. See Chapter IX

However, the psychologists were not to take over the field unchallenged upon the appearance of Head's study. S. E. Henschen, a Swedish physician, published views almost the exact antithesis of Head's. His strict localization views, based on analysis of almost 1500 autopsy findings, greatly influenced the present-day neurologist J. M. Nielsen.

There evolved in the next few years, a realization that to successfully study aphasia one needs to use all three frames of reference--anatomical, physiological and psychological. This synthesis of methods of approach appears to be gaining wide support by the authorities of the present.

For a more complete comprehension of the changes in aphasic concepts since the turn of the century, various of the outstanding authorities have been treated in greater detail in Part II of this study.

PART II

HISTORICAL VIEWPOINTS

CHAPTER III

THE PSYCHOLOGICAL VIEWPOINTS OF HUGHLINGS JACKSON

Persuing his solitary way during the age of the diagram makers was the founder of English neurology, Hughlings Jackson. His minute clinical studies extended over an interval of thirty years and included the first recorded studies in speech psychology.

Although Jackson began publishing as early as 1864, his papers were quite generally neglected until Arnold Pick,[1] realizing their value, dedicated his "Die Agrammatischen Storungen" to Jackson in 1913. In view of Jackson's importance in the history of the study of aphasia, at first it is difficult to understand why his writings, which spanned a period from 1864 to 1893, were so long neglected. One of the reasons given is that "Jackson derived all his psychological knowledge from Herbert Spencer,[2] and adopted his phraseology almost completely. This has tended to alienate psychologists, blinding them to the truths underlying this somewhat uncouth nomenclature."[3] Another major reason for the

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1. (Arnold Pick:--neurologist and Professor of the German University of Prague)

2. (Herbert Spencer:--English philosopher, 1820-1903)

3. Henry Head, Aphasia and Kindred Disorders of Speech, (Cambridge: The University Press, 1926), p. 39.

comparative neglect of Jackson's writings might be his formidably difficult style of writing which is apt to discourage even the most enthusiastic student of language, speech, psychology and/or neurology. In addition, Jackson presented his dynamic approach in the face of an all-out functional interpretation at that time.

Speech to Hughlings Jackson is a function of mental activity. It is primarily a psychological problem, regardless of how closely it may be linked with the integrity of some portion of the brain-substance. Henry Head, in 1915, says in comment that "no one except Jackson has recognized that all the phenomena are primarily psychical and only in the second place susceptible of physiological or anatomical explanation."[4] Because of this belief he insisted upon an absolute distinction between psychological terms and anatomical or physiological terms.

Jackson recognizes that this mental process can be divided into two distinct forms: "(1) Intellectual, i.e., the power to convey propositions. (2) Emotional, i.e., the ability to exhibit stress of feeling."[5] But before we can elaborate on this dual aspect it is necessary first to understand Jackson's concept of propositionizing.

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4. Ibid., p. 32.

5. Ibid., p. 35.

Jackson's views of the problems of speaking and thinking deviate most strikingly from those of the neurologists, and anticipate by many years a viewpoint popular with students of language. He points out that it is not in words or signs only that we speak, but "in words or signs referring to one another in a particular manner."[6] Thus we visualize a sentence not as a pile of words but as a unit. Actually, Jackson considers single words as meaningless; they are propositions only if other words in relation are implied. This viewpoint was voiced in 1866 when scientists believed that we thought in words. Clarifying his conception of the proposition being the expression of a relation between two things, Jackson states: "It is not enough to say that speech consists of words. It consists of words referring to one another in a particular manner; and, without a proper interrelation of its parts, a verbal utterance would be a mere succession of names embodying no proposition."[7] Speaking is not simply the utterance of words; speaking is "propositionizing."

After stating that speech is essentially the formation of propositions, Jackson further points out that there is no difference in this respect between external and internal speech. The only difference lies in that uttered, or external,

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6. Ibid., p. 40.

7. Ibid., p. 40-1.

speech has not passed over the vocal organs. Or in Jackson's own words

> Anatomically and physiologically regarded, we say that the same nervous processes are concerned in internal as in external speech. The difference is that the excitation of these nervous processes in speaking to oneself is so slight that the nerve currents developed do not spread to the articulatory and vocal muscles; in speaking aloud the excitation is strong, and currents do reach those muscles. This fundamental similarity and superficial difference between internal and external speech must be kept well in mind.[8]

Or, in other words, we can say with Jackson that

> the anatomical substratum of a word is a nervous process of a highly special movement of the articulatory series. That we may have an "idea" of the word, it suffices that the nervous process for it energizes; it is not necessary that it energizes so strongly that currents reach the articulatory muscles.[9]

What then do we mean when we speak of the loss of the power to speak? Jackson would say we have lost the power to propositionize, implying either internally or externally. When Jackson speaks of the loss of the power to speak he does not mean that

> the speechless man has lost any "faculty" of speech or propositionizing; he has lost those words which serve in speech, the nervous arrangements for them being destroyed. There is no "faculty" or "power" of speech apart from words revived or revivable in propositions, any more than there is a "faculty" of co-ordination of movements apart

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8. Hughlings Jackson, "On the Nature of the Duality of the Brain," Medical Press and Circular, 1:19, Jan. 14, 1874. (Reprinted in Brain, 38:82, 1915)

9. Ibid., p. 85.

from movements represented in particular ways.[10]

Because of the tendency to attribute the involvement of some mental property, through their use, Jackson seldom used Greek terms such as agnosia, alexia, apraxia, et cetera.

If, then, the person who has lost speech cannot propositionize in any fashion--either internally or externally--

> we must not say that speech is external thought, for there is no essential difference betwixt internal and external speech. We speak not only to tell other people what we think, but to tell ourselves what we think. Speech is a part of thought--a part which we may or may not exteriorize. Again it is not well to say that thought is internal speech, for the man who is speechless (the man who has no internal speech) can think.[11]

Jackson explains the speechless man's ability to think in terms of his still possessing in automatic forms all the words he ever had. However he does recognize that the patient "will be lame in his thinking because not being able to revive words he will not be able to register new and complex experiences of things."[12]

"There are two ways in which words serve in thought; speech is but one way, and this, whether it be internal or external, is speaking physiologically, a function of the left

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10. Hughlings Jackson, "On Affection of Speech From Disease of the Brain," Brain, 1:304, 1879. (Reprinted in Brain, 38:114, 1915)

11. Henry Head, "Hughlings Jackson on Aphasia and Kindred Affections of Speech," Brain, 38:16, July, 1915.

12. Ibid., p. 17.

cerebral hemisphere."[13] In what other way do words serve thought? Jackson differentiates between propositionizing and receiving propositions. When receiving a proposition, the process is entirely automatic. To illustrate:

> When anyone says to me "Gold is yellow", I am, so to speak, his victim, and the words he utters rouse similiar ones in me; there is no effort on my part; the revival occurs in spite of me if my ears be healthy. Moreover, the speaker makes me a double gift; he not only revives words in my brain, but he revives them in a particular order--he revives a proposition. But if I have to say, "Gold is yellow," I have to revive the words, and I have to put them in propositional order. The speechless man can receive propositions, but he cannot form them--cannot speak.[14]

To understand this difference we must understand Jackson's dual concept of the brain's function and how the functions of the two hemispheres differ. Here Jackson voiced one of his major breaks with the doctrine of cerebral localization and its attendant ignoring of the recessive hemisphere. He believes that the brain acts as a whole in the language function and that a speech "function" could not be localized in the left hemisphere. Jackson observes that patients who had lost the power of speech still retained some forms of utterance. If a person whose left language area was destroyed could still utter words, the assumption of an

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13. Hughlings Jackson, "On the Nature of the Duality of the Brain", Medical Press and Circular, 1:19, Jan. 14, 1874. (Reprinted in Brain, 38:83, 1915)

14. Ibid., p. 84.

anatomical basis for them on the other side was mandatory. In 1874, Jackson set forth his belief that the right hemisphere of the brain--emotionally motivated--facilitated the automatic use of words, and the left hemisphere facilitated both automatic and voluntary use. These views were published in the same year as Wernicke's major contribution. Hence they were presented prior to the establishment of the anatomical basis for sensory aphasia. While he considers them both alike in that they both contain processes for the automatic use of words, the left hemisphere has additional processes for the voluntary use of words for speech. Specifically, "The left half of the brain is that by which we speak, for damage of it makes us speechless; the right is the half by which we receive propositions."[15] "The right is the automatic side for words and the left the side where automatic use of words merges into voluntary use of words (speech). In healthy persons, I suppose there is automatic revival of words prior to their voluntary revival (speech)."[16]

What then precedes the proposition? The prior process of the mind is an arousing in propositional form. This initial process Jackson called the "subject-proposition". This is a sort of "pre-conception" occurring in all voluntary

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15. Ibid.

16. Ibid., p. 86.

operations.

> The operation is nascently done before it is actually done; there is a "dream" of an operation as formerly doing before the operation; there is dual action. Before I put out my arm voluntarily I must have a "dream" of the hand as already put out. So, too, before I can think of now putting it out I must have a like "dream", for the difference betwixt thinking of now doing and actually doing, is, like the difference between internal and external speech, only one of degree.[17]

What proof, if any, have we that words must be automatically revived before voluntarily used or uttered in a proposition? Jackson states:

> That automatic action must precede voluntary action is, I submit, certain. In gross physical operations we see that it is so. We cannot use the hand before we fix the wrist, nor the arm before the shoulder is fixed, and in heavy strains with this limb, the chest is first fixed and the glottis is closed, after a full inspiration. The more automatic muscles must be in action before the more voluntary, or there would be no point d'appui. Taking a psychological illustration, we note that we desire before we will.[18]

Returning now to the theory of Jackson's with which this chapter opened, let us examine further his dual aspect of intellectual and emotional expression. Jackson designates those dual aspects as superior and inferior speech. The inferior speech, or automatic service of words Jackson believes to be associated with activity of the right hemisphere. The left hemisphere (in right-handed people) not

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17. Head, op. cit., p. 18.

18. Hughlings Jackson, "On the Nature of the Duality of the Brain (Continuation)", Medical Press and Circular, 1:41, Jan. 21, 1874. (Reprinted in Brain, 38:93, 1915).

only subserves the use of words in propositional forms, but also had a lower use in the automatic service of words. Although these more automatic activities are centered in both hemispheres, the power of exciting words in propositional forms is in the left only.

This line of thought can be easily traced to Herbert Spencer who said in his essay, "The Origin and Function of Music":

> All speech is compounded of two elements, the words and the tones in which they are uttered--the signs of ideas and the signs of feelings. While certain articulations express the thought, certain vocal sounds express the more or less of pain or pleasure which the thought gives.[19]

Through the means of voice we give outlet to our emotions. Voice "expresses various states of feeling common to man, rather than ideas special to the individual."[20]

Jackson likens swearing to a strong expression of the emotions. In fact, he does not consider it a part of language, strictly speaking. Rather,

It is a habit which has grown up from the impulse to

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19. Hughlings Jackson, "Clinical Remarks on Emotional and Intellectual Language in Some Cases of Disease of the Nervous System," The Lancet, 1:174, Feb. 17, 1866. (Reprinted in Brain, 38:45, 1915.

20. Hughlings Jackson, "Loss of Speech: Its Association with Valvular Disease of the Heart and with Hemiplegia on the Right Side--Defects of Smell--Defects of Speech in Chorea--Arterial Lesions in Epilepsy," Clinical Lectures and Reports, London Hospital, 1:388, 1864. (Reprinted in Brain, 38:53, 1915).

> add the force of passing emotions to the expression of ideas. It belongs to the same general category as loudness of tone and violence of gesticulation.[21]

While it is true that oaths consist of articulate words, they nevertheless are generally used

> not to express ideas, but to make up by vigour in delivery what is wanting in precision of expression. They may, indeed, be considered as phrases which emotion has filched from the intellect, to express itself in more definite terms than it could do by mere violence of tone or manner.[22]

Similarly Jackson explains the stereotyped swear-phrase so frequently the only vestige remaining of language in an aphasic. Expressions such as "Damn it", "Bless my life!", and "Oh Dear!", when used in anger or vexation, are used to clothe feelings rather than to convey intellectual propositions. Jackson feels that these ejaculatory expressions are prompted by the emotions and not by the will. As a possible explanation of the production of these phrases Jackson suggests that they may be reflexes:

> It is quite obvious that they are not voluntary, as the patients cannot repeat the phrases. The will cannot act, but, somehow, an emotion, e.g., anger, gets the words passed through the convolution of language. Just as a paralysed foot will jump up when the sole is tickled, so these words start out when the mind is ex-

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21. Ibid., p. 40.

22. Ibid.

> cited. Such ejaculations as I have mentioned would have become easy of elaboration by long habit, and would require but slight stimulus for perfect execution.[23]

It is quite obvious from the foregoing that Jackson believes that both sides of the brain are probably educated. However, it is the left that begins to act. Those phrases which are in a sense involuntary have become so by habit and may be the result of the action of the right hemisphere only. In comment Jackson states: "How from the right side they are set a-going I do not know any more than I know how a person laughs, not only without effort, but even when he tries to keep quiet."[24]

If the function of the two hemispheres now seems to be confused, let us review Jackson's analysis.

> The division we made was not that the left half of the brain serves in speech, and the right in receiving speech and in other ways, but that "nervous arrangements for words used in speech lie chiefly in the left half of the brain", and "that the nervous arrangements for words used in understanding speech (and in other ways) lie in the right also." It is believed that the process of verbalizing and every other process is dual, but that the more automatic a process is, or becomes by repetition, the more equally and fully is it represented double in each half of the brain. But the utterances show too, for the most part, that the speech possible by the right side of the brain is inferior speech. In

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23. Ibid., p. 41.

24. Hughlings Jackson, "Notes on the Physiology and Pathology of Language", Medical Times and Gazette, 1:659, June 23, 1866. (Reprinted in Brain, 38:53, 1915).

> nearly all cases it was well organized, automatic or "old", and nearly every utterance required a special occasion; was, to speak popularly, surprised out of the patient by a sudden accustomed stimulus. And it is to be borne in mind that the patient cannot repeat, say voluntarily, what he thus utters.[25]

In cases of brain damage, it is intellectual language that suffers most; emotional language is conserved. Not only do vocal inflections go undisturbed, but so do smiles and gestures. Jackson expounds his general law underlying these speech disturbances that "voluntary power is diminished with retention of the power to carry out the same movement in a more automatic manner."[26] J. M. Nielsen in 1947 makes essentially the same explanation:

> There is no doubt that the anatomic and physiologic patterns for the utterance of any word are still present in the brain after destruction of Broca's convolution of the major (left) side...The patterns are located anatomically in the right Broca's convolution as Jackson thought and need only the proper stimulus to bring them into action. They can be stimulated through the emotions, though the mediation is unknown.[27]

Far too frequently we hear that disease or injury has <u>caused</u> the utterances peculiar to various aphasics. To these erroneous observations of speech disturbances Jackson, as far

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25. Hughlings Jackson, "On Affections of Speech From Disease of the Brain--II", <u>Brain</u>, 2:208, 1880. (Reprinted in <u>Brain</u>, 38:146, 1915).

26. Head, <u>op. cit.</u>, p. 9.

27. Nielsen, <u>op. cit.</u>, p. 21.

back in 1868, applied the general law of nervous activity that "a destructive lesion can never be responsible for positive symptoms; pure destruction produces negative effects, and any positive symptoms are the consequence of the released activity of lower centres."[28]

In explaining the speech phenomena he notes that

> the disease is destruction of nervous arrangements, and that could not cause a man to do something; it has enough to answer for in leaving him unable to speak. The utterances are effected during activity of nervous arrangements which have escaped injury. This remark may seem a truism here, but in more complicated cases it is very common to hear of positive symptoms being ascribed to negative lesions--to loss of function of nervous elements. It is common at any rate for disease to be thought of vaguely as something "disordering the functions of the brain". . . It is an error to ascribe such positive symptoms as the recurring utterances in speechless men, the erroneous words uttered by those who have defect of speech. . . etc., to loss or to defect of function. These positive mental symptoms arise during activity of lower centres or lower nervous arrangements which have escaped injury.[29]

With this analysis, then, we must recognize the existence of a negative and a positive side to every speech affection due to neurological pathology. The negative aspect is the patient's inability to speak, to write, to read, or to express by signs. The positive symptoms such as swearing or

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28. Head, op. cit., p. 5.

29. Hughlings Jackson, "On Affections of Speech From Disease of the Brain--III", Brain, 2:323, 1880. (Reprinted in Brain, 38:154-5, 1915).

uttering other emotional expressions are the expression of lower mental activities. Whatever function exists in a given case does not depend directly on the lesion, but on the function of the cerebral structures remaining. Thus, "the disease causes loss of speech; it permits the increased dischargeability of the right half."[30]

> Destruction of function of a higher centre is a removal of inhibition over a lower centre. The lower centre becomes more easily dischargeable, or popularly speaking, "more excitable", and especially those parts of that centre which are in activity when control is removed.[31]

> That parts suffer more as they serve in voluntary, and less as they serve in automatic operation, is, I believe, the law of destroying lesions of the cerebral nervous centres.[32]

In analyzing the recurring utterances of one whose left half of the brain is so extensively damaged that he cannot speak, Jackson offers the hypothesis that "the words were being revived at the time when the patient was taken

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30. Ibid., p. 158.

31. Ibid., p. 157-8.

32. Hughlings Jackson, "On the Anatomical and Physiological Localization of Movements in the Brain", The Lancet, 1:84, Jan. 18, 1873. (Reprinted in Brain, 38:76, 1915).

ill."[33] He notes that these words are the result of certain nervous arrangements which remain permanently in a state of dischargeability far above normal.

It would seem, from this discussion of Jackson's notes, that we have begun stressing the psychological aspect of speech only to end up emphasizing the anatomical basis of speech. To clarify his double emphasis Jackson states:

> We have, as anatomists and physiologists, to study not ideas, but the material substrata of ideas (anatomy) and the modes and conditions of energizing of these substrata (physiology). Where most would say that the speechless patient has lost the memory of words, I would say that he has lost the anatomical substrata of words.[34]

In the light of this explanation we can more readily understand the "altering" of a person's disposition in cases of deterioration of the brain--how our more animal instincts and desires are no longer subordinated. "There is reduction to a more automatic condition; there is dissolution, using this word as the corresponding opposite of evolution. The weaker the mind the more do the more automatic desires have

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33. Hughling Jackson, "On Affections of Speech From Disease of the Brain--III", Brain, 2:323, 1880. (Reprinted in Brain, 38:157, 1915).

34. Hughlings Jackson, "On the Nature of the Duality of the Brain", Medical Press and Circular, 1:19, Jan. 14, 1874. (Reprinted in Brain, 38:84, 1915).

their own way."[35]

In retrospect, Weisenburg[36] and McBride list the following ideas of Hughlings Jackson as having become foundation stones in modern theories:

> his emphasis on aphasia as a psychological problem, his attack through the clinical findings rather than the pathological destruction, his concept of higher and of lower, more automatic forms of response, his appreciation of the complex relations of language and thinking, his discovery of related disorders later called apraxia and agnosia, and some of his important views on localization.[37]

The same authorities list as no longer significant

> his differentiation of types of disorder, much of his theory of pre-verbal formulation, his opinions on the role of the right hemisphere in speech, and his observational methods as opposed to the test methods now in use.[38]

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35. Hughlings Jackson, "On the Anatomical and Physiological Localization of Movements in the Brain", The Lancet, 1:84, Jan. 18, 1873. (Reprinted in Brain, 38:79, 1915).

36. (Theodore Weisenburg:-American neurologist, 1876-1934)

37. Theodore Weisenburg and Katherine E. McBride, Aphasia: A Clinical and Psychological Study. (New York: The Commonwealth Fund, 1935), p. 19.

38. Loc. cit.

CHAPTER IV

THE CONTROVERSIAL LOCALIZATION IDEAS OF MARIE AND OPINIONS OF HIS CONTEMPORARIES

In 1906 a series of papers appeared which caused a considerable controversy in neurological circles. Pierre Marie published three papers in the Semain Medicale on "Revision of the Question of Aphasia". These, and the subsequent series of papers culminating in Moutier's[1] monograph of 1908, demanded a revision of the whole subject of aphasia. Marie boldly struck out with the unqualified declaration that the inferior left frontal convolution (Broca's region) played no particular part in language functions; it could not be considered a speech center. Since Marie's theory explicitly denied one of the hitherto incontrovertible facts of the literature, opponents arose in storms of abuse and hotly contested his localization views.

Besides denying that lesions in Broca's convolution take no part in the production of aphasia, Marie denied the existence of auditory, visual and motor speech centers wherein speech memories are registered in the cortex. Marie recognized no distinction between motor and sensory aphasia and, in place of the classical doctrines on localization, postulated

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1. (F. Moutier:--Marie's co-worker)

only one speech center diffusely localized in the left temporo-parietal lobe. Hence, he insisted that there existed only one type of aphasia from a lesion of the cortex--sensory, or Wernicke's aphasia, which he called "true" aphasia. This type, he stated, always resulted from a destruction of the region which he called Wernicke's zone (an area around the end of the Sylvan fissure). Since he interpreted this center as a region of intelligence specialized for language, and not solely a center of sensory images, he also challenged Broca's classic description that the aphasias were a disturbance of speech with preservation of intelligence. "He further complicated the picture by introducing a psychological factor, in the form of a disturbance of comprehension and a reduction of the intellectual capacity."[2]

Thus we have Marie's "true" aphasia defined as

> not the inability to utter words, but the inability to understand what is said, what is written or what is heard, and the disability to represent mental concepts by their accepted vocal or written equivalents. It is not, therefore, the result of the destruction of a "centre" in the old sense of the word, but a disorder of the integrating mechanism of the nervous system (Sherrington) a condition of"diaschisis" (v. Monakow) or "apraxia" according to Liepmann.[3]

Wernicke's zone he defines as

> composed of the supramarginal (inferior parietal) and inframarginal gyres and the posterior extremities of the

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2. German, op. cit., p. 365.

3. Core, op. cit., p. 314.

> first two temporal convolutions,--in other words that cortical region composed of the gyral masses which curve around the extremities of the Sylvian and the parallel fissures.[4]

A lesion to this zone gives rise to a disturbance of intelligence, Marie contended.

Pure motor aphasia (or subcortical motor aphasia) he called anarthria. This defect came with subcortical lesions of what he called the "lenticular zone" in either hemisphere. The lenticular zone, or "quadrilateral field" of Marie, is

> bounded anteriorly by the white substance of the third frontal convolution, posteriorly by Wernicke's area, externally by the insula, and internally by the wall of the third ventricle. It, therefore, comprises the external capsule, the caudate and lenticular nuclei, the anterior and posterior portions of the internal capsule, and the optic thalamus.[5]

"Above, this quadrilateral area was prolonged to include the outlying gyri of the convexity of the cortex, while below it included the subthalamic region."[6] Any lesion within this zone causes anarthria; lesions behind it cause aphasia. (J. M. Nielsen calls our attention to the fact that Marie "overlooked the fact that an anarthric cannot properly use his muscles for any purpose. An aphasic may use them for any

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4. Charles K. Mills, "Discussion on Aphasia, Especially With Reference to the Views of Marie", The Journal of Nervous and Mental Disease, 34:459, 1907.

5. German, op. cit., p. 365.

6. Osnato, op. cit., p. 420.

purpose but speech").[7]

Broca's aphasia he declared a combination of the true aphasia and anarthria due to a double lesion: one in the quadrilateral area, and one in the general speech intelligence area (Wernicke's area) or the tracts to or from it.

In evaluating Marie's contribution we must not overlook the value of his "calling attention to a chapter of pathological physiology which no longer could remain unchallenged."[8] He is praised by J. M. Nielsen for "reopening the whole subject, which really needed reconsideration"[9], and by Henry Head for his fight for simplification. Both of these outstanding men, however, label much of his work as being merely a matter of changing definitions--"the greater part of the discussion of the clinical manifestations was purely a verbal battle."[10] He simply redefined aphasia and this made his statements sound startling.

While Marie's theory of intellectual defect has enlisted many adherents, outstanding being Michael Osnato, Albert G. Odell[11] noted in 1930 that "by far the greater

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7. Nielsen, op. cit., p. 9.

8. Alfred Gordon, "A Contribution to the Study of Aphasia", New York Medical Journal, 97:14, Jan-June, 1913.

9. Nielsen, op. cit., p. 9.

10. Henry Head, "Aphasia: An Historical Review", Brain, 43:399, 1920.

11. (Albert Grove Odell:--Clifton Springs, N. Y., neurologist and psychiatrist, 1878-____)

number accept the idea of organic disturbance and locate speech centers in the cortex."[12]

In 1942 J. M. Nielsen and Arnold P. Freidman published the results of a study to determine what semblance of truth there may have been in Marie's teaching. In a clinical study of 12 cases they found that

> within the quadrilateral space of Marie the external capsule has much to do with language by virtue of its conveying of impulses from the language formulation area forward to Broca's convolution. Destruction of it causes either paraphasia and agraphia or motor aphasia and agraphia.[13]

They go on to say that

> after a destructive lesion of the external capsule the patient will either have to use the speech mechanism of the minor hemisphere as a unit or he will perform one of the two functions (formulation or emission) on the minor side and the other by means of the major side. If the minor area of Broca is utilized, motor aphasia results until time is available for training. If the minor area of formulation is used and the impulses are conveyed across the corpus callosum to the major Broca's convolution paraphasia results. A lesion of the anterior limb of the major internal capsule will produce dysarthria or anarthria by virtue of interrupting the projection fiber tracts from the precentral gyrus to the pons and medulla. Such a disturbance has no relation to aphasia.[14]

They conclude that his observations have some use if properly appraised. However, he exerted a definitely harmful influence by assuming his predecessors completely in error. He could

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12. Albert G. Odell, "Aphasia, A Case Report", The Clifton Medical Bulletin, 16:4:163, October 1930.

13. J. M. Nielsen and Arnold P. Friedman, "The Quadrilateral Space of Marie", Bulletin of the Los Angeles Neurological Society, 7:3:135, 1942.

14. Ibid., p. 135-6.

have helped to clarify the subject instead of retarding progress if he had given due credit for scientific observation.

One of Marie's most articulate followers was Michael Osnato of New York City. In attempting to cope with the problem of aphasia Osnato refers to the failure of anatomy and pathology in elucidating the problem. He therefore sought to enlist the aid of clinical medicine.

Prior to the time of Marie and Moutier's publications, Osnato felt that classifications of "centers" were based more on theory than upon trustworthy pathological evidence. These two men, however, examined all the autopsied cases of aphasia which were reported in the literature from the time of Broca in 1861 up to 1905. They called attention to the sparcity of material for Broca's original statement. Osnato gives a good account of the clinical statistics underlying Marie and Moutier's contentions:

> Between the years of 1861 and 1906, 304 cases with autopsy have been published upon the subject of the third left frontal convolution, or Broca's motor speech area. Of these, 201 cases were not of value for statistical purposes because in 26 of these cases the clinical or pathological records were incomplete and in the remaining 175 cases the lesions were too vast for accurate localization. There remained, therefore, 108 cases in which the lesions were localized, the records and clinical histories being of sufficient detail for use in the study of the subject. Of these 108 cases only 19 showed facts which appear to support Broca's theory, and in 11 of these, the lesion was not cortical at all, but was found in the subcortex. Of the 108 cases which were considered of sufficient value, only 8 showed the cortex of the left third frontal convolution to be affected. All the other cases were almost entirely against the classical theory that

> Broca's area was an area for the motor mechanism of speech. Including the 11 subcortical cases, only 19 were apparently favorable to this theory and even in a number of these cases there were ample lesions. In one there were extensive lesions of the right temporal lobe with no mention as to whether this patient was left-handed or not.
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> Both Marie and Moutier agree that there does not exist in medical literature one observation of Broca's aphasia in which a single lesion strictly localized to the foot of the left third frontal gyrus has been proved to exist upon autopsy.[15]

Citing other investigations, Osnato observes that only four of the 104 cases studied by Frankel and Onuf (cited by Osnato, 1921) seemed to confirm Broca's theory of the localization of motor speech. However,

> they found at the same time 5 cases of motor aphasia in which the left third frontal convolution was absolutely intact and the subcortical association fibers going from and to this part of the cortex showed no trace of degeneration.[16]

In view of these clinical statistics Osnato concurs with Marie's warning that "to use the symptom of aphasia as a localizing diagnostic aid is impossible and a dangerous procedure."[17]

In attempting to disprove the existence of any sharply defined, diagrammatic localization of speech functions in any

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15. Osnato, op. cit., p. 420.

16. Michael Osnato, "Speech, Its Development and Integration With the Intelligence", Neurological Bulletin, 3:2:58, Feb., 1921.

17. Ibid., p. 56.

group of "centers", Osnato quotes Piersol[18] on the development of the cerebral cortex. (Except for the immediate vicinity of the upper end of the Rolandic fissure)--

> the cerebral cortex at birth is unprovided with association fibers which have acquired their medullary coat and are therefore capable of functioning. Within the early months after birth, however, the myelination of these, as well as of other tracts, progresses rapidly, although this process is not even moderately completed until after the lapse of several years. Indeed there is sufficient evidence to believe that myelination of additional fibers continues so long as intellectual effort is progressive, the demands made by education and special mental exercise being met by a corresponding completion of additional association fibers.[19]

In addition to this argument based on anatomical evidence, Osnato describes the development of speech in the individual child and notes how the method of education of one child differs from another.

> since the variations occurring with the individual child in the use of the various special senses (including the proprioceptive as one of these) are so great for the production of simple associations, and later of conception and thought which find their expression in speech, the nature of the development in the brain of this function will also vary greatly.[20]

Insofar as one child may learn speech by purely auditory means, while another has the concurrent visual representation, and still another may grasp the art of talking by tactile and

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18. (George A. Piersol:--American physician, 1856-1924)

19. Osnato, op. cit., p. 55.

20. Ibid., p. 54.

kinesthetic training, each has developed, to some extent, different parts of the brain and different association fibers. This extreme possibility of variation--when we consider that each receptor is in potential synaptic reference to every effector--undoubtedly is the reason why no diagrammatic scheme of definite speech intelligence centers can be applied to everyone alike.

Further militating against the theory of one particular speech center is the clinical observation that aphasics improve with re-education. Undoubtedly for any improvement to occur it would be necessary for new association pathways to replace those previously used which are now destroyed by injury or disease. Therefore, "This observation alone would be enough to cast serious doubt on any notion that only certain parts of the cortex are capable of developing the power to control speech."[21]

Osnato, in attempting to prove his contention that the whole cortex is indivisibly associated with the development of the function of speech and its expression, quotes at great length various of the neurologists of his day. He voices his agreement with such men as the neurologist C. J. Herrick in the belief that

> no cortical area can properly be described as the exclusive center of a particular function. Such "centers"

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21. Ibid., p. 59.

> are merely nodal points in an exceedingly complex system of neurons which must act as a whole in order to perform any function whatsoever.[22]

The only sense in which there is a specific localization of function in the cerebral cortex is

> in the sense that particular systems of sensory projection fibers terminate in special regions (the sensory projection centers), that from other special regions (the motor projection centers), particular systems of efferent fibers arise for connection with the lower motor centers related to groups of muscles concerned with the bodily movements, and that between these projection centers there are association centers, each of which has fibrous connections of a more or less definite pattern with all other parts of the cortex. The destruction of any part of the cortex or of the fiber tracts connected therewith, involves, first, a permanent loss of the particular functions served by the neurons affected, and, in the second place, a transitory disturbance of the cortical equilibrium as a whole (diaschisis effect of von Monakow). Specific mental acts or faculties are not resident in particular cortical areas, but all conscious processes probably require the discharge of nervous energy throughout extensive regions of the cortex, and the character of the consciousness will depend in each case upon the dynamic pattern of this discharge and the sequence of function of its component systems. This pattern is inconceivably complex and only the grosser features are at present open to observation by experiment and pathological studies.[23]

The two leading contestants of Marie's doctrine were M. and Mme. Déjerine. They maintained that aphasia was not dependent upon intellectual loss or deficit. Rather, "the diminution of intelligence sometimes exhibited by aphasics is dependent upon disruption or disturbance of the cerebral

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22. Michael Osnato, "Aphasia and Associated Speech Problems (Continued)", Neurological Bulletin, 2:1:51, Jan. 1919.

23. Ibid., p. 50-1.

mechanism of speech."[24]

The Déjerines insisted upon the localization of speech and attributed true motor aphasia to a lesion of the "anterior speech zone"--"a region that is very variable in its extent, but which may be said to comprise the area of Broca at the foot of the third left frontal convolution, the anterior segment of the island of Reil and a variable area of the adjacent regions."[25] Sensory aphasia they attributed to a lesion in the zone of Wernicke. Anarthria was believed to be due "either to a bulbar lesion, or if of central origin, to a degeneration of the internal capsules."[26]

Also aligned against Marie was the American investigator, Charles K. Mills[27]. Although he was a firm believer in cerebral localization, "he was also aware of the philosophical and the psychological aspects of the problem, but seemed to slight them in favor of more tangible and, as he saw them, more practical facets of the question."[28] He wrote of functional areas, sub-areas and centers.

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24. Mills, op. cit., p. 460.

25. Core, op. cit., p. 317.

26. Ibid.

27. (Charles K. Mills:--Philadelphia neurologist, 1845-1931)

28. Anderson, opcit., p. 18.

His interpretation of "centers" is indeed interesting:

> Much objection has been made at times to the use of such expressions as centres and memorial images, because of their supposed lack of definiteness; but it matters little how these centres of function are designated or explained, if the fact is recognized that they are in some sense foci of activity resulting in special phenomena.[29]

In limiting the region, lesion of which produces motor aphasia, Mills includes the anterior portion of the insula in addition to the hinder part of the left third frontal convolution. He also believed "it is not impossible that it may also in some individuals take in a part of the first temporal convolution where this merges with the insula."[30]

Although Mills accepted Marie's idea that an intellectual deficit underlay disturbance and impairment in the comprehension of language, he demanded the necessity of adhering to Dejerine's explanation that "this deficit in its turn is dependent upon destruction or disruption of a mechanism which is made up of associated centres."[31] If we did not accept this maxim, Mills felt we would be thrown back upon some ancient metaphysical theory of the existence of mind outside of the body.

Another protagonist in the theoretical battle at this

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29. Charles K. Mills, "The Different Theories of Aphasia", New York Medical Journal, 99:861, May 2, 1914.

30. Ibid., p. 864.

31. Ibid., p. 863.

time was von Monakow.[32] He considered speech a function of the entire cortex. He insisted on the difference between locating speech itself and localization of aphasic symptoms. In his Die Lokalisation im Grosshirn he described the anatomical region attacked by aphasia, extending the motor speech area to include Broca's area, the operculum, the pars triangularis, and the anterior part of the insula.[33]

Von Monakow is of interest chiefly for his "diaschisis" theory. This is a doctrine based upon recognition of integrating mechanisms. When one suffers a cerebral lesion, von Monakow ascribes its importance, not as damage to this one particular area, but as a disturbance of a widespread function due to damage of one of the vital parts. He suggests that the concept of both centers and associating mechanisms be accepted.

> The very existence of associations indicates something to be associated. Associating structures are not simply paths which come from nowhere and go nowhere. They are lines of connection between important structural and dynamic foci.[34]

In 1910 the voice of Adolf Meyer[35] made itself heard on the subject of aphasia through the Harvey Lecture. Drawing upon his wide clinical experience, he warned against strict

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32. (Constantin von Monakow:--Swiss histologist, 1853-1930).

33. German, op. cit., p. 365-6.

34. Mills, op. cit., p. 863.

35. (Adolf Meyer:--American psychiatrist, 1866-____)

adherence to the popular diagrammatic conception of aphasia. He pointed out the keen desire for condensation and simplification, but at the same time noted that the disorders were compound and could not be thus easily simplified. He did not want mixed terminology, but insisted that "the only real and lasting solution comes from the utilization of adequately defined anatomical and functional mechanisms."[36]

Jeanette Anderson notes that it was Meyer who bridged the gap between the anatomical and the psychological approaches to the problem of aphasia.[37] In the clinical handling of aphasics Meyer "emphasized the importance of a knowledge of the patient's educational level, a complete neurological examination, and accurate tests of hearing, vision, and other senses."[38]

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36. Anderson, op. cit., p. 18.

37. Ibid., p. 19.

38. Weisenburg and McBride, op. cit., p. 26.

CHAPTER V

HENRY HEAD AND REVIVAL OF THE PSYCHOLOGICAL APPROACH

Just as the Marie-created tempest was dying down, the medical world was given another interpretation and system of aphasic classifications with which to contend. Henry Head's theories, appearing in the early 1920's, were in many ways a modernized form of one of the earliest theories, that of Hughlings Jackson. Jackson's theory that aphasia is a disturbance of the power to form propositions was couched in different terminology. Head and his followers looked upon all aphasic manifestations as "defects of symbolic formulation and expression" rather than a disturbance of the memory of words or the faculty of language. By symbolic formulation and expression Head meant "a mode of behaviour, in which some verbal or other symbol plays a part between the initiation and execution of the act."[1]

Thus he considered aphasia "a defect in the special mental activity of symbolic expression such that the greatest difficulty will occur in higher propositional speech or more abstract symbolization; it is not a defect in general intelligence."[2] Jeanette Anderson explains Head's basis for the

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1. Henry Head, Aphasia and Kindred Disorders of Speech, (Cambridge: The University Press, 1926), p. 211.

2. Bender, op. cit., p. 518.

latter statement:

> Head tended to emphasize the formulative and expressive side of speech rather than the integrative, and the symbol as a unit of expression rather than as a part of the symbolic significance of the whole. For this reason perhaps, he did not consider speech as a part of the higher integrative function, which is after all general intelligence and which would tend to rise and fall together with speech in lesions of the cerebral centers. He did, however, recognize that in aphasia the symbol is affected so far as it expresses relational processes in constructive thinking.[3]

Aphasics appear more stupid than others because they cannot satisfactorily participate in verbal intercourse. Because of this isolation from the sources of mental life, many aphasics gradually deteriorate. However, "general intelligence," according to Head, is not primarily affected. Behavior suffers specifically.

In Head's classification cerebral localization relinquished its predominant importance. He insisted on a psychological interpretation of speech defects. He stressed the need for studying aphasia as a disorder of function. It is the disturbance of a highly organized act which results in a defect of cerebral activity, not a destruction of a strictly definable anatomical center.

> Gross organic injury does not remove the structural basis of a "faculty" or the repository of "images", but disturbs the march of events necessary for the perfect accomplishment of some acquired act; this can be relearnt, even after

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3. Anderson, op. cit., p. 518-9.

the ablation of the whole of its "cortical centre".[4]

Head definitely denied the existence of "centres" for speaking, reading, writing, or any other form of behavior comprised in the normal use of language. Instead of localizing certain cortical areas which actuate speech, Head noted that

> the processes which underlie an act of speech run through the nervous system like a prairie fire from bush to bush; remove all inflammable material at any one point and the fire stops. Clearly, it is as reasonable to call the site at which there is discontinuity of the nervous mechanism a "speech centre" as it would be to call the place where the fire ceased a "fire centre".[5]

He maintained that the normal production of any psychic act has no exact correspondence with the independent activity of any particular group of cells. These "centers" are solely integrating foci where central neural activities undergo integration and other changes in relation to one another. When they are physiologically disturbed by a lesion, Head postulated the resulting disturbance as a loss of "vigilance," or of the "high-grade physiological efficiency"[6] underlying all types of activity from muscle tonus to the complex

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4. Sr. Mary Jean Schwalback, "Motor Aphasia: A Case Study" (Unpublished Master's Thesis, Marquette University, Milwaukee, Wis., 1940)

5. Tudor Jones, "Brain and Speech", Nature, 113:499-500, April 5, 1924.

6. Henry Head, op. cit., p. 486.

processes of speech.

When this vigilance is lowered by any organic or functional condition, the most complex forms of behavior are the first to suffer.

> Vigilance seems in some way to express the tone of the nervous activity which functions by way of integration and expresses itself in intelligence on the psychic side. Focal brain lesions prevent the normal fulfillment of some specific form of behavior, but nevertheless the reaction that follows any given situation still expresses the response of the organism-as-a-whole under the new conditions, or, one may say, the new-organism-as-a-whole, which is, perhaps, reacting at a lower organized level or like a more primitive organism[7]

The aphasic clinical manifestations, he contended, could not be classified as "motor" and "sensory" (or as motor, visual, and auditory), since speech is an integrative function standing higher in the neural hierarchy than motion and sensation. It is "built up on a level of integration superior to that of motion."[8] To analyze in terms of motion and sensation would be unsuitable since they imply interference with a definite physiological function.

Head's classification was entirely new, discarding all of the previous ones. He used none of the old terms, and based his study on exhaustive examinations with living material. His classification uses grammatical terminology and is based on language concepts. The defects are divided into

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7. Bender, op. cit., p. 518-19.

8. Jones, op. cit., p. 500.

verbal, nominal, syntactical, and semantic aphasia, according to the most prominent clinical symptom. In setting forth this arbitrary classification, he acknowledges that they are not fixed and that there is overlapping and indefiniteness.

Verbal aphasia manifests itself in disturbances in external speech, or articulate expression. A difficulty is experienced in evoking words. The vocabulary is greatly restricted. Errors are recognized, however. Drawing and the understanding of pictures remain unimpaired. The British physician J. M. Gill attributes this type to a lesion "situated in the lower part of the precentral gyrus."[9]

In Syntactical aphasia the patient tends to talk jargon. He is apt to omit or slur prepositions, conjunctions, and articles. In addition to the articulation of the words, the rhythm of the phrase is also defective. There is also a want of grammatical coherence. Writing, too, is impaired. However, that which is read may be understood. The lesion, according to Gill, is "in the temporal lobe."[10]

Nominal aphasia is the defective use of or understanding words as names or indicators of letters, words, or objects. The patient may recognize objects, but he cannot name them on written or verbal command. Names may be correctly repeated

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9. J. M. Gill, "On Aphasia", The Medical Journal of Australia, 1:4:125, Jan. 27, 1934.

10. Loc. cit.

when heard, but they are not understood. In addition, both writing and reading are affected. "The lesion is in the occipital lobe."[11]

In Semantic aphasia the patient cannot recognize the full significance of words and phrases. He cannot comprehend "the general significance of a word as part of a complete act of language."[12] The ability to draw or play games is also lost. The lesion is "in the supramarginal gyrus."[13]

The related defect of "apraxia" consists of inability to execute purposive movements or complex acts in the absence of paralysis and with perfect comprehension of the meaning and ultimate intention of the commands. "Agnosia" Head defined as a perceptual defect--the failure to recognize objects, pictures or sounds in the absence of sensory defects.

In comparison with the classifications generally accepted at the time of Head's publications, his classification seems superior to them in theory. "It is based on the right principle; that is, the differentiation of forms of language deterioration in terms descriptive of the predominant psychological changes."[14] Although excellent in theory, it is inferior in practice in that "it fails to cover too many cases

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11. Ibid.

12. German, op. cit., p. 376.

13. Gill, op. cit., p. 125.

14. Weisenburg and McBride, op. cit., p. 51.

of so-called motor and amnesic aphasia."[15]

Later attempts to correlate these types of defects with localized cerebral lesions led to the conclusion that "neither the areas of localization nor the resulting loss of function determines the 'elements out of which language is composed', but rather the manner in which 'the use of language can be broken up'".[16]

Head's greatest contributions to the study of aphasia have been cited by Anderson as his "exposition of heirarchies, his study of functionizing at higher levels and his discussion of semantic aphasia."[17]

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15. Anderson, op. cit., p. 30.

16. German, op. cit., p. 366.

17. Anderson, op. cit., p. 25.

CHAPTER VI

S. E. HENSCHEN AND THE ANATOMICO-CLINICAL INTERPRETATION

Almost diametrically opposed to the psychological analysis of Head was the work of the Swedish professor emeritus of medicine at Stockholm, Salomon Eberhard Henschen. Henschen began his major research on aphasia in 1917 at the age of 70. He insisted that the question of aphasia could be placed on a sound basis only through a thorough study of the clinical aspects of aphasic cases, correlated with the pathological findings in the brain. As a result of his insistence on a sound anatomico-clinical basis, he gathered together 60 of his own autopsies and personally collected autopsy material in Germany, Austria, France, England and Italy. In addition, he wrote to Russia and America for further cases. His efforts resulted in the accumulation of detailed records on nearly 1500 aphasic patients.

Henschen's synthesis of all this data resulted in a monograph of three large volumes of abstracts and analysis published in 1920 and 1922. He carried anatomic localization in the brain so far as to assert that every psychological activity could be localized--even logical thought.

The question of the function of the minor cerebral hemisphere in speech was discussed by Henschen in great detail. He agrees with Jackson in the contention that the

minor hemisphere takes over the function of speech in cases of injury to the major hemisphere. Specifically, he states that "the localization of a motor speech center, or any other specialized speech center, could not be upheld were it not for the initiation of a vicarious action of the right hemisphere."[1] He believes that the theory of vicarious activity of the cortex in the immediate vicinity of the damaged third frontal gyrus (or the so-called Monakow's extended region of Broca) is far less plausible than the theory that the third right frontal convolution acts as a substitute in case of destruction of the third left frontal. To further support his contention, he goes on to say that "if both the left and the right frontal gyri are destroyed speech disappears completely and forever."[2]

Fifty years prior to Henschen's research Jackson postulated that the right side of the brain functioned in emotional speech. In 1920 Henschen asserted that the entire structure of aphasia would disintegrate if we could not utilize the right side of the brain in our explanation of facts in aphasia.

For comprehension of spoken language he concludes that "the right side fails entirely in about 30 per cent of cases; it performs to some extent but imperfectly in about 60 per

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1. Schwalback, op. cit.

2. Loc. cit.

cent."[3] In the few cases which show a remarkable degree of recovery, the right temporal lobe takes over the function very well. He admits his inability to explain satisfactorily the variability of recovery.

The disturbance of the faculty of speech as shown in echolalia, paraphasia, senseless reading, etc., Henschen attributes to the performance of the right cerebral hemisphere. His cases showed the inferiority of the right half in being unable to carry on the highest function of reading.

Some of Henschen's conclusions of interest in this study follow:

1. A lesion in the left third frontal convolution results in motor aphasia, or, in the term Henschen prefers, "aphemia".
2. When in addition to the left third frontal convolution, the neighboring parts of the precentral gyrus are affected, the patient will show dysarthria as well as aphemia.
3. The transverse gyrus is the hearing center; the first temporal convolution the center for the hearing and understanding of speech. With bilateral destruction of the first temporal, there is permanent word-deafness; with destruction only on the left in the right handed there may be partial recovery through the activity of the right.
4. Lesion of the angular gyrus produces a second form of sensory aphasia, word-blindness. The right angular gyrus, which may take over some of the function of the left, is a very imperfect substitute.
5. The left third frontal convolution in front of Broca's area is the important region for singing, but the right third frontal may suffice if the left is injured. The left temporal pole is probably the important area

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3. J. M. Nielsen, "Function of the Minor (Usually Right) Cerebral Hemisphere in Language", Bulletin of the Los Angeles Neurological Society, 3:67, 1938.

> for the hearing and comprehension of music, but activity on the right can often compensate for destruction on the left.[4]

On the basis of the extent of J. M. Henschen's collection and analysis of the available postmortem literature, Nielsen states that "his views must have more weight than those of any other one man."[5] Harry A. Teitelbaum[6], on the other hand, suggests that in analyzing Henschen's data the student take cognizance of

> the many discrepancies that prevent precise correlation between clinical symptomatology and post-mortem findings, and, the existence of the various disturbances in higher cortical functions in all sorts of combinations, thus permitting the interpretation that these disturbances are interdependent to a great degree, rather than each being the direct effect of focal impairment of cortical tissue.[7]

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4. Weisenburg and McBride, op. cit., p. 30-1.

5. J. M. Nielsen, op. cit., p. 68.

6. (Harry Allen Teitelbaum:--Baltimore neurologist and psychiatrist, 1907-____)

7. Harry A. Teitelbaum, "The Principle of Primary and Associated Disturbances of the Higher Cortical Functions as Applied to Temporal Lobe Lesions," The Journal of Nervous and Mental Disease, 96:265, July-Dec., 1942.

CHAPTER VII

S. A. KINNIER WILSON'S ECLECTIC ANALYSIS AND OTHER RECENT WRITINGS

In 1926 a monograph on aphasia appeared under the authorship of S. A. Kinnier Wilson[1] of England. He advocated striving for an anatomico-physiologic nomenclature. He found himself unable to accept a psychologic terminology. Yet he also warned against divorcing the psychological component from its anatomico-physiological counterpart. He insisted on a separation of anatomic, physiologic and psychologic classifications of terms. His contention was that all three types of approach are necessary, but that the terms should not be used interchangeably. To clarify,

> it is the confusion of the spheres of thought and avenues of approach in the past which has led to the drawing of a psychologic concept center, an anatomic foot of the third frontal convolution, and a physiologic writing center all on the same diagram of a cerebral cortex, and the drawing of lines from one to the other as though association fibers connected them and impulses passed along these lines.[2]

Wilson believes speech to be an intellectual process behind the expression of which lies thought. Hence, he considers it unwise to attempt to distinguish speech disorders

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1. (Samuel Alex. Kinnier Wilson:--English neurologist, 1877-1937)

2. J. M. Nielsen, Agnosia, Apraxia, Aphasia. (New York: Paul B. Hoeber, Inc., 1947), p. 13.

from intellectual disorders. He warns against the psychologist's tendency to ignore "the patiently acquired facts of cerebral localization of function."[3] Attention is called to the evidence from the work of the Swiss School (von Monakow, Berze, Frankhauser, etc.) that "certain layers of the cortex, considered in their whole extent, constitute the anatomico-physiological basis for certain forms of psychical activity."[4]

Physiologically, many aphasics experience an inability to use certain motor cells of the cortex--those innervating mouth, lips, tongue and palate--for speech, while still capable of other functions such as chewing, swallowing, etc. It would appear foolhardy to localize the physiological defect in the cell groups concerned. Theoretically, therefore,

> apart from clinical and pathological considerations, disorders of speech must be disorders, physiologically, of higher mechanisms which play on those of a lower physiological level.[5]

Wilson's chief contribution to the subject is his insistence that an aphasic symptom-complex be looked at in three ways:

> What is the anatomical site of the lesion producing the defect? What are the physiological mechanisms involved? What is the nature of the psychological disorder? No study of the subject can make any pretense at completeness

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3. S. A. Kinnier Wilson, "An Introduction to the Study of Aphasia", The Lancet, 201:2 for 1921:1143, Dec. 3, 1921.

4. Loc. cit.

5. Ibid., p. 1144.

> if any of these three aspects is ignored, nor, on the other hand, can it be harmonized with clinical data, if anyone is elaborated at the expense of the others.[6]

In recent years another outstanding study of aphasia has been completed by Theodore Weisenburg and Katharine McBride. Their study, presented in the publication "Aphasia: A Clinical and Psychological Study", was more concerned with mental functioning from the psychologist's viewpoint than with therapeutic measures to be used for specific speech problems.

The authors contributed another system of classification--a four part grouping of aphasic manifestations including the following types: predominantly expressive, predominantly receptive, expressive-receptive, and amnesic.

While the study contributed little to the theoretical consideration of localization or retraining, it does have merit--according to Jeanette Anderson--in being a "careful study and presentation of the functioning of neural tissue and the nature of the loss and return of function without, in most cases, specific re-training."[7]

Another recent publication is Louis Granich's "Aphasia: A Guide to Retraining". As its title implies it is essentially an aid to therapy, rather than an inquiry into the nature of aphasic disturbances.

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6. Ibid., p. 1143.

7. Anderson, op. cit., p. 35.

PART III

CONTEMPORARY VIEWPOINTS

NOTE

Chapters VIII, IX and X are condensations of the respective authorities' writings which seemed most applicable to this study. They are digests, in the authors' own words, of their published thoughts pertinent to concepts of aphasia. The articles drawn upon are listed in the Bibliography under the author's name. With the exception of one article, the contents of which were first published in 1930, all the source material for these three chapters dates from 1938 on.

The chapter contents were sent to the individual authors for editing and approval. The revised chapters, herewith presented, are the endorsed statements of Nielsen, Goldstein and Anderson. They have, therefore, the added merit of being current and of being endorsed by the authorities to whom they are attributed.

** The author regrets to note that as of the date of the binding of this thesis, Miss Jeanette Anderson's edited copy of her viewpoints had not been received.

CHAPTER VIII

THE NEUROLOGIST DISCUSSES APHASIA

by J. M. Nielsen **

All efforts to base the study of aphasia purely on anatomic structures have failed because of insufficient knowledge. Physiology without anatomy to support it will always fail to provide localizing data as will a purely psychologic study. We must grant that in the present state of our knowledge of aphasia all three methods of approach must be simultaneously considered: psychological, physiological and anatomical. It is impossible for any single approach to be successful.

Until 1947, except for the few results of electrical stimulation of speech centers by Foerster the science of aphasia rested on clinico-pathologic material. Penfield has now outlined some of the speech areas by electrical stimulation. The defects in clinico-pathologic material are partly due to incomplete studies of the clinical manifestations, partly to the poor general condition of patients with cerebral vascular lesions, and partly to incomplete study of autopsy specimens. But, the greatest difficulty encountered by

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** See Note on p. 71.

students of the subject is the fact that nature does not often produce discrete lesions so placed as to make the case a good experiment.

For the sake of a coherent discourse, let us begin with the premise that language is symbolization which is used to exchange thoughts with one's peers.

The human brain has developed in its cortex (probably with its cortex) a highly complex system of language symbolization, the anatomical structures of which are neurons with cell bodies situated in the cerebral cortex, the complex function of which when comprehended by the student is called physiology, and when conceived functionally but not anatomically is called psychology. Various portions of the cerebral cortex are variously constructed and have different functions. The different areas have been elaborately studied and mapped by Brodmann, Campbell, Elliot Smith, the Vogts, Economo and Koskinas, and by Bailey, and physiologically by Foerster and Bailey and his co-workers. Certain of these areas of the cortex may be rendered functionless by natural experiment, and the resultant disturbance of function may be recognized as the direct result of such localized destruction. By correlation of certain functional defects with certain specific anatomic defects, signs of agnosia, apraxia, and aphasia may be valuable in cerebral localization. Thus it comes about that a patient who has been able to read and is rendered incapable of reading

while still relatively unimpaired in other functions of symbolization is known to have a lesion in the major (usually left) angular gyrus.

One must be careful to say "relatively" unimpaired, because there is always some degree of impairment of general cerebral symbolic and other functions when the defects make possible a diagnosis of agnosia or aphasia. This is obvious, as the deleted portion of the brain is part of the area used in general symbolic mentation, and the structure as a whole does not function so well after the deletion of a part as it did before that deletion.

Language is a highly complex cerebral function requiring for its use one side of the brain behind area 6 of Brodmann. The fact that the portion of the brain anterior to area 6 is not essential to language is shown by cases of bilateral prefrontal lobectomy.

Areas of cortex having apparently certain specific functions have been called centers. There is frequent reference to centers in the literature in the anatomic sense, the physiologic sense, and in the psychologic sense. What we are concerned with here is the definition of a center in the clinico-pathologic (anatomico-physiologic) sense. From this viewpoint, a center is a cortical area the functional removal of which causes a definite deficiency syndrome which can be recognized clinically. Such a center is not a functionally

independent area devoted to one certain function exclusively as it is often conceived of. It is an area essential to normal performance of a certain function. It may be an area where a certain group of impulses pass or where synapses occur in the ordinary process of certain functions. This leaves the inevitable conclusion that although an aphasic disturbance may seem to be either more sensory or more motor in character, it can be neither of these fundamentally. Rather, it is associational.

In ordinary speech one hears what one has already said, "checks up" on it, analyzes it, and corrects it; and this often causes one to change one's diction or even one's phrases in the course of a sentence. The portion of the brain which thus holds the thought and checks up on the speech to determine whether the speech mechanism properly expresses the concept is the attention or the concept center. It would seem that the reasons for the impossibility of placing the concept center in the diagram are that the center is constituted of much of the cortex and that we know practically nothing of the multitudinous paths traversed in the elaboration of a thought.

Speech is a mental process. Differences of opinion center about the degree of divisibility of the psyche for various elements of speech. Certain writers have taken the stand that there is a disturbance of the entire psyche in

every case of aphasia. They have correctly pointed out, for example, that in every case of motor aphasia the patient does not comprehend spoken language as well as the normal person does and that in every case of acoustic verbal agnosia the patient fails to speak spontaneously as well as the normal individual. These statements are true. How, indeed, can the brain function as well after the functional deletion of a portion as it could before? On the other hand, there are innumerable cases on record to show that except for the specific loss in the various types of aphasia, as the great diminution of spontaneous speech in motor aphasia, forgetting of names in amnesic aphasia, loss of comprehension of spoken words in acoustic verbal agnosia, or loss of recognition of written words in visual verbal agnosia, the remainder of the psyche is often comparatively little affected. The general loss which is seen in every case seems to correspond to the form well named by Head--semantic aphasia. We grant, then, that there is an element of semantic aphasia in nearly every case.

What physiologic and anatomic data do we have with which to work? The first transverse gyri of Heschl, one on each side, are the sites for perception of sounds. An area immediately adjacent to these (para-transversal area), in the superior temporal convolution (Wernicke's area) is the site essential for recognizing memory pictures of the sounds of

words.

To consider the comprehension of sounds of words, associations must be taken into account--not merely the sound itself. The associations are composed of other auditory memories, of visual memories, kinesthetic memories, and usually many other types. This means, then, that before the significance of the word can be determined, the greater portion of the cortex must be consulted, i.e., impulses must travel over it. In other words, there cannot be such a center as a word-meaning center.

In Broca's convolution are engrams laid down by efforts to make the movements necessary for production of vocal sounds. When these engrams or the neurons which constitute them are destroyed, the patient still knows what he wishes to say, i.e., he still has clear thoughts; he can still hear the sounds silently in his head, but he has forgotten how to make the movements necessary to produce the sounds. But as the area of Broca with its association neurons to and from the center of Wernicke are part of the mechanism of speech, there is imperfect comprehension. He can still move all the muscles for chewing, clearing his throat, expectorating, etc.; it is only movements to make sounds constituting symbols of speech that he has forgotten. This is apraxia, a very specific apraxia. Motor aphasia is then a type of apraxia. But since it pertains to language and symbols, it is by definition a

type of aphasia.

A review will convince any unbiased observer that Broca's convolution contains patterns of motor speech, i.e., engrams (neurograms), which constitute the anatomic basis of memory of how to articulate words. Penfield has now shown that electrical stimulation of that small gyrus, while the patient is speaking, prevents him from uttering words.

Just how Broca's area of the major side receives its impulse to speak is not entirely clear. It certainly cannot alone initiate impulses. Even when Broca's area on the left side is destroyed the patient can still say something. Jackson noted that emotional language was not curbed in aphasia. His idea (1878) that expletives and similar emotional expressions are produced by the right side of the brain has gained great support from the ideas of Moxon and the corroboration by Liepmann (1906) that the left side of the brain takes the lead and hence that the right may not be entirely passive in speech. Since subsequent evidence has borne out Jackson's contention, he can be credited with reaching an anatomic fact through a psychologic approach. Zollinger's patient with the entire left cerebral hemisphere (pallium) removed had ability to say a few words. The impulses must have originated on the minor side. I have had numerous cases proving that the right Broca's convolution performs after the left is destroyed.

Nomenclature is the chief hindrance to one's trying to

bring order to the vast accretion of chaos found in the literature of agnosia, apraxia, and aphasia. Wilson (1926) points out the desirability of having an anatomic basis but at the same time the impossibility of this at the time of writing. A physiologic basis is also impractical. A psychologic basis is easy to obtain, especially as Head (1923) has already presented one, but a nomenclature based on psychology dissolves all connection between cerebral anatomy and pathology and the subjects to be classified. I believe the acceptance of such a nosology would be too destructive to justify itself. I have adopted a terminology which is so far as possible based upon usage, which avoids the coinage of new elemental terms but attempts a systematization on the basis of physiologic (functional) hierarchies, or levels of integration.

To clarify this last expression somewhat, attention is directed to the obvious facts that, physiologically speaking, the first cerebral activity in the reception of an impulse is the function of primary perception. For vision the anatomic site of perception is on the borders of the calcarine fissure, for audition, in the first transverse gyri of Heschl. So far as language is concerned, the first element of cerebral function takes place mainly at these two sites. This, then, is the first functional hierarchy.

If we continue to follow the paths physiologically, the

next step is, for visual impressions of language, the formation of engrams of symbols seen in the region of the left angular gyrus (cortex), for hearing, the formation of engrams of sounds heard in the region of the posterior superior left temporal convolution. This is the second functional hierarchy. So far, we have localization of cerebral function. Still speaking physiologically, above this, though there are higher hierarchies of function, there is very little, if any, localization known as yet. There are at least three hierarchies in the sensory sphere: perception, formation of engrams for recognition, and higher elaboration. This higher elaboration is not all on the same level, at least two levels being definitely discernible, significance and recall.

While this division of aphasias into those on a lower level and those on a higher level has not to my knowledge been done before it seems to me particularly appropriate. Reflection will show that, while agnosias are disturbances of recognition, all of the aphasias--above the level of agnosia--so far described are disturbances of recall, or to use a better term, of reminiscence. The fundamental difference between recognition and reminiscence is that whenever one recognizes an object or a symbol one has the object or symbol before one; one receives a visual, auditory, tactile, gustatory, or olfactory impression of it and this impression is compared with engrams previously formed by similar impressions. If the two

are essentially identical the object or symbol is recognized. The crux of the matter is that one starts with the object or the symbol and looks for its meaning. But in reminiscence one starts with the idea or concept and searches one's cortical engrams of memory for the corresponding object engram or the symbol engram. The two processes are reversed.

The same process (of starting with the concept) applies to motor speech; one starts with an idea and looks for the motor pattern with which to express the concept. The motor pattern is found through the medium of the language formulation area in which the synthesis of concept into speech symbols is carried out and from which the motor patterns are stimulated.

There is a still higher hierarchy of language, however, higher than that of reminiscence. This is the hierarchy of semantics and calculation. Head's most valuable contribution to the subject of aphasia was the introduction of the term semantic aphasia. By it he meant one's inability to determine the full significance of speech though able to comprehend spoken or written language to some extent. In the study of cerebral localization in aphasia it is useful to draw a rather sharp line between recognition and comprehension because there are all degrees of comprehension. A physician comprehends much more of a medical article than does an attorney, but the reverse is true of a legal document. It is question-

able whether anyone ever gathers the full significance of what an author writes and we must thus all have semantic aphasia if we use the old terminology. If, however, we draw the line between recognition of a word and its significance we have a valuable distinction. Loss of the first is agnosia, of the second, aphasia. Significance is still not reminiscence; it is a higher function because one can recall a word of which the significance is unknown.

It is customary to speak of the two cerebral hemispheres as being dominant and recessive, respectively. The terms are based on the conception that the one ordinarily takes the lead and leaves little or nothing for the other to do in the associative sphere of activity. The basal ganglia, the motor and sensory projection fiber systems, and the primary perception areas for the special senses are commonly exempt from inclusion in the concept of unilateral cerebral dominance.

While there is a hereditary determinant to make a certain hemisphere the major one, there is no essential difference between the functional capacity of major and minor hemispheres at birth. This has been shown repeatedly by instances of severe trauma to the major hemisphere early in life. In such cases the minor hemisphere invariably takes over the function of the major with such precision and dexterity that observers do not notice any disturbance of language. Handedness does not develop until the age of about 9 months to a

year. Even when it begins to manifest itself the superiority of the major does not appear great. This is proved by the fact that injury to the major hemisphere at the age of 4 or 5 years causes only transient aphasia. Whenever a minor hemisphere is called upon to take over a function it at first fatigues with astonishing rapidity. As it becomes trained it develops endurance rather than specific capacity.

The question of unilateral dominance in the sphere of language is intimately related to handedness but the question of handedness is extremely complex. Handedness and brained-ness are not necessarily contra-lateral. Instances have been reported in which persons who considered themselves right-handed have become aphasic as a result of right cerebral lesion. These occurrences have been ascribed to conversion of the patients from left- to right-handedness so early in life that they themselves were unaware of it. This explanation does not hold in the cases equally well established in which strongly left-handed persons have developed aphasia from a lesion of the left hemisphere. When one in addition considers that, regardless of handedness, children who suffer a severe cerebral lesion early in life never develop aphasia at all, or recover in a remarkably short time, it becomes clear that both sides are almost equally capable of assuming any function of language if compelled to do so. There is apparently no way to determine absolutely whether a given person is left-

or right-brained for a given function except to study the case after a unilateral cerebral lesion and determine the laterality of it by some sign not pertaining to language.

While there is undoubtedly an hereditary tendency to unilateral dominance in general and some of the cases of right cerebral dominance in language in right handed persons can certainly be explained on the basis of stockhandedness as outlined by Foster Kennedy, there are certain important elements relative to ontogenetic development of dominance which must be considered. A child learns during the first year to recognize objects and geometric relations in its environment long before visual-language associations are established. In these matters the child may develop dominance of one occipital lobe but there seems to be no reason for developing this on the left any more than on the right side. If by chance the right occipital lobe becomes dominant for non-language functions before the right or left temporal lobes does so in comprehension of auditory language, the occipital patterns may not shift laterality of dominance; it may be easier to have the right occipital lobe dominant over the left and send all necessary impulses across the corpus callosum to the left temporal lobe (indirectly) than to suppress the right occipital patterns and develop similar ones on the left side. This is the explanation here offered for the occurrence of crossed occipital-temporal dominance. If by

chance the laterality of dominance is the same for the occipital and temporal lobes there is no problem or question or retraining an occipital lobe.

It is suggested that crossed temporo-occipital dominance (major temporal lobe on the left and major occipital lobe on the right) may be due to establishment of occipital dominance on the right before temporal dominance (dependent on language) becomes established, i.e., during the first year of life. In some cases the occipital dominance is formed on the left at first; in others it shifts to the left with establishment of language dominance; in some nature finds it more economical to leave the right occipital lobe dominant and shunt impulses across the corpus callosum rather than to establish engrams in the left occipital lobe to replace those already well-established in the right.

Right hemispheres travel through life with the left until an accident occurs to the leader. Some learn to understand; fewer learn to speak, still fewer learn to read, and only an occasional one learns to write.

In the evaluation of a given case of aphasia all this must be taken into account. The ability of a patient to speak does not prove the integrity of the left frontal lobe, nor to comprehend, the preservation of the left temporal lobe. But the loss of a specific function proves destruction of the areas concerned. What a patient can do with language after a

lesion of the major side depends on the inherent ability of the right hemisphere. Hughlings Jackson was entirely correct in his views; proof has come after sixty years.

An analysis of cases places the clinical manifestations following lobectomy (i.e., the performance of the minor (right) temporal lobe) beyond the realm of chance and proves that a person without any left temporal lobe is in general far superior to one with a slightly damaged lobe still in situ.

Necessary surgical operations in the last few decades, more particularly in the last decade, have also given us cases which prove crucial points. One of the best is the removal of the entire pallium of the major side. This operation showed that the minor side can, to some degree, perform the function of language.

Inasmuch as both cerebral hemispheres have a function in language but that one is far more capable than the other the two may be designated as major and minor. If cases of destruction of the entire major language area are gathered for study we can readily tell what capacity the minor hemisphere possesses. Destruction of the area supplied by the sylvian artery is equivalent to hemispherectomy so far as language is concerned because the occipital and frontal extremities have no function in language.

"How complete must the defect be to constitute agnosia or aphasia?" If, following a lesion of the convolution of

Broca, a person is able to speak but with stammering and hesitancy, some students might say that he does not have Broca's aphasia. But we know that from the standpoint of cerebral localization he does have a lesion and that the reason for his being able to talk at all is either that the crippled area is performing or the imperfectly trained minor convolution of Broca is doing the work which we observe. We should therefore say that the patient does have aphasia (apraxia of speech). If the patient, after such a lesion, speaks as well as he did before he either had two equally capable convolutions of Broca or it was the minor one which was destroyed, the minor one merely having been located on the side where we expected the major one to be. Our misinterpretation is due to the erroneous assumption that handedness is an infallible guide to brainedness.

A word of caution is offered regarding the diagnosis of aphasia after acute cerebral injury. One cannot study aphasia resulting from cerebral trauma until general cerebration has been recovered and only specific defects remain. It is necessary, therefore, to await recovery from the acute effects of trauma before attempting the study of aphasia.

In testing the elements of speech, the functions which should be tested separately and specifically are:

1. Motor speech (spontaneous speech and repetition).
2. Comprehension of spoken language (questions such

as "Hold up two fingers; Put your finger on top of your head; Put your hand on your right ear; Touch your index fingers together."

3. Ability of the patient to find the words he wishes and to make orderly sentence.

4. Reading. Note is made of ability to recognize and ability to comprehend significance of written and printed matter.

5. Writing. Spontaneous writing of something other than the patient's own name, the ability to copy from print to handwriting should be tested.

If these five tests are carefully interpreted the site of the lesion is usually determined.

1. If the patient is unable to speak spontaneously except for a few simple words or phrases and if that defect is his only aphasic manifestation, it may safely be concluded that the trauma has involved the convolution of Broca or the external capsule between the insula and the putamen (major side). A subcortical hemorrhage is just as potent as a cortical contusion in causing motor aphasia. The reason that a lesion of the external capsule can cause motor aphasia is that the convolution of Broca functions merely in coordination of the muscles of speech for enunciation of words. It does not contain the engrams of ideas to express or engrams of the organization of words into grammar. Such functions are

performed in the (posteriorly located) language formulation area, chiefly area 37 of Brodmann, and Broca's convolution cannot perform its function unless supplied with organized language for it to emit. The pathway from the area 37 to Broca's convolution is via the external capsule.

2. Comprehension of spoken language is mediated through Wernicke's area (the para-transversal portion of the superior temporal convolution of the major side). The function of recognition is performed by a relatively small cortical area but comprehension necessitates coordinated function of a larger portion of the temporal lobe in the direction of area 37. Hence rapid fatigue of comprehension of spoken language indicates that the trauma has affected the major temporal lobe.

3. "Word-finding", i.e., the ability of a person to find in his mental index any appropriate word corresponding to an object or to an abstract idea, requires coordinated function of the language formulation area and Wernicke's area. Hence a lesion of the posterior portion of the temporal lobe will lead to "amnesic aphasia". That symptom means, therefore, a lesion of the temporal lobe. The patient says, "I know it but I can't say it."

4. Reading may be disturbed in several ways. If the patient is unable to recognize written or printed words which formerly were familiar to him the angular gyrus is destroyed or its connections with the occipital or the temporal lobe are severed. If he is merely unable to determine the signi-

ficance of what he can read the lesion may be anywhere in the general parieto-temporal region. If he is able to write but is unable to read what he has written there is a subcortical lesion of the major angular gyrus. This fact is based on the necessity of the angular gyrus cortex for the function of writing and the capacity for a sub-cortical angular gyrus lesion to prevent occipital (visual) impulses from reaching the cortex of the angular gyrus.

5. Writing is so complex a function that a localizing value must not be ascribed to agraphia unless the agraphia is isolated. When agraphia is the only defect of language present in a given case there are still two possible sites which the lesion may occupy. One is at the writing center in the frontal lobe, the other is at the border between the angular gyrus and the occipital lobe of the major side.

The examiner must never lose sight of his general knowledge of cerebral physiology. For example, a lesion of the temporal isthmus (area between the posterior extremity of the insula and the posterior horn of the lateral ventricle) completely disrupts all language function by separating the entire temporal lobe from the other language centers. The patient in such a case cannot speak, cannot comprehend spoken language, cannot read or write. This is the "global aphasia" of the older writers.

If an anatomical area subserving a functional unit of

language is destroyed it never recovers. Recovery of the function depends on training of the homologous area of the other hemisphere. When a language center is irrecoverably destroyed the prognosis depends entirely on the ability of the patient to train the corresponding area on the minor side. Prognostic factors of importance are then: the age of the patient (young persons may train the opposite side in a week), the general health, the intellectual capacity and the incentive.

It is an aphorism that one can operate practically with impunity in the language areas of a child below six years, with little damage to the function of speech between the ages of 6 and 15, with recovery in a few months between 15 and 20 but with a progressively higher incidence of prolonged troubl e later in life. Of course, there are surprising exceptions, cases late in life in which recovery is rapid due either to ambidexterity or to unusual natural resourcefulness and dynamism.

CHAPTER IX

THE PSYCHOLOGIST'S CONCEPT OF APHASIA

by Kurt Goldstein **

My concept of the function of the organism was based on analysis of a great number of physiologic and psychologic phenomena--normal and pathologic--in man. I came to the conclusion that the basic motive of organismic life is the trend of the organism to actualize itself, its "nature," its capacities, as well as possible. I have tried to formulate the rules which govern behavior of the normal organism and guarantee self-actualization.

Applying my point of view to the study of the nature of language and its disturbances, I came to the following conclusion: Language is a means of the individual to come to terms with the outer world and to realize himself. Hence, the aphasic patient tries to achieve a condition which allows him to react as well as possible to the tasks arising from the environment. If he is successful in this endeavor, at least to such a degree that he can fulfill those performances which are "essential" to his nature, he will be in a new order, will avoid catastrophic occurrences, and be able to use his remaining capacities. From this point of view, it follows that every individual speech-performance is under-

** See Page 71.

standable only from the aspect of its relation to the function of the total organism in its endeavor to realize itself as much as possible in the given situation.

How does a definite lesion modify the function of the brain so that a definite symptom comes to the fore? We are by no means justified in inferring directly from a correlation between a localized defect and a defect in performance a relationship between the concerned area and a definite performance corresponding to the defect. The facts allow only localization of defects, but not a localization of performances.

We are confronted with two realms of facts which we try to bring into relation: anatomic findings and observation of disturbed performances. If we survey the findings of microscopic studies of the cerebral cortex we are confronted with differences of structure which surely hold a special significance for different functions. We can assume with a fair degree of certainty that there are areas which are designed to receive stimuli from the outer world, and others to mediate motor performances. It remains unsettled how these cortical areas, which I called the periphery of the cortex, do function. Analysis of the defects in lesions of this part does not at all allow us to bring a circumscribed defect into relation to a circumscribed lesion, still less to localize a definite performance in a definite group of cells, etc.

The so-called classic theory of localization is based mainly on the material gained from postmortems. It should be observed that the objections against the theory stem first from a more careful consideration of the pathologic-anatomic data. There are the so-called negative cases: on the one hand, absence of symptoms in a lesion affecting an area which was considered characteristic of this locality; on the other hand, appearance of symptoms without the presence of a correspondingly localized lesion. A critical consideration of these numerous cases shows that they are inexplicable if one considers the symptoms as simply depending upon locality and extension of the lesion.

A lesion of a special locality in different cases may differ very much regarding the degree to which the substratum in general is affected, and particularly its different strata. Such a selective character of the process may be of paramount significance for the development of symptoms. It is very difficult, indeed, to evaluate the degree of damage; it is not only dependent on the direct destruction of the nerve cells but also on the condition of the glia, blood vessels, etc. Further, we have no idea of the relationship between a definite anatomic condition and a specific performance. We are far from being able to decide whether the preserved tissue is still functioning sufficiently to allow for a certain performance or not. We have no definite criteria for this

decision. We are facing here a methodologic difficulty which seems scarcely surmountable.

Whether certain symptoms will appear or not on account of a local injury certainly depends on many factors other than locality: i.e., on the nature of the disease process, on the damage of all or only some structures of the cortex, on the condition of the rest of the brain, on individual differences in cooperation of both hemispheres, on the state of circulation in general, on the functional reactions of the organism to the defect, on the psycho-physical constitution of the personality, etc.

The symptoms which are related to a localized lesion cannot be understood from the destruction of so-called residuals of previous performances; they cannot be understood simply as due to memory defects. Each performance is due to the function of the total organism in which the brain plays a particular role. In each performance, the whole cortex is in activity, but the excitation in the cortex is not the same throughout. There is a definite configuration in which one part of the cortex is in that particular excitation which corresponds to the "figure," while the rest is in that excitation which corresponds to the "background". Distribution in this configuration varies depending on whether motor activity or perception or a mental attitude is requested. A particular locality in the brain matter is characterized

by the influence which the structure of this locality exercises on the total process, by the contribution of the excitation of this locality to this process--as effect of its particular structure. Thus, for instance, the area striata contributes to this process something which is necessary for the experience of vision, the frontal lobes, something which is the presupposition for the mental phenomenon we call abstract attitude, etc., but, to reiterate, we may not localize corresponding functions in these parts of the brain.

To each performance corresponds an excitation of definite structure in the cortex, indeed, not in a circumscribed area but widespread over the whole cortex, differently in each performance. This is what we should term localization. To restate, localization may be defined as a definite distribution of excitation within a structurally differentiated nervous system, with a particular structuration of the excitation in a definite area.

Even if the entire cortex participates, certain well known speech areas stand forth as special performers on the ever-present background. The play of figure (or the activity of a special center) and background (or the general cortex) is variable and always changing, hence it is that a patient will show a certain constellation of aphasic symptoms under one set of circumstances, whereas a few hours later some

differences in the responses will be noted.

The various symptoms of aphasia are the manifestations of a single functional disorder, loss of the ability to grasp the essential nature of a process. This is equivalent to the Gestaltstorung or the inability to differentiate the "figure" and the "ground".

I think it is correct to assume that to each mental performance there corresponds a particularly constructed substratum in the brain, that functions in a definite way. The nervous mechanisms are, so to speak, predisposed. They develop during the growth of the individual and gain a particular organization by experience. They extend from the peripheral sense organs to the substrata of the most complicated mental performances. If such a substratum is damaged in its functioning, the organization as a whole may be maintained, the mechanism losing only some of these functions acquired by training. In such cases it may be possible and appropriate to retrain the functional mechanism by the same means by which it was built up in childhood. Hence it may be possible to acquire again the lost performances. This happens also in spontaneous recovery, where the apparatus is retrained by the demands of the environment.

If after thorough analysis of the symptoms, one is convinced that the brain matter is damaged to such a degree that some former functions are irreversibly lost, it will be necessary to proceed in another way. We shall have to help

the patient to build up compensatory procedures in the same performance field.

In order to better understand the various symptoms, I have devised a classification of four general types of symptoms. The first general type of symptom represents direct sequelae of impairment of the substratum and consists of defects of performance. (These are the negative symptoms of Hughlings Jackson). All direct damage causes a rise of the threshold and retardation of excitation. The receptivity of the patient is reduced. It takes him much longer to react. Excitation of a damaged field requires an abnormal amount of energy. If excitation takes place despite the obstacles, it spreads abnormally and lasts an abnormally long time. The effects of the lesion are generalized because the increased energy output for the reeducated performance of the destroyed function diverts the added energy from channels normally subserving other modes of behavior. A word grasped by an aphasic with great difficulty sticks and influences subsequent performances, perseverates. A further characteristic effect of the damage is that performances of the organism are determined to a much greater extent than normally by external factors. As a result of loss of structure, patients are deprived of former experiences; thus external stimuli acquire an abnormal importance.

A second category of symptoms is of those due to

separation of an undamaged area from a damaged one. (Here we are dealing with the positive symptoms of Jackson). It is reasonable to assume that in the normal nervous system all parts function in interrelation with the whole, or at least with more extensive fields. Performances in a given field are therefore also determined by the functioning in other fields. Loss of certain performances, through damage of their substrata, brings about the modification of other depending performances.

A third group of symptoms is due to the effect which the pathological process in the damaged area exerts on other parts of the nervous system, for instance by irritation of the immediate neighborhood.

The fourth group of symptoms are expressions of the tendency of the organism to avoid catastrophic conditions. In situations of success, the total behavior is typified by an ordered condition. The person is steady, interested, cooperative, more at ease. In situations of failure, however, the person looks definitely dazed, changes color, becomes agitated, starts to fumble, his pulse becomes irregular, he is sullen, evasive, exhibits temper, or even becomes aggresive. This catastrophic condition prevents the patient not only from doing the required task which exceeds his impaired capacity, but also from doing performances which he is able to carry out in the ordered state. The sick organism has a

strong urge to meet all demands as well as possible; its existence is bound up with such an endeavor even to a greater degree than in the case of the healthy organism. In order to avoid catastrophes the patient resorts to: self-exclusion from the world, i.e., loss of consciousness; he seeks tranquility, avoids company; he tries to avoid anything unexpected; he seeks to surround himself with a protective fence that will prevent impinging stimuli from entering. He may build such a fence by everlastingly doing something. Concentration upon a particular activity makes him relatively impervious to the undesired and dreaded stimulation from outside. When he is confronted with a task he cannot cope with he will stick to the performances he is able to fulfill. Thus he appears abnormally fixated and rigid. A particularly interesting and important phenomenon resulting from this tendency to avoid catastrophies is excessive orderliness. Everything in the patient's environment must have a definite place. Behind this formal geometry there is quite a utilitarian motive, namely bringing each object within the patient's reach with a minimum of effort in his part. He gets excited if somebody changes the arrangement.

Through this description of aphasic symptoms one can see more clearly how a definite abnormality is an expression of the struggle of the particular individual with his defect and his attempt to perform as well as possible the tasks arising from the environment.

There is one group of patients where the difficulty in finding words is mainly an expression of a personality change, an expression of impairment of "abstract attitude". To clarify this, it is necessary to understand my theory of the types of language--concrete and abstract--and their corresponding aphasic symptom complexes.

Concrete language, which belongs to concrete behavior, consists of speech automatisms, of the "instrumentalities of speech": of sounds, words, series of words, sentences, one form of naming and of understanding of language in familiar situations for which it has been conditioned, and finally of emotional utterances.

Abstract language belongs to abstract attitude: volutional, propositional, rational language. It is disturbed somewhat isolatedly if abstract attitude is impaired; further, in slight damage of instrumentalities which may leave intact other speech functions but disturbs the highly complex performance of voluntary actions.

It would seem more correct to speak of disturbance of language belonging to the abstract attitude and of language belonging to concrete behavior, than of rational and emotional language, and to consider emotional language a particular form of concrete language.

Not all patients with brain injury have impairment of abstraction. Whether it is present or not depends on the localization and severity of the injury. It is particularly

prominent in frontal lobe lesions, even when the lesion is relatively small and circumscribed, but it can occur also in other localizations.

Nomenclature in the field of aphasia is somewhat confused as a result of the use of different points of view from which the symptoms are considered. Disturbances of speech are biologic phenomena. As such they can be considered from two different aspects: as modifications of performances of the patient, or as modification of the anatomic structures, and their modified functioning (the anatomico-physiologic aspect). The anatomico-physiologic approach usually appeared in the form of localization of separate performances and their disturbances in circumscribed areas of the brain cortex. As I have explained, the results of this approach are very ambiguous in general.

A nomenclature based on an analysis of the characteristic picture of performance disturbances promises more success; this means analysis by psychologic means. Opposition to this approach was raised long ago. Men like Wilson and Nielsen are not much in favor of it. But this opposition overlooks the fact that all clinical distinctions of aphasic symptom complexes are finally based on psychologic analysis.

In this state of affairs, our nomenclature cannot be very homogeneous. Sometimes we may base it on anatomic findings, sometimes on psychologic analysis, sometimes on

clinical experience. The main point is that the nomenclature should allow for such a clear characterization of symptom complexes that the names can be used for comparison and correlation. Thus, the following list of terms is considered only as a means of orientation.

Disturbances of the expressive side of language due to cortical lesions:
1. Dysarthria.
2. Peripheral motor aphasia (cortical and subcortical aphasia of the old nomenclature, Broca's aphasia, pure aphasia)
3. Central motor aphasia: Disintegration of motor speech due to impairment of abstract attitude and the function of motor instrumentalities

Disturbances of the receptive side of language due to cortical lesions:
1. Cortical deafness: disturbance of acoustic perception due to cortical lesion
2. Noise and music cortical deafness: Disturbance of perception of the characteristic sounds of noises and music with maintenance of hearing
3. Sensory aphasia
   a. Peripheral sensory aphasia (pure sensory aphasia, "reine Worttaubheit")
   b. Central sensory aphasia (cortical sensory aphasia, combination of pure sensory aphasia and central aphasia, and more or less amnesic aphasia)

Central aphasia

Amnesic aphasia

Transcortical aphasias, characterized through better preservation of repetition than understanding and spontaneous speaking
1. Transcortical motor symptom complexes
2. Transcortical sensory symptom complexes
3. Mixed transcortical aphasia

(PLUS: the various agraphias and alexias with which this study is not concerned)

Due to various interpretations in the past of some of these identical terms, certain of them need clarification.

Motor aphasia, as I interpret it, is that difficulty in

uttering sounds and words which is due to a defect of the learned specialized motor speech performances, i.e., due to a defect of definite motor "Gestalten," or of the function by which these motor complexes are built. By this we distinguish motor aphasia from apraxia of the speech muscles.

We distinguish now, primarily on the basis of Broca's observations: the foot (pars opercularis frontalis), the pars triangularis (designated as "cap" by Broca) and the pars orbitalis. Later investigators have sometimes used synonymously the expressions foot and posterior third of the third frontal convolution.

For a long time, the foot of the third frontal convolution was assumed to be the place lesioned in cases of motor aphasia. Ferrier added the neighboring parts of the Insula and the pars opercularis centralis, other investigators also the pars triangularis and orbitalis. Later, particularly after the critical discussion of von Monakow, the crucial area was extended in anterior and caudal direction. Monakow's delineation of Broca's area includes: the third frontal convolution, the anterior part of the Island of Reil, the small gyrus connecting the third frontal with the operculum of the precentral convolution, and the operculum itself.

In order to understand the variety of syndromes observed in lesions of the extended area of Broca we have to consider the relationship of the third frontal convolution to the

operculum Rolandi and to other parts of the brain. From such a survey, we come to the following conclusion:

Without any doubt there are in the operculum Rolandi the foci for the musculature of mouth, tongue and larynx. The injury to this substratum produces impairment of the use of these muscles with regard to non-linguistic as well as linguistic activities, e.g., sound formation. We assume that the more complicated a given motor activity, the larger the area of its representation: in the case of language activity it includes the third frontal convolution. Indeed, this is probably a very essential part, since in cases of lesions exclusively in the third frontal convolution, with intact operculum Rolandi, severe motor aphasia has been observed, occasionally even of permanent nature. This can hardly be explained by assuming that the focus in the third frontal convolution causes the aphasic symptoms be affecting secondarily the neighboring operculum Rolandi, which is free of primary defect.

There are numerous anatomic components involved in the structure of motor speech and, in any given case, we have the greatest difficulty in attempting to determine the significance of the various defects for the impairment of motor speech, for restitution or absence of restitution; similar lesions can produce very different symptoms and we can only speculate about the reasons therefor.

Central motor aphasia is a combination of motor speech disturbances similar to peripheral motor aphasia plus an expression of impairment of abstract attitude. In central motor aphasia, the lesion is located in the anterior part of the brain, damaging the frontal lobe and the motor speech area, and thus producing a picture in which, besides impairment of abstraction, motor speech phenomena are prominent.

On the receptive side, understanding of heard language is based on perception of sounds and sound complexes. Sensory aphasia exists if sound and sound complexes are perceived in a normal way but are not recognized as language, or, if this is the case, are not comprehended in their meaning. Three forms of sensory aphasia have been distinguished: pure sensory aphasia, where sounds are not recognized as language. The patient is not able to understand or repeat them. There are no other speech disturbances; particularly speaking, reading and wording are intact (also called pure word deafness, subcortical sensory aphasia, peripheral sensory aphasia). A second type, sensory aphasia proper (Cortical sensory aphasia, Wernicke's sensory aphasia, central sensory aphasia), is a complex picture where perception of sounds as language is somewhat preserved, but not so well that the patient would be able adequately to recognize the heard language--he cannot perceive the heard words in order to recognize what they mean. This sensory defect is accompanied by other speech defects,

particularly paraphasia. In a third form, the so-called transcortical sensory aphasia, perception of language is perfect, but the words do not evoke the right meaning. The patient is able to repeat heard language, but does not understand the heard word. Repetition is more or less echolalic.

My concept of the organization of the temporal lobe in respect to speech is not so much in contrast to that of Henschen as it may appear at first. He assumes that we have to distinguish three parts: one center for word hearing, one for perception of word sounds, and a word sense center. This corresponds to my dedifferentiation of a smaller part of the area sufficient to guarantee hearing in general, a larger area to guarantee hearing of word sounds and another part which belongs to the area for "inner speech". Indeed, I do not assume separate areas but more or less complicated functions of a great region.

Apart from the significance of this result for understanding this special form of aphasia, it is of general importance for the theory of brain function:

1. In so far as it brings an aphasic picture which was often considered as effect of interruption of pathways into relation with a damage of cortical function, and thereby considerably undermines the theory that destruction of pathways can produce aphasia--a theory which I have always rejected.

2. In so far as it makes the assumption of acoustic perceptions unnecessary and considers the perceptive speech phenomena only as complicated phenomena of these. This brings us to a standpoint similar to the interpretation of motor aphasia which is, for our interpretation, also the effect of impairment of more complicated motor activities which do not differ essentially from the other voluntary movements.

In central aphasia we find damage of inner speech. Inner speech is the totality of processes and experiences which occur when we are going to express our thoughts, etc., in external speech and when we perceive heard sounds as language.

In addition to these functions, inner speech consists also of material fixed more or less by previous functioning of the "apparatus," by experience. The concepts of letters, words, phrases, are more or less fixed wholes, patterns which we are aware of in the framework of an inner speech attitude and used as wholes to start the speaking activity. These patterns are developed to a different degree in different individuals. They can be impaired to a greater or less degree in dedifferentiation of inner speech. In this field, dedifferentiation follows also the laws of impairment of figure and ground configuration valid for pathology in general.

To summarize our ideas of central aphasia, it is a symptom complex which consists mainly of: (1) impairment of spontaneous speech and understanding, the latter less than the first; (2) literal and verbal paraphasia; (3) paralexia and particularly paragraphia; (4) disturbance of repetition; (5) disturbance of spelling and of the capacity to combine letters into words. To be noted as not infrequent complications are amnesic aphasia and signs of pure acousticaphasia.

The outstanding symptom of amnesic aphasia is the lack of nouns, adjectives, verbs and especially names for concrete objects in speech. The defect shows most strikingly in the task to name objects; the patient is unable to find the names for the most ordinary objects of everyday life. He need not present any additional disturbance of speech; at least others need not become manifest immediately. He repeats words without hesitating, he accepts among a number of words presented orally or written, only the one which belongs to the object; he shows no paraphasia, no disturbances in writing and reading.

We have explained before that difficulty in finding words can be due to different causes. Only those cases with word finding difficulties where this defect is related to impairment of abstract attitude should be called amnesic aphasia. Thus, symptoms due to impairment of abstract attitude belong intrinsically to this form of aphasia.

Anatomically, if amnesic aphasia occurs in a localized affection, the latter usually concerns the temporo-parietal region; but it must be of a particular kind, it must not damage this region too much, otherwise disturbances of instrumentalities of speech and of the mental phenomena are so predominant that amnesic aphasia does not come clearly to the fore. A fine damage in this region which is suited to produce a fine diffuse damage of the cortical function, can take place particularly in tumor or abscess in this region--the two conditions in which particularly amnesic aphasia appears. I stressed such an origin of this symptom complex years ago. Impairment of abstraction appears in lesion of various parts of the brain, in lesions of the frontal and parietal lobe, and in both cases we observe the defect in naming. However, it seems that the clearest development of symptom complex occurs if the lesion is located in the temporo-parietal region; this would mean if the speech area were affected to a certain degree too. One may argue that with this statement I give up my theory of the origin of amnesic aphasia as due to impairment of abstract attitude. Not at all. Impairment of abstract attitude remains, to my experience, the prerequisite without which the defect in "naming" never occurs. A lesion of the "speech area" alone is never followed by the amnesic-aphasic defect in word-finding.

The main characteristic of <u>transcortical aphasia</u> is damaged understanding, much more than spontaneous speech.

The patients speak much, they usually show some reduction of words, particularly lack of nouns, probably due to a concomitantly existing amnesic aphasia. If one subtracts this latter defect, the language appears correct, meaningful, the syntactical and grammatical structure quite well organized. Some paraphasia may be present, probably as effect of a slight damage of the central speech area.

Special difficulties and uncertainties regarding restitution are based on a lack of knowledge regarding the relation of the "other" hemisphere to the functions of the active hemisphere. This problem demands special consideration, since the assumption of restitution by means of the corresponding area of the other hemisphere is extensively recognized. The main difficulty with this conception is that in a given case it is impossible to know how much the "inactive" hemisphere had not always been cofunctioning, especially in a restored function. In a patient in whom this was the case, one should not speak of restitution, but rather of the taking over of the entire function by a part of the appropriate apparatus, since it is not a question of the taking over of a function by a substrate that formerly was not engaged with this function.

It must also be denied that one part of the brain can substitute for another part, that latent material can take over the function of destroyed tissue and that the corres-

ponding opposite hemisphere can replace the damaged one. A true return of function takes place only by the reestablishment of the substrate or, under definite, rare and limited circumstances, through painstaking reeducation with the assistance of the remaining part of the substrate which had already been of service in the function. The acquired functions are usually incomplete, especially in adults.

We could assume a real taking over of a speech function, a new learning by the other hemisphere only under the following conditions: (1) If the patient had a definite premorbid prevalence of one hemisphere (that allows us, within definite limits, to assume that the other hemisphere did not act together with the "leading" one); (2) If we can assume a total destruction of the leading hemisphere, or at least of that part which we bring into relation to a certain performance; (3) If the symptomatologic defect was of a longer duration and recurrence of the performance took place slowly, possibly under the influence of definite training.

In general, one can say that adaptation to an irreparable defect can occur in two ways. The organism can yield to the defect; it restricts its performances to the preserved reduced ones, but the normal functioning of the organism is in principle unchanged. It is the more natural procedure, seems more automatic, demands less voluntary activity on the part of the individual, and hence secures more security than

the second way in which the organism may react to a defect. Here the organism gives up the old procedure; because its maintenance would give too little effect, it tries to compensate by other performances. This represents a more volitional kind of behavior, leads more readily to fluctuation, involves less constancy and less security and admits a greater possibility for catastrophic situations.

Restitution or real return of former function takes place only when complete anatomic restitution takes place. If complete anatomic restitution does not take place, there is no return of function, but repair. Repair is recognized by a number of characteristics. The following are always present: (a) A greater breaking up occurs, corresponding to the damage in the function; (b) the functions are changed in a qualitative way; the reactions in a definite milieu are more constant and the stimuli more bound; in changeable stimuli there is a greater inconstancy of the reaction than in the normal; (c) rest and orderliness of control are easier than in the normal, and more readily disturbed. An orderliness in qualitative normal relation in this sense is observed only by a narrowing of the milieu, in finding an adequate milieu for the disturbance. There the orderliness and rest are even greater than in the normal.

The restitution picture depends on: (1) the total capacity of the organism before the disease set in (the

amount of former ability and especially of the functional quantity); (2) the greater or lesser degree of inviolability that the entire organism can withstand (circulation of the blood, general condition of strength, etc.), especially in regard to the brain; (3) the energy present for the use of a function, which as a result of the relative constancy of the total energy is dependent also on the use of energy in other places; this results in the retraining of certain functions at the cost of others. The selection is determined by the tendency of the organism to maintain those functions which, from the standpoint of the total task of the organism (its nature), are the most important, and, among the most important ones, those best capable of accomplishing the given objectives of the surrounding world. The favorable result of restoration is therefore dependent on the milieu in which the particular organism is living, as well as on the demands being made. A defect is the more restored the more the organism is capable of reaching its surrounding world with the restitution.

From the foregoing considerations one learns a definite therapeutic procedure, which must be considered as a general rule: Never attempt to improve a definite disturbance in itself; its importance must always be considered as a task for the entire organism, for the corresponding nature of the organism, the more so as the nature is altered by the illness,

and the therapeutic measures are to be determined from such considerations. An attempt is to be made to bring the patient into a situation in which an adequate milieu may develop for the changed organism; in which the organism, regardless of the defect when compared with its former being, may perform its most useful functions, and in which it can work as regularly as possible without the intercurrence of too severely catastrophic reactions. When this is accomplished, the organism, although not normal, is none the less not sick. Subjectively it feels well, and objectively it shows little disturbance. This condition appears as the goal in the process of restitution; it must also form the object of any therapeutic attempt. From this it follows as the most important negative problem that one should not proceed therapeutically with certain symptoms, because an organism, thus functionally more able, will "recover better" than when attempts are made to control all symptoms. The task is therefore to determine which defect is to be controlled and which is not. The scientific considerations of the manifestations, however, are not sufficient for such a decision; a comprehension of personality in its essence and a willingness to enter into the personality of the patient are essential here.

CHAPTER X

THE SPEECH PATHOLOGIST'S VIEWPOINT OF APHASIA

by Jeanette O. Anderson **

In formulating a basic concept of aphasia, the speech pathologist is compelled to consider in parallel the psychological, physiological and anatomical aspects of the problem. A study of aphasia made from any one specific point of view is incomplete. This perhaps explains why there seems to be no universally satisfactory classification of aphasias.

The speech pathologist can follow the psychologist in his concept of aphasia as a disorder of propositionizing or of symbolic formulation, but he cannot agree with him that there is little or no localization of function. He can say with the anatomist that certain cerebral areas tend to subserve specific language function, but he cannot subscribe to exact anatomical localization. In general, he can follow the lead of the physiologist in admitting that certain areas subserve certain linguistic functions more than others and that the entire brain is needed for normal linguistic activity.

The speech pathologist's concept of aphasia should be not only as precise as that used in localization by the surgeon; it must also be as general as that of the psychologist who insists that the linguistic mechanism is a Gestalt

** See Note on p. 71.

and functions wholly. More than any other investigator in this field, the speech pathologist must see both the parts and the whole clearly and simultaneously in their mutual relationships.

In order to discover aphasic manifestations and to prescribe techniques of special re-education in speech, the speech pathologist needs to consider not only three aspects of the problem, but a fourth as well: the possibility of retraining in terms of all three. Although concerned with the receptive and expressive formulation of linguistic symbols, he is even more interested in the restitution of function than in gross or specific losses or even remnants of linguistic ability. It is not enough for him to realize the extent of the damage, to have an idea of functioning in both physiological and psychological terms; he must plan to use the uninjured neural tissue and to reroute language activities over, to and through this sound brain-stuff.

A basic concept of aphasia for the speech pathologist evolves: aphasia is a linguistic impairment; there tend not to be "pure" aphasias; each aphasic patient exhibits a disturbance made up of certain aphasic manifestations peculiar to the given case; any classification is of aphasic manifestations and not of aphasias. Examination reveals the manifestations; they form the specific aphasic picture seen in each case. Once he has this concept of the inconstancy,

unpredictability and irresolution of the aphasic problem, the trained speech pathologist is ready to examine and to attempt re-education of aphasic patients.

The speech pathologist is confronted with two tasks in work with aphasics: (1) to examine patients carefully and diagnose speech impairments accurately, and (2) to prescribe and administer suitable therapeutic measures for each case. The examination and diagnosis is fundamentally important in that localization of brain lesions is often made on the basis of detailed language observation in conjunction with other neurological and clinical findings; and therapeutic measures prove more efficient if we know at once the exact abilities and disabilities of the patient.

The following classification of aphasic manifestations is suggested as a first step toward a practical solution of the problem confronting the speech pathologist:

I. Expressive manifestations
  A. Implicit (probably caused by a parietal lesion)
    1. Ideational aphasia
    2. Ideo-kinetic aphasia
  B. Overt (probably caused by a frontal lobe lesion)
    1. Spoken language
      a. Broca's aphasia (third left frontal convolution, cortex)
      b. Subcortical aphasia (subcortical Broca's region)
      c. Transcortical aphasia (questionable)
    2. Written language
      a. Agraphia (Exner's writing center, second left frontal convolution
II. Receptive manifestations
  A. Auditory (superior temporal convolution)
    1. Agnosia (Heschl's convolution)
    2. Aphasia (Posterior part of superior temporal convolution)

B. Visual (temporo-parieto-occipital area)
   1. Agnosia
      a. For linguistic symbols
      b. For objects and colors
      c. Geometric-optic
   2. Aphasia
      a. Alexia
      b. Agraphia
      c. Anomia
C. Other sensory media
   1. Tactile
   2. Cutaneous
   3. Kinesthetic

III. Associative manifestations
   A. Aphasia on lower levels
   B. Aphasia on higher levels

IV. Combinations of manifestations
   A. Wernicke's aphasia
   B. Total aphasia
   C. Other combinations of aphasic manifestations

V. Manifestations indicating special abilities useful in planning speech therapy
   A. Expressive
      1. Oral facility for building new motor synergies
      2. Manual facility for building new motor synergies
      3. Kinesthetic imagery
   B. Receptive
      1. Visual imagery
      2. Auditory
         a. Imagery
         b. Memory span
      3. Other sensory imagery
   C. Assotional
   D. Constitutional predisposition of patient

Although its fundamental purpose is to guide the speech pathologist in diagnostic, prognostic and therapeutic procedures, this classification may prove of some use in the consideration of aphasic patients by other specialists. It is realized that any classifications used are merely arbitrary; repeated examination seems to reveal few cases that can be grouped under any system of classification without further explanation.

Neither the aphasic child nor the aphasic adult profits from speech correction until he wants so much to express his inner verbalizations that he spontaneously attempts some form of overt communication with other organisms, human or not, in his environment. This effort to speak may be made orally, manually, grimacingly, graphically, or chirographically. Once the aphasic tries to communicate, he will respond to speech training or retraining.

There can be no one testing procedure set up for determining the presence of aphasia; procedure must be adapted to each patient. On the basis of language findings combined with clinical findings, surprisingly accurate localizations of intra-cranial pathology may be made; however, we need to take into consideration the fact that pressure and edema affect language centers quickly and that the causative lesion or neoplasm may be some distance from the centers affected. This means, of course, that language findings alone are not sufficient basis for diagnosis. They do help, however, and in the study of aphasic patients we have found an excellent opportunity for the speech pathologist to work in close co-operation with the neurologist. This pre-therapeutic work is invaluable to the speech scientist in planning his reeducational procedures and techniques. In our experience, neurologists welcome the testing of language functions that we may do in conjunction with their work and the post-operative or

post-traumatic retraining that we may help to effect.

Diagnostic techniques include testing receptive abilities (obeying oral commands, following written instructions, etc), checking writing and copying abilities, and testing expressive abilities (producing spontaneous speech and repetitive ability). In addition, tests for aphasias on higher levels than those of simple recognition, comprehension and expression are necessary in many cases. These would include mathematical calculations, following complex instructions, giving opposites in word lists, etc. In testing, the examiner should always record relative speech of response, emotional stability or lability, perseverations, following of instructions, use of objects, alertness, spontaneous speech, as well as how the patient reacts to the testing.

With no identical cases and therefore no comparable test results, we cannot treat our findings statistically and say that a given correlation indicates the presence of aphasia and that anything less does not; the pattern that constituted aphasia for one individual need not point in that direction for another person of lesser intelligence and/or training. We are compelled to treat each case separately; the thorough case-study methods recommended by Adolph Meyer in psychiatric practice are adaptable to aphasic patients.

Perhaps the most important factor to keep in mind is Backus' restatement of Yacorzynski's tenet: "the process of

rehabilitation must itself be part of the reality of living for the patient. He will not wait to live while those powers of implicit and explicit verbalization are growing again in the months of re-education."

The adult aphasic patient suffers an emotional aberration that interferes with what language abilities he has. This breakdown of morale tends to inhibit language in an individual who is adult in all except linguistic functions and who is able, moreover, to compare his present linguistic failures with his past achievements.

After the first few lesson periods, retraining in speech seems to contribute to increased emotional stability in these aphasic patients. Morale-building is a fundamental part of speech therapy with aphasic patients. It is one of the jobs of the therapist to make the patient willing to start his linguistic rehabilitation where a child starts and to see his linguistic successes and failures in their proper perspective.

In the initial stages of training, the aphasic child learns well when objects, toys, pictures, and situations are associated with speech sounds. The adult, on the other hand, usually responds better to written or printed symbols than to the realities for which they stand. Only when situations, objects, persons, colors, or pictures are fraught with emotional significance do they provoke an attempt at speech

as rapidly as do printed or written symbols.

Although it may be necessary at first to employ strongly emotional stimuli to secure overt response from a patient, this practice is to be avoided and discouraged because it leads in the aphasic, even more than in the so-called normal, to noncortical language, to uncontrolled speech, to excesses of emotion that eventually inhibit rather than stimulate volutional speech, to hysteria, to behavior on a thalamic level. It is important to guard an aphasic from emotional excesses. He is no longer able to guard himself very effectively. He has to relearn cortical control not only of speech but of all his overt behavior.

Concomitant sensory stimuli and corollary motor activities are helpful in the retraining process. The simultaneous strengthening of many pathways seems to reinforce each of them and to contribute to those of speech. The aphasic will speak and/or write a word more quickly if he hears it and sees it while he tries to write it and say it than if only one stimulus were presented and only one response requested. There are at least two reasons for this: first, each stimulus and response tends to reinforce others made simultaneously; second, if one avenue of reception or expression is blocked, anatomically or psychologically, concomitant stimuli and responses provide alternate routes by which necessary neurological associations may be effected.

To effect these associations, neural impulses must be rerouted to their usual association areas through healthy tissue; remaining tissue of the dominant hemisphere must associate impulses previously synapsing elsewhere; or the intact, usually nondominant, cerebral hemisphere must take over the function of damaged brain areas. This is the theory upon which neurologists and psychologists explain restitution of linguistic function.

Ontogenetic development of speech seemed to recur in restitution of linguistic activities and can be utilized by the speech therapist. The last four stages, babbling, lallation, echolalia and verbal utterance, in the child's normal speech development can be used effectively as a therapeutic device in teaching aphasic children and in retraining aphasic adults.

In the teaching of aphasic children, speech ought to be an outgrowth of daily experience. For adults, speech should never be taught as an entity, an end in itself, but always as an integral part of the business of living, a means to an end. It cannot be separated from emotional, social, and vocational retraining. Only as a patient achieves personal, social, and economic stability will his speech stabilize. He cannot be taught speech while he waits to live. He has to go on living and his speech training will be useful to him only as it keeps pace with his needs.

The success of re-training in speech seems proportional to: (1) active cooperation of the patient; (2) former training of the patient; (3) success in re-building morale in connection with linguistic problems; and (4) resolution of economic, personal and social problems attributable to the patient's illness and residual disabilities.

Formal re-education in speech for mature aphasic patients seems valuable for at least five or six years; however, plateaus are reached in relearning long before the regaining of former levels of excellence. Further training seems not to aid the patient in going beyond such plateaus but does appear to aid materially in the maintenance of re-learned linguistic skills.

CHAPTER XI

DIAGNOSTIC AND THERAPEUTIC APPROACHES OF BACKUS, SHEEHAN, AND OTHERS

With what sort of person are we dealing when an aphasic appears in the speech clinic for diagnosis and therapy? Usually, in addition to the obvious speech deficiency, three major complications will be present: paralysis, personality change, and emotional instability. With every attempt to speak, the patient has experienced mental confusion and, ultimately, fatigue. The frustrations experienced through continually futile speech attempts bring about despondency, resignation, apathy. Mentally he is capable of comparing his speech failures with past accomplishments. The resultant breakdown of morale tends to cause the patient to retreat more and more within himself.

> Add to these things the actual personality changes, the tension, the need for rest and sleep, the anxieties, (the contrary euphoric state), the concern about and yet lack of understanding of his condition, the almost complete lack of a sense of humor, and the hundred and one additional almost freakish neurological defects which may be involved in individual cases, and you have a picture of the state of confusion which is typical of the aphasic.[1]

This brief picture of the patient should be sufficient to point out the great needs beyond formal speech training.

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1. Vivian Mowat Sheehan, "Rehabilitation of Aphasics in an Army Hospital", The Journal of Speech Disorders, 11:2: 150, 1946.

Along with the regaining of verbal adequacy, the patient is in dire need of personality adjustment. "Speech is an integral part of the whole individual. It cannot be taught as a mechanical process apart from the complex behavior pattern to which it belongs."[2] As Kurt Goldstein points out, its restitution must be considered as a task for the entire organism. It is the speech clinic that must meet the socio-psychological needs of the patient. Yet, zealous therapists must realize that "speech re-training can be instituted successfully only after there is some spontaneous attempt at communication through overt expression, or otherwise."[3]

Preparatory to the speech examination, the therapist need s to know the patient's general medical condition and the neurologist's diagnosis of whether the cerebral condition is static or progressive. Entering into the prognosis of recovery are many factors: the general health of the patient, the site and severity of the cerebral injury, the condition of the brain as a whole, the patient's age, his previous type of mental and linguistic habits, his intelligence, educational and cultural status, the type and amount of impairment of motor skills, as well as the patient's motivation--his emotional drives, determination, and stability.

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2. Joseph Jastak, "The Treatment of Expressive Aphasia in a War Veteran", Delaware State Medical Journal, 17:5:104, May, 1945.

3. Anderson, op. cit., p. 168.

The techniques of examination have had almost as wide a variance as the number of examiners. The methods of inquiry have ranged from

> (a) the brief, informal clinical approach of the conventional neurological examination, and (b) the relatively systematic, though unstandardized, clinical enquiry recommended by Nielsen, to (c) use of formal tests with prepared materials as employed, for example, by Head and by Weisenburg and McBride.[4]

Because of the many different conceptions of the nature of aphasic disturbances, there is little uniformity in the tests employed. The therapist is faced with the dilemma that "tests are bound to differ as long as theoretical differences exist, and on the other hand, theoretical differences must exist and must even appear greater as long as the tests lack uniformity."[5]

Regardless of the method of testing to be used, we must determine what the patient can do, what he cannot do, and what things he needs most to meet his immediate social and economic demands. The actual diagnosis should result in the establishment of immediate and reasonable sub-goals which the patient can understand, accept, attain, and appreciate. Goals of this type will permit the successes necessary for further motivation.

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4. Ward C. Halstead and Joseph M. Wepman, "The Halstead-Wepman Aphasia Screening Test," The Journal of Speech and Hearing Disorders, 14:1:11, March, 1949.

5. Weisenburg and McBride, op. cit., p. 92.

To ascertain the abilities and disabilities of the individual case it is necessary to make an analysis of the following spheres: speech, reading, calculation, time sense, drawing, comprehension of pictures, vision, visual imagery, auditory performance, auditory imagination, tactile sense, tactile imagery, the imagination of movements of his own body, and measurements of reaction times.[6]

In order to "determine the functional mental capacity, to estimate loss of capacity by comparison with previously recorded test scores, and to measure improvement as training progresses,"[7] the following tests are recommended by the U.S. War Department for the use of clinical psychologists in the testing of aphasics: the A.G.C.T. (retest) or Basic Battery for permitting comparisons, the Wechsler-Bellevue Intelligence Score for the determination of amount of mental deterioration and loss of mental efficiency, and the Goldstein-Scheerer Cube Test of Abstract and Concrete Behavior to ascertain the impairment of abstract thinking or ability to generalize.

The modified Head serial test #1 used by many therapists includes the following activities: naming objects seen, repeating single words, understanding the spoken word, reading

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6. Konrad Zucker, "An Analysis of Disturbed Function in Aphasia", Brain, 57:109-27, 1934.

7. War Department Technical Bulletin (TB MED 155), "Aphasic Language Disorders", War Department, Washington 25, D.C., April, 1945, p. 2.

the printed word aloud, understanding the printed word, writing spontaneously, writing from dictation, copying, spelling aloud. Daily testing in these activities permits a graphic recording which allows comparisons in self improvement as well as comparison in improvement with others.

There are three types of training facilities available for aphasic adults: individual treatment supervised by a speech therapist and carried out by a tutor, nurse, or member of the patient's family, the out-patient clinic where the patient reports for short training periods several times a week, and the in-patient training center. This is usually a hospital where the patient's training extends over many hours a day. For the training of the aphasic child, Alice G. Rooney recommends the school for the deaf. Here, as in no other service of an educational system, "endless associated repetitions are obligatory in order to strengthen the weakened faculty of association between the cortical senses that should function integrally."[8] In the school for the deaf the child patient can have a thorough sense training program in the development of muscular tone and in the cultivation of sight, touch, and hearing, in addition to training in reading and speech.

Since the brain-injured represent problems in every

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8. Alice G. Rooney, "An Aphasic Child in a School for the Deaf", The Volta Review, 47:559, October 1945.

phase of living, every agency available should be utilized--the doctor, neuro-surgeon, psychologist, psychiatrist, social worker, physio-therapist, speech therapist, educational agencies, vocational counselors, and members of the patient's family.

Of the techniques basic to speech re-education Dr. Backus lists voluntary inhibition (to stop trying so hard and eliminate random facial movements and sound), voluntary relaxation, sense training (lip reading, hearing, kinesthesia), rhythmic training, and shifting of handedness, in addition to formalized speech re-training. Shift of handedness is advised except when a shift was made earlier in life, or in cases of confused dominance, or paralysis of both sides. The shift will help develop function in the association areas of the undamaged hemisphere and can be aided through such activities as throwing a ball, playing ping pong, writing on blackboards, and eating with the opposite hand.

In most of the pedagogic methods the utilization of the five senses is stressed in aiding the retention of speech symbols. Thus, the patient is more apt to recall a word if while re-learning it he is exposed to the auditory symbol simultaneously with being able to see the object named, feel it, and in some cases, smell and taste it. This technique plus constant repetition of material aids greatly the regaining of speech. Gestures and pantomime will also enable

faster learning since "speech is a total bodily response and accompanies motor activity."[9]

In aphasic re-education Backus stresses the fact that "the impulse for speech grows out of group activity in real life situations."[10] Because of this she advocates an intensive program of from five to seven hours daily utilizing a great deal of group rehabilitation. Vivian Mowat Sheehan, another enthusiastic advocate of the benefits of group therapy, finds that "the contact with others has a therapeutic value in itself. It lessens self-pity and shame."[11] In actual therapy she conducts work on greetings and farewells, identification (names, home towns, age, names and values of coins and currency, names of days, months, etc.), special holidays, directions, parts of the body, numbers, the alphabet, weather, members of the patient's family, furniture, food, shaving, smoking, maps, automobiles, meeting people, telling time, naming colors, playing cards, sports and games, animals, etc.

Among the specific techniques utilized by Mrs. Sheehan is that of training in conversing. She trains patients in asking questions by using such common question words as how,

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9. Ollie Backus, et. al., op. cit.,

10. Backus, op. cit.

11. Sheehan, op. cit., p. 151.

when, what, etc. Telling little incidents, describing people, indicating locations, and keeping chronological order (speaking on topics involving time sequence) are among other conversational devices. Additional help is given in varying responses, listening to others converse, telling something in a crowded room, interrupting politely, maintaining eye contact, and using contractions.

Her "small talk" helps give the aphasic a degree of certainty in the use of a practiced pattern or recurrent phrase. This can be used in giving introductions, paying compliments, thanking the hostess, asking a girl for a date, commenting on the weather, greeting friends and strangers, begging pardon, and telephoning. The use of these phrases will automatically help put the patient at ease, enable him to save face, and make further and more difficult learning easier.

Work under Mrs. Sheehan's guidance is also done in attempting to eliminate verbal crutches such as pet phrases, over-used gestures, and substitutions. Avoidances are detected and the patient is then prepared forthe difficult situations which he is avoiding and sent deliberately into them. Nor is the patient allowed to let others say something for him. In this event, the therapist insists on the patient's repeating any word said for him.

Special attention is given to using the "little words"

such as to, is, but, or, was, on, in, from, out, etc. Assignments are made to use these words three times a day.

Use of the above devices has indicated that aphasic training can be accomplished without the use of fatiguing drills and other monotonous activities. Mrs. Sheehan further advises therapists to

> keep assignments exact and specific, limited to a simple task which can be completed. The clinician must be wary of the assignment which sets such high standards of performance that the aphasic is doomed to failure. . . He must work toward specific and well-defined sub-goals of achievement which he can reach within a reasonably short period of time. Let general improvement be a by-product instead of an ever-elusive goal.[12]

The therapist must be continually on guard to encourage and maintain realistic levels of aspiration.

Summarizing her methods for the re-training of aphasics, Mrs. Sheehan lists "stating the goals specifically in terms of immediate needs, specialized techniques, using questions and answers, class discussion, the assignment method, and individualized group therapy."[13]

Since many aphasic patients have greatest difficulty in regaining those parts of speech concerned with qualifying and correlating (adjectives, adverbs, prepositions, articles and conjunctions), Charles K. Mills advocated the use of primers

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12. Vivian Mowat Sheehan, "Techniques in the Management of Aphasics," The Journal of Speech and Hearing Disorders, 13:246, Sept., 1948.

13. Loc. cit.

and grammars in retraining--especially with patients who have had a good educational background. He recommended that

> patients should be taught the grammar as a child is instructed, in other words, by teaching him the meaning of the different parts of speech and the exact methods of using them in phrases and sentences.[14]

Also

> the patient with the book before him should be taught to conjugate verbs, decline nouns and pronouns, compare adjectives, and in other ways to go through the routine methods of studying language employed in the schools, these being modified by the particular requirements of the case.[15]

Above all, he warns against a method of simple memorization. He contends that the patients must relearn all the parts of speech and how they are used in the construction of a sentence to express thought.

Augmenting the speech therapist's techniques, attempts have been made to aid treatment medically through narcosynthesis. Although this technique has not undergone any long-term rehabilitation programs to the writer's knowledge, it can be noted that in the cases exposed to this treatment, the patient relaxed and found themselves able to speak more easily and confidently after injection with a solution of sodium amytal. The elation over their success carried over

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14. Charles K. Mills, "Treatment of Aphasia by Training", The Journal of the American Medical Association, 43:26: 1945, December 24, 1904.

15. Loc. cit.

and they continued to improve after the drug wore off. The experimenters attributed the immediate improvement displayed by postulating the removal of the inhibiting effect of anxiety.

> The individual with organic brain disease is aware that he no longer possesses his original mastery of the environment. He responds to this awareness with anxiety and feelings of personal inadequacy. He reacts to new situations by withdrawal, to avoid the anxiety engendered by failure.[16]

Regardless of the techniques utilized in aphasic training, one general principle stands out as mandatory: the aphasic patient must be surrounded by an atmosphere of cheerfullness and confidence. The therapist must at all times exhibit a calm, interested, and understanding attitude. All indications of hurry, irritation, or disappointment in progress must be absent. The therapist must continually help the patient to understand his difficulty. Sheehan uses

> drawings of the brain, simple explanations, stories of others' eventual success despite their losses. We are busy offering encouragement, getting rid of false notions, and overcoming disillusionment. We build a cheerful working atmosphere with plenty of informality and freedom from tension. We try to develop a sense of humor through teasing, telling jokes, pulling little pranks, etc. We try to help the men look forward to the future with a feeling of pleasant anticipation rather than dread.[17]

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16. Louis Linn and Martin H. Stein, "Sodium Amytal in Treatment of Aphasia; Preliminary Report", The Bulletin of the U.S. Army Medical Department, 5:6:707, June, 1946.

17. Vivian Mowat Sheehan, "Rehabilitation of Aphasics in an Army Hospital," The Journal of Speech Disorders, 11:2: 155-6, 1946.

The very hope of success does much to combat the depression to which these patients are so susceptible. Being kept busy with occupational therapy helps to occupy time which might otherwise be spent brooding.

Keeping the patient cheerful is of prime importance. This can be aided by group work and group singing. Developing a sense of security will be helped by keeping the patient productively busy. He has a need for consistent routine with all needless confusion avoided. Being stimulated to take an interest in other things and people is another factor of great importance in the patient's rehabilitation.

To be most successful, rehabilitation must be adapted to the patient's special interests and abilities. In this respect the patient's family can be helpful in supplying information relative to the patient's early preference for handedness in writing, educational background and achievement, previous employment, interests and hobbies, friends and associates, family, temperament and disposition, likes and dislikes, attitudes, personal habits and peculiarities, etc.[18]

The patient's family must be fully instructed in the nature of the disorder so they do not hide the patient from others who might think him insane or mentally deficient. They should be redirected in any false displays of affection

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18. Backus, op. cit.

manifesting themselves in keeping the patient home and idle and administering to his every wish. They must have impressed upon them the importance of treating the patient as a normal person and not as a handicapped dependent. They must conceal any feelings of worry or repulsion they may entertain. They should help him to realize his maximum potentialities socially and vocationally by enabling him to have the best of specialized training--not only surgical and medical, but rehabilitation in speech as well.

Of supreme importance in therapy through family co-operation is the need to convince members of the patient's family that "they will be a greater source of inspiration to him and will be happier themselves about him if they turn from past thoughts of what he used to be to the reality of the present with its hopes for what he can become."[19]

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19. Ibid., p. 47.

CHAPTER XII

A LAST WORD

A retrospection of this study must necessarily include an explanation of its omissions. The authorities primarily treated have been those which seemed, to the writer, at the time of his writing, to epitomize the thoughts of the researchers in his particular field of approach--be it anatomical, physiological, psychological, neurological, etc. Availability of material also accounts for various omissions and minimized treatment of certain authorities. Of necessity, there has been omission of reference to hundreds of men who have directed, influenced, and inspired the thoughts of their more articulate disciples.

In one circumscribed respect, the completed work represents a compilation of what Henry Head felt was deleterious to further progress in understanding aphasia--namely, the evolved hypotheses of the various authorities, while their clinical investigations have been neglected. Head noted that

> the world clings to theories, for they are easier to remember, can be reproduced with effect and lead to a clarity of exposition foreign to a description of the crude experimental facts. There is in consequence a tendency to carry over the conceptions of one age on to the observations of the next.[1]

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1. Henry Head, Aphasia and Kindred Disorders of Speech, (Cambridge: The University Press, 1926), p. 134-5.

However, despite this viewpoint in which there is a good deal of truth, a compilation of clinical facts would be of little value without some sort of synthesis. Since much of the world autopsy literature has been collected and synthesized by far more qualified authorities than this writer, it was the purpose of this study to present the main trends in thought as evolved from the observed facts. It is this task which the compiler hopes has been in some measure accomplished.

Reviewing, then, the material in hand, we find at the beginning of the nineteenth century, the idea of physiologic uniformity of the entire brain. All parts were thought to serve the same function; injury to any portion would cause another portion to take over its function.

Into a field of medicine imbued with this belief, Franz Joseph Gall introduced the concept of localization. Certain faculties (moral and intellectual qualities) were described as localized in definite areas. Control of movements of speech was attributed to a cerebral center located in the frontal lobes. These were our first ideas of anatomic localization.

With Pierre Paul Broca's localization of the speech center in the third frontal convolution of the left hemisphere, the anatomic viewpoint gained great impetus and was accepted by nearly everyone exposed to it.

This type of localization was shortly supplanted by

localization in terms of motor and sensory areas of the brain and their inter-connections. Sensations, images and perceptions were thought to lie dormant in definite areas awaiting excitation. Using the hypothesis that we think in words, Bastian noted that "words were revived as sound impressions in the auditory perceptive centres, whence they were transmitted to the more immediate motor centers for speech."[2]

Over a period of time Bastian's views began to deflect from their original adherence to strict anatomical localization of function. However, he apparently pointed out the path which led to the period of the "diagram makers."

The increasing amount of attention given to individual differences during this period demanded some deviation from the strict concept of rigidly marked cortical areas. The explanation was supplied by Charcot and his "auditives", "visuals" and "motors" for speech. Thus the use, in varying degree, of the different centers during the learning process offered an explanation of differences which did not conflict with the localization.

From this stand it was but a minor step to emphasizing the importance of the association pathways in explaining individual differences. Slowly the use of the word "localization" was changing from an anatomical to a physiological

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2. Ibid., p. 137-8.

connotation.

No longer could speech be considered a combination of independent elements. Disease could not be thought of as resolving language into the various elements of articulatory, visual and auditory activities. The psychologists began to demand a dynamic interpretation of mental activities. No longer could they see conscious processes reduced to sensory or motor presentations and laws of association.

Foremost in causing this rupture with the old ideas was Henry Head. Many of his ideas stemmed from Hughlings Jackson whose works had been so long ignored or slighted. Head denied the idea of centers for the use of language and granted only "certain areas within which destruction of tissue produces a disorder of some particular mode of behaviour."[3]

Head's final views seem but the prologue to those of Goldstein, just as Jackson's views anticipated Head's. The following quotation climaxing Head's great work anticipates the foundation for Goldstein's organismic approach:

> When some act or process is disturbed in consequence of an organic or functional lesion, the abnormal manifestations are the result of a fresh integration carried out by all available portions of the central nervous system. It is a total reaction of the organism to the new situation, in which conscious processes play their part as a mode of response. Certain aspects are

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3. Head, op. cit., p. 137.

> psychical, others are somatic, but there is no separation of mind and body so long as we are examining the consequences of disintegration.[4]

Thus the three milestones in the psychological approach are Jackson, Head, and Goldstein. The latter argues against the study of postmortem findings and advocates instead the study of living tissue. This insistence--in our present state of knowledge--would seem to minimize much of the neurologist's findings since we are not yet able to clearly interpret the relationship between a definite anatomic condition and a specific performance. Indeed, even the psychologist can say little more than that speech disturbances due to brain lesions are symptoms of the disturbance of certain functions or mechanisms.

In disagreement with--if not in opposition to--the psychologist's view are those of Henschen and Nielsen. These men have insisted on an anatomico-clinical basis. Indeed, Henschen went so far as to state that every psychological activity could be localized, even logical thought.

Although primarily a neurologist, Nielsen is more cautious and asks instead for a three-way approach (psychological, physiological, and anatomical). His work, however, favors the anatomico-physiologic side. The psychological side, although slighted, is not denied.

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4. Ibid., p. 549.

At the present time there is no one generally accepted system of classifying aphasic disorders. Aphasia can be classified according to anatomical, physiological, or psychological schemes. Much of the confusion over terminology has arisen by the carelessness of various writers in transfering terms from one frame of reference to another.

Anatomically we could designate aphasia in terms of the site of the lesions calculated to produce it. Then we could speak of frontal, parietal, temporal aphasia, etc. Physiologically the aphasias could be classed as motor, sensory, mixed, or transcortical. The aphasias are also capable of psychological classifications such as that of Head.

Classifications in each of these frames of reference have proven unsatisfactory by themselves. Wilson sums up the dilemma in that "a psychological arrangement has the disadvantage of losing touch with cerebral function, while an anatomico-physiological arrangement is calculated to introduce an artificial simplicity into a particularly complex subject and to ignore what must always be its fundamentally significant side."[5]

Perhaps, the words of Henry Head most fittingly describe and explain the many systems of classification. He remarks that

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5. S. A. Kinnier Wilson, "An Introduction to the Study of Aphasia", The Lancet, 41:2 for 1921:1145, Dec. 3, 1921.

> The tendency to appear exact by disregarding the complexity of the factors is an old failing in medical history. Each patient with a speech defect of cerebral origin is stretched on the procrustean bed of some theoretical scheme: something is lopped away at one part, something added at another, until the phenomena are said to correspond to some diagrammatic conception, which never has and never could have existed. And yet neurologists continue to cling to these schemes, modifying them to suit each case, conscious that they do not correspond in any way to the facts they are supposed to explain.[6]

However, in the systems of classification that do exist, there is a quite prevalent agreement on the division of aphasias into two general categories--motor or expressive, and sensory or receptive. These both, in turn, have been divided into so-called pure and complex types. There is a third general division not properly included in these main two categories. It is the disorder known as amnesic aphasia or anomia. In this disturbance the patient has difficulty in using nouns.

In another sense there also are aphasias on the lower levels and aphasias on the higher levels. Those on the lower level are so severe that they are easily recognized. Those on the higher levels are quantitative rather than qualitative limits.

The Broca's aphasia (or motor or expressive) is the loss of the power to speak, write, and/or draw, although the comprehension of writing and speech is relatively retained.

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6. Henry Head, "Hughlings Jackson on Aphasia and Kindred Affections of Speech", Brain, 38:1-2.

The motor aphasic is not mentally dull; he is unable to express himself by spoken words. He is speechless but not wordless. (It is assumed that the few words he can utter he does so by means of the uninjured speech centers in the opposite cerebral hemisphere). The defect is believed attributable to a lesion in the cortex of the left third frontal convolution, "but it may extend into neighboring parts of the frontal lobe and even into the insula."[7]

Various writers have described two varieties of motor aphasia: the pure (Dejerine) or subcortical (Wernicke), and the cortical (Wernicke) or true cortical (Dejerine). In the pure or subcortical variety speech alone is involved. The patient can speak neither spontaneously or by repeating what he hears, though he understands all that he hears or reads. "In this form the pathway from the center in the cortex is blocked although the center itself is intact."[8] This subcortical motor aphasia is frequently found in hysterical states. "It is likely that it is the mental state and not the location of the lesion that is important."[9] This type is recognized by Goldstein and Kleist, while Marie described it as anarthria.

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7. Andersen, op. cit., p. 186.

8. Odell, op. cit., p. 164.

9. Leland B. Alford, "The Mental State Associated With Cerebral Lesions", Psychosomatic Medicine, 5:1:16, 1943.

The cortical variety--synonymous with Broca's aphasia, Head's verbal aphasia, and Goldstein's central form of motor aphasia--is like the pure type plus a certain loss of co-ordination of memories of words either spoken or written by himself. The patient has difficulty in articulation and retarded, scanty speech. The lesion underlying the sub-cortical type is "thought to involve not only the third frontal convolution, but a larger area including the neighboring part of the frontal lobe and the anterior part of the Island."[10]

In transcortical motor aphasia (Wernicke) the patient is usually able to repeat speech and to write from dictation and to copy, although he cannot speak or write spontaneously. Although the cortex in the region of the posterior part of the third frontal convolution may be responsible for this type, this localization is in great dispute.

Wernicke's (or sensory or receptive) aphasia is characterized by an inability to understand the spoken or written word. There are two main divisions of the pure or subcortical forms: auditory aphasia or word-deafness, and visual aphasia or word-blindness. In auditory aphasia, or word-deafness, the patient hears sounds but does not understand spoken words. In visual aphasia, or word-blindness (or alexia), the patient can see but not understand printed or

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10. Weisenburg and McBride, op. cit., p. 60.

written characters.

The cortical sensory aphasia (Wernicke's aphasia, or Marie's true aphasia) involves defects in speaking and writing in addition to difficulties in the understanding of speech and reading. He continues to talk even though he may recognize many errors in his speech. "His volubility seems to be due more to an inability to cease rather than a press of activity."[11] The localization of cortical sensory aphasia

> is considered to be, roughly, the Wernicke area, the posterior part of the first temporal convolution together with the neighboring part of Heschl's convolution, the parietal lobe, and the posterior section of the Island."[12]

Transcortical sensory aphasia is similar to Wernicke's aphasia except that the power of repetition is retained. In other words the patient manifests a loss of understanding although still able to repeat and write to dictation. He also reads aloud without comprehension.

Total aphasia is a combination of the symptoms of Broca's aphasia and Wernicke's aphasia. Hence the patient is speechless and wordless, usually with loss of ideation.

Amnesic aphasia (Goldstein, Pick, Heilbronner) is "difficulty in finding words as names when words and phrases in certain uses could be evoked more easily and when other

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11. Nielsen, op. cit., p. 69.

12. Weisenburg and McBride, op. cit., p. 71.

language processes remain relatively undisturbed."[13] The patient knows the word he wishes to use but cannot recall it. He accepts it (but no substitute) as soon as he sees it written or hears it spoken. The statement "I know but I can't say it" expresses the essence of amnesic aphasia. Although there is a good deal of argument over localizing the lesion underlying amnesic aphasia, it would seem to be

> a lesion of the major posterior temporo-occipital region or its superior connection with the rest of the brain through the temporal isthmus. In terms of the areas of Brodmann those potent in production of amnesic aphasia when a lesion occurs in them are the area 37 and the posterior portions of areas 21 and 22.[14]

In addition to the traditional two-fold classification of aphasias, with its various subdivisions, there is the four-way classification of Weisenburg and McBride. These authors listed expressive, receptive, expressive-receptive, and amnesic categories. Those which they listed as predominantly expressive showed the most serious disturbancesin expression in speech or writing. Those predominantly receptive showed greater deficiency in the receptive processes than in the expressive, and changes in speaking and writing most of which differ in nature from those in the expressive disorder. The expressive-receptive type showed extreme limitation in speaking

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13. Ibid., p. 78.

14. J. M. Nielsen, "The Unsolved Problems in Aphasia III. Amnesic Aphasia", Bulletin of the Los Angeles Neurological Society, 5:84, 1940.

because of defective articulation and word formation. Errors in spelling and gross changes in word formation would also reveal themselves in writing. The amnesics show qualitative defects by being unable to evoke words as names of objects.

K. Kleist (cited by Weisenburg and McBride, 1935) has given us a system of classification which embraces word-sound-deafness, word-deafness, name-deafness, and sentence-deafness as sensory or receptive disorders. His motor disorders are divided into speech-sound-muteness, word-muteness, name-muteness, and sentence-muteness.

Another set of four major categories is postulated by Head. These are verbal, nominal, syntactical, and semantic. (See Chapter entitled "Henry Head and Revival of the Psychological Approach"). His nominal aphasia is evidently the same as the previously described amnesic aphasia. It is a tendency to forget names. His verbal aphasia definition quite accurately fits Broca's aphasia.

Sir James Purves Stewart's comment on Head's new classification in 1920 would seem to apply equally to several of the other systems of classification. Commenting on the fact that each was describing the same thing in different terminology, Stewart noted that

> Dr. Head by a series of ingenious and elaborate tests has arrived at a new, useful and philosophical basis of classifying aphasias. But this does not necessarily mean that other classifications may not also be correct. Just as one observer may classify a crowd of people into men, women and children; whereas another may classify them them into Englishmen, Scotsmen, Irishmen, Welshmen and foreigners; or another again into soldiers and civilians.

> It all depends on the angle from which the facts are viewed. Dr. Head has opened a new window into the chamber of aphasia. But this does not compel us to close up all the other windows, especially the anatomical and pathological windows, through which so much light has been admitted in the past.[15]

At the present time research on aphasia is dominated by neurologists who, in general, neglect its psychological implications. Perhaps symposiums attended by the leading neurologists and psychologists might aid our understanding of aphasia as a result of their concerted efforts in intelligent argumentation and the exchange of ideas.

In closing this study, I cannot help but speculate, as did the Philadelphia neurologist James Hendrie Lloyed nearly half a century ago, whether we are attempting to try to make an anatomical dissection of a complicated psychological process.[16] Since attempts at viewpoints blending psychology, anatomy and physiology have proven unsuccessful, perhaps we need to think more along the line delineated by Samuel Brock[17]--that it is a matter of the art, rather than the science, of knowing when to leave off the one approach and take up the other.[18]

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15. Sir James Purves Stewart, (Discussion on Aphasia), Brain, 43:433, 1920.

16. James Hendrie Lloyd, "Sensorimotor Aphasia", New York Medical Journal, 99:916, 1914.

17. (Samuel Brock:--New York neurologist and psychiatrist, 1893-___)

18. Samuel Brock, "Apraxia and Aphasia", Bulletin of the Neurological Institute of New York, 7:3:321, Dec., 1938.

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