APHASIA AND ASSOCIATED SPEECH PROBLEMS

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BY

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PREFACE

It is not without much hesitancy that one approaches any of the problems related to speech, whether this be its purely linguistic phase or the complex mechanism of its production and defects. As a field of physiological investigation, it has had its noted pioneers followed by many skilled craftsmen. It has also been especially fertile in its crops of spirited controversy. Yet the larger part of this development has been well within the last half century; indeed, the first serious attention to the subject had its inception in a notable contribution by Broca in 1861.

Speech so unquestionably is a subject inviting controversy that one is aware of an inadvertent disposition to take sides. Whether your allegiance be with the exact localizationist, the extreme diagrammatist, the comprehensive synthetist or the lenticular quadrilateralist will depend upon the degree to which you have been imbued by the ideas of Broca or Wernicke, Hughlings Jackson or Pierre Marie.

However helpful the several hypotheses concerning speech production and its dis-

turbances have been (and there is every reason for admitting their real value) it is not difficult to detect the dogmatic inflection in them all. A general critique of the physiology of speech must of necessity limit itself to a discussion of the theories by which, in lieu of a more satisfactory foundation at present wanting, the subject is indoctrinated. Nor is it probable that any other approach to the speech problem in and of itself will bring more satisfying results in understanding.

The vocal articulation of language, one of the most delicate of all skilled acts, differs from other skilled performances only in the degree of its complexity. Not until the neural mechanism of such motor performances is thoroughly understood will the nature of speech production and its defects be intelligible.

There can be no dispute that a definite synthetic process of nervous energy is indispensable to the acquisition and execution of all skilled acts. The reflex and tonic status of the muscles must be adequate, the synergic and automatic associative control must be exact, but even more essential than these is that composite of cerebral influences derived from the sensory and psychic elaborations which enter into the formation of intelligence.

Preface

Articulate speech is the result of such a synthetic process. Its control by the nervous system cannot be attributed to any center or group of centers in the brain. It is dependent upon a number of parts and areas correlated to mediate the several specific influences which enter into its composition.

Dr. Osnato has rendered a distinct service by emphasizing the difficulties of the problem in his thoughtful and illuminating review of the theories concerning speech production and its defects. His thesis will prove a stimulus as well as an aid to those interested in the subject.

FREDERICK TILNEY.

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INTRODUCTION

The problem of speech from a medical and purely psychological standpoint is so unsettled that contributions to our knowledge, or aids to the understanding of that knowledge which we already possess concerning speech, are surely to be welcomed. Particularly are the difficult problems of speech disturbances in the various types of aphasia in a chaotic state, nor do we have precise knowledge of the mechanisms which cause disturbances of speech in the various so-called functional conditions as in hysteria, dementia precox and manic depressive insanity, etc. Indeed, the speech disturbances seen in dementia precox and general paresis, both organic diseases of the central nervous system, are not entirely clear as to the mechanism of their causation. Further, it has always been an interesting problem to explain precisely the cause of speech defects occurring in Friedreich's ataxia, paralysis agitans, multiple sclerosis, cerebellar ataxias, the various cerebral and cerebrocerebellar diaplegias, and finally, but not of least interest, speech defects occurring under extraordinary conditions of fatigue, stress or emotion. The orthodox

views on the development of speech and the localization of the various speech centers are faithfully presented. In them one searches in vain for a clear understanding of normal or defective speech. The influence of the cerebellum in connection with speech has been almost entirely neglected. In these pages an attempt is made to ascribe to the cerebellum an important place in the exhibition of that exclusively human function.

At the outset we desire to have it understood that our contribution is based in no instance on any original anatomical investigation, either of our own or other material. In all fairness, it must be conceded that anatomy, both experimental, normal and pathological, has failed to clear up the problems of speech in the various types of aphasia, for instance. It has appeared to the author that a revision of our conception of aphasia is important and, therefore, this clinical study was undertaken. My inspiration for undertaking this work was received from Professor Frederick Tilney, and some of the clinical cases used in this study were observed with his aid and guidance. Others of the cases are from my private practice and from the neurological wards of the Kings County Hospital. The chapter which represents the generally accepted orthodox views on

speech is made possible by a translation of Emil Villiger's monograph, "*Sprachentwicklung und Sprachstörungen beim Kinde—Unter Berücksichtigung Hirnanatomischer Grundlagen* (1911)" on speech disturbances and speech development in the child. For this translation I am greatly indebted to Dr. I. S. Wechsler, Instructor in the Department of Nervous Diseases, Columbia University.

After presenting in the first chapter the views of Villiger in abstract, the attempt will be made, by reference to Moutier's figures as the pathological basis and to Marie's argument for a revision of the question of aphasia which appeared in his writings of 1906, to break down the conception of aphasia as it is now usually understood. In doing this, certain references will be made to clinical material of my own and others to show the absolute necessity of such a revision. Following this, our conceptions of speech will be laid down in very broad viewpoints which will be upheld in so far as clinical material can be made to do so by reference to specific cases observed by the author. Clinical studies will be advanced in illustration of theoretical conceptions. Anatomy and pathology having failed to elucidate the problem, we shall in these pages attempt to call clinical medicine to our aid.

APHASIA AND ASSOCIATED SPEECH PROBLEMS

CHAPTER I

VILLIGER'S CONCEPTION OF SPEECH DEVELOPMENT

The first sound made by the human being is a reflex cry, brought about by the need for respiration in the child. Soon, however, the child seeks to give tangible expression to its needs, likes and dislikes; sensations of cold and of being wet, of skin irritation, of hunger and thirst are now often accompanied by crying. Even pleasurable sensations are accompanied by crying, and in the beginning none of these cries can be with any certainty differentiated. At the end of several weeks, however, a specialization of the various cries occurs and there can be distinguished two groups, those cries which indicate a state of well being and those which indicate the contrary. States of well being are characterized by a cry which is associated with smacking, sucking and "lalling." The lalling activity of which Villiger speaks

consists in the production of sounds, syllables and multiples of syllables. Vowels are combined with consonants in a lall monologue. According to Villiger, the first sounds produced are not always those which require the simplest physiological strain, for gutturals are very often pronounced before the labial sounds. Many exceptions to the general rule, however, regarding this is possible, because the environmental influence surrounding the child, particularly the amount of care and attention a child receives from its parents or relatives, will determine the speed and character which speech development shows in the individual child. There are two periods in the child's development when these undifferentiated sounds are made. The first one occurs in most children between the ages of three to six months and is characterized by the formation of laryngeal and palatal sounds in combination with Ah, Ay and Oh, besides many inarticulate sounds. The second period occurs between the ages of six months and one year, a time during which we have the production of tongue, lip and palatal sounds, the first two predominating. In the first period, the child does not initiate these sounds with any purpose but amuses itself in the production of them. The frequent repetition of sounds in the first lalling period

is explained by the structure of the articulating organs and the manner of sound production. The air current in passing the larynx and the mouth is impeded and made into tones by the speech organs. As the air first passes by the palate, the palatal sounds are the first to be formed. The exercise which the lips get in sucking conditions the early production of labials and dentals. Every child is born with a psycho-motor tendency or instinct to speech, and irrespective of any sound stimuli in the environment, all children will seek expression in speech.

The child is a motor being, much more so than the adult. In play, it gives expression to all its inner feelings and thus the speech organs in the beginning are used for this purpose; later, all the other organs of the body over which the child exercises any control are used. For unpleasant emotions there is the cry, and soon pleasant emotions are differentiated through another form of sound production which Villiger designates as lalling. After the first period of lalling, namely, from three to six months, a new factor enters to shape the development of speech, which is hearing. In the first weeks of babyhood there is deafness. Soon, however, it shows the ability of hearing by blinking or shrinking in fear at sudden

sounds. At the third month a child will turn its eyes and ears in the direction of sound and will be attentive and seek eagerly for acoustic impressions. Hearing is much more important for the development of speech than seeing, not only for speech but also for the psychic development of the child as a whole. The loss of hearing robs the child of any approach to its soul. As the child learns to hear it begins to pay attention to its own lalling and noise production, it soon begets a correct interpretation of its own speech sounds and gains control over the musculature which produces them. Speech sounds which the child itself makes and the sounds which it hears are the basis of speech. He first hears what he himself speaks and then speaks what he hears from others. From this spring imitation and repetition. At first there is self imitation and then there is gradually added to this, imitation of speech which is heard or of other natural sounds. Repetition such as we understand it in the adult appears much later than this stage of development in the child. Other senses enter into this process of imitation of speech and can be seen and even felt by the child, but nevertheless, hearing is much more important than any of the other senses for the development of speech. This is illustrated by

the case of deaf-mutes. However, by watching the lips and the mouth, the child (as may the adult) learns to understand speech. Helen Keller can understand speech by holding her hand on a speaking person's larynx. Therefore, she employs the sense of touch in the understanding of speech. But we soon notice that children mutilate speech in imitating it, either because of lack of attention, difficulty in hearing or inability to repeat difficult sound combinations or even possibly because of indistinct speech on the part of adults who speak to the child. Further, this mutilation of speech in imitation may be due to the fact that the child repeats sounds which he hears which are not speech. The first imitations are altogether voluntary. Actual imitation comes much later in children than is generally supposed. Stern found that this ability in children to imitate shows itself at nine months, but mostly is limited to gestures, unarticulated sounds and to the cadence of voice. Repetition of articulated sounds develops with a bound and in the third year this is quite prominent. Indeed, words are now understood and a few are spoken with understanding. In the course of learning speech, there is often a peculiar period of echolalia.

As soon as the child learns to lall, another

factor enters. The adult who listens learns to understand some of the words from the babbling of the infant and repeats them to the child, singly or in sentences. These combinations picked by the adult from the child's lalling are in turn repeated by the child and meaning may be attached to them by both. This adult-child language varies with the mentality of the infant, but often children will show some discrimination in choosing from the language which is offered them by the adult. This discrimination is determined by the fact that it chooses the sounds which are phonetically most easy for it to produce. Other factors may influence this choice; namely, the surroundings and the lines of interest which the child may be trained to show in its selection. To the theory that children can invent words, Villiger answers that children have often been observed to speak words which have never been spoken to them by adults. This, however, he explains by the fact that infants mutilate words and in the beginning do not hear properly; or meaningless, badly pronounced words are spoken to them by adults, and thus the nature and quality of its speech and sound intake are not properly controlled or standardized, and in this way the child will employ words for things other than those for which

they are intended. Again, even adults will attribute meaning to senseless articulations and will often call them words when it is perfectly obvious that the child has absolutely no meaning to convey by the sound produced. Speech understanding is of great importance for the production of speech imitation. Very early one can observe the impressions which are made on the child's feelings by the mother's voice or other signs. At first the child's feelings may not show a differentiation in reaction to what is spoken to it. Later we notice how the child's feelings are expressed in various movements as shown when certain things or persons are mentioned, the infant responding to these words by turning the head or the eyes in the direction of the objects mentioned. For instance, on saying "tick, tick," and holding a watch before the child, it learns to gaze in the direction of the watch, and even if the words "tick, tick," are said and no watch is immediately visible, the child will look in the direction from which the sound is made. The child cannot be said at this stage to have a visual conception of the object. These traces of speech understanding appear more clearly in attempts at training the child. Thus, when a child is taught to stretch out its hand

upon "Give me your hand," it learns to associate action with sound. This, however, is really not actual speech understanding but only a mechanical repetition of practiced action.

Soon the child learns to attach great importance to speech. He sees in it a close connection with objects and occurrences and finds that it is the only means for communication and discrimination. In time he learns to recognize words as signs for objects and finds understanding is also furthered by gestures. Thus mimicry acts as a connecting link between sound complexes and expressions. Finally, speech is understood without the accompanying mimicry or gesture. In many cases speech understanding may be far in advance of actual speech, for normally there exists in all children a period when a great deal of what is said is understood but only a very small proportion of this can be uttered by the child. This is designated by Villiger as hearing mutism and might be compared to what we see in the condition known as motor aphasia, or in some types of psycho-motor retardation such as we see in depressions.

According to the psychologist Neumann, the first words which a child utters give expression to emotions and desires. Following this,

the volitional stage ensues, finally the intellectual stage of speech is developed, and in this stage objectivity is attached to words and they are thus used for designations and communication. Only in much older children are words used associatively, for a child may employ one word for a number of objects or events, simply because the word was first used in a certain way, the child unthinkingly connecting it with all things that bear a resemblance to that particular instance. The child may recognize and designate objects by signs or by combining parts, and may employ one of these parts for the whole object. For instance, it may call all quadrupeds "Bow-wow" or dog, and all women "Grandma." The reason for this lack of associativeness is the fact that there is still an incomplete perception of things. This lack of logical conception of words as symbols may be due to want of attention, lack of power of memory and narrowness of consciousness. Gradually, however, the child learns to know that separate word symbols are used for different objects and by means of constant questions and answers and corrections acquires the proper use of these words and various conceptions from which grow systematic and conscious ability of speech. It is fairly well established, first, that there are no

sharp lines of demarcation between the various stages of speech development. Secondly, that the development persists at varying degrees of speech, and thirdly, that speech development and the development of intelligence usually have nothing to do with each other. This is almost a literal translation of Villiger's view, shared by a great many, that speech and intelligence may be entirely separate things in the development of the individual. How much we disagree with this conception will be brought out later. That this is a fundamentally important point is obvious, for if this statement is true then the anatomical localization of various speech centers is justifiable, and the conceptions which have allowed so many to place diagrammatically limited centers over various speech functions in the brain are correct. However, we hope to be able to prove that this statement is not logical, and is, therefore, not true and that the development of speech and intelligence go hand in hand and are interdependent to such an extent that they may be, to all intents and purposes, one and the same process. Villiger further states that normally articulated speech should be developed by the eighteenth month and that it should be a fully developed function by the end of the fourth year. Normal variations may,

however, place the outside limit up to six or seven years, particularly if one considers sentence construction and stylistic expression to be part of speech development. Indeed, after this, cultivated refinements of speech are included within speech development and may be said to continue throughout the school period.

The building up of the speech mechanism furnishes the second part of Villiger's interesting little book. The first part of this deals with a review of the evolution of the conception of psychic functions and their localization in the brain. He calls attention to the work of Gall in 1820 as showing that the cerebral cortex was the seat of psychic functions. Flourens and Foville established this conception as a fact by experimental and pathological work in 1830. Marc Dax in 1836 observed speech disturbances in patients with right hemiplegia. His son, G. Dax, in 1863 showed that the lesion in such cases was in the left hemisphere; and, working independently, P. Broca, in the same year, showed by a report of his first two cases that the localization of the seat for articulated speech was in the foot of the left third frontal convolution. He further describes how, as a result of pathological research and histological differ-

entiation of the various layers of the cortex, it was determined that different cortical regions had specific functions. He describes the failure of observers, particularly Beaunis and Fournier in France, and Nothnagel in Germany, to localize centers in the cortex by injecting liquids. The oft-quoted experiments of Hitzig and Fritsch in 1870 proved by electrical stimulation the presence of motor areas in the cortex of animals.

Ferrier, working with the faradic current in 1872, confirmed their findings. Sherrington and Greenbaum in 1901–02 confirmed this work on apes, and finally F. Krause added his confirmation, working on human beings. References are made to the work of numerous physiologists, such as Monk, Nothnagel, Corville and Goltz, which definitely showed that motor or sensory activities disappear on destruction of definite parts of the cortex. Attention is called also to the clinical work of Charcot, Hitzig, Kussmaul, Wernicke and Dejerine which confirm the experimental work of the others. From all this, Villiger decides that it has been established that the cortex consists of physiologically differentiated sections in the sense that definite portions have specific elementary functions. This conception belongs particularly to Wernicke and

is reenforced by the work of Berlin in 1858, Arndt in 1867, and Meynert in 1886–72, who showed differences in the structure of various convolutions, and of W. Betz in 1874, who showed the presence of the giant pyramidal cells in the ascending frontal convolutions. Following these, the researches of Bevan and Lewis, Clarke, Obersteiner, Golgi, Ramon Y. Cajal, etc., first showed that the cortex in imbeciles and idiots lacked functionally active nerve cells, establishing thereby the conception that nerve cells are essential in psychic activity. Gennari, Soemmering, Vicq-d'Azyr, Rennak, etc., showed the presence of fibers, both projectile and association, in the cortex, and Tutzek and Zacher observed the disappearance of these fibers in general paresis and senile dementia. Vulpius and Kaes studied the development of fibers at various ages. Flechsig showed that fibers and tracts, both in the cortex and in the cord, become myelinated at different periods of development. By means of the special methods discovered by Golgi, it was observed that fiber and cell are one structure. Cajal gave exact description of the motor and sensory cortical layers and Broadman worked out the typographical localization. The keen and masterly work of Hughlings-Jackson is not mentioned with

those of Charcot, Hitzig, Wernicke, Dejerine, etc., as the clinicians who helped in the localization of cortical function. Nevertheless, Head regarded Jackson's work of such great importance that he undertook to gather and edit a number of his publications which appeared together in an issue of Head's periodical, *Brain*, parts 1 and 2, Vol. XXXVIII. Further reference to this work will be made later.

To continue with Villiger's conception of speech, we find that in the third or fourth month of embryonal life the cortical cells are undifferentiated. Toward the last quarter of fetal life, one can distinguish six layers. In the interval between these two periods there occurs a rearrangement of cells into layers of lesser or greater density with differentiation in cell structure, together with a regrouping of fibers. The further development of this histological localization continues in the child up to the second or third year and one may assume a parallelism between organic and psychic development from these facts. This may be borne out by the observation of H. Vogt, who showed that there was no structural differentiation in idiots. This has been shown further phylogenetically, for the same cortical area differs in different animals during the

various stages of development. The lower the animal, the more granular cells there are; the higher the animal is, the more pyramidal cells are found. According to Rondoni, those layers in the human which are developed earliest show a preponderance of granular cells, and those which develop latest, a preponderance of pyramidal cells. Further, there arises an association between the various cells in the same cortical area, as well as between different cortical areas. From which may be adduced that while for more limited functions there are distinct cortical fields, for higher performances a great part, if not the whole, of the cortical area is necessary. Because of this very point do we differ from Villiger's conception that speech and intelligence develop separately, for the remarks just recorded from Villiger's pen are susceptible of being called upon to prove that speech is merely the visible expression of intelligence and develops apace with the latter. Villiger says that the following main centers are distinguished: First, the motor center which embraces the central convolutions, particularly the precentral, the posterior part of the frontal lobe and a large part of the lobulus paracentralis. Second, sensory centers; (a) touch, pain, and temperature in the post-central convolution

and the adjourning part of the parietal lobe, possibly also the precentral convolution. In this region is found sense of position and movement, of space and place; (b) hearing in the middle and lower part of the temporal convolution and particularly the convolution of the insular lobe; (c) a visual center in the occipital lobe, particularly the cortex of the calcarine fissure; (d) a small center in the front part of the gyrus hippocampus and cornu ammonis which are for taste and smell—the outer part probably for taste and the inner part for smell.

These motor and sensory areas occupy perhaps a third of the brain cortex, the rest of it being considered by Flechsig as areas for association centers which are the seat of higher psychic functions. This latter author explains the development of centers on the ground of myelinization as follows: At birth only smell and taste centers are developed, then come in order, myelinization and function to the centers for touch, vision and hearing. Later, the individual territories of the psychic centers. According to this author also, the projection areas, motor and sensory, are the only ones in touch with the lower brain centers, whereas association centers are connected only with one another and with the

projection centers. The cytological studies of Broadman have shown histologically different areas in the so-called association centers. While little is known of the function of the anatomically circumscribed areas, the existence of memory centers seems certain. Thus, there are lesions in centers of perception which destroy perceptions but not memory pictures, and on the other hand, there are lesions near perception centers such as the visual or auditory, where the memory picture may be lost but not the actual perception. For instance, we may have psychic blindness wherein the patient knows the form and color of an object but cannot tell what the object is, or in psychic deafness, the speech may be perceived as sound, but not recognized as a symbol. Or again we may have tactile agnosia wherein objects are felt and perceived through the sense of touch but not recognized.

CHAPTER II

THE DIAGRAMMATIC CONCEPTION OF SPEECH CENTER

Speech centers embrace a definite cortical area of the left hemisphere, according to Villiger, wherein he follows a great number of authors on this subject. These are placed (a) at the foot of the lower frontal convolution (Broca's area) which possibly impinges on the lowest portion of the precentral convolution and the insula. Injury to this area leads to loss of voluntary speech, repetition and also reading aloud. This statement may be read again and again in various works on this subject despite the fact that it has been proven time and again that lesions in Broca's area have been observed at autopsy without any speech disturbance whatsoever. (b) In the posterior third of the superior temporal convolution. Next to the lower parietal lobule lies the sound picture or auditory center. This is the Wernicke center which contains the memory pictures of heard and spoken words. It is the center which is injured when word deafness occurs, that is, such a patient hears

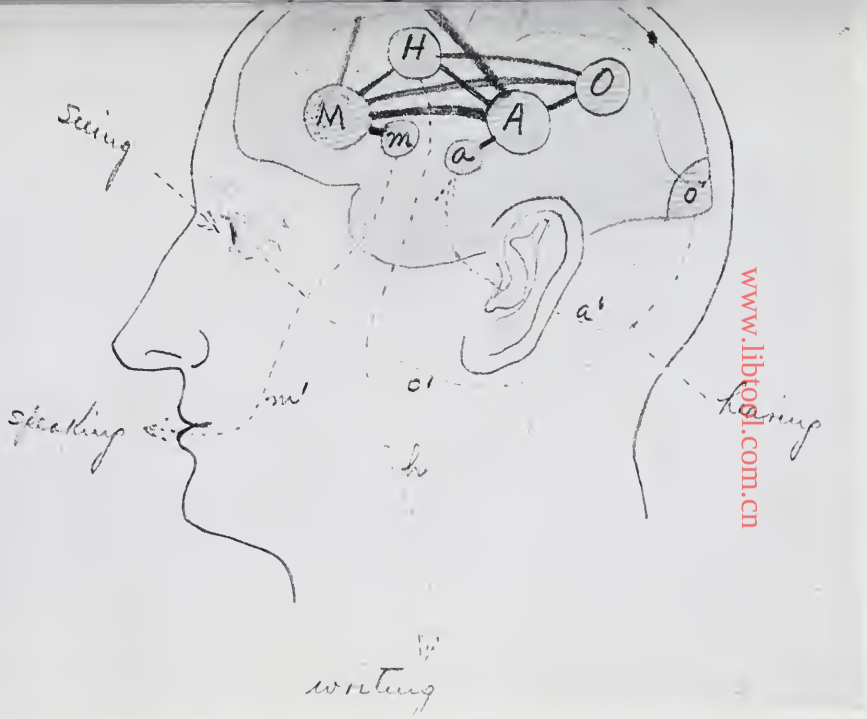

FIG. 1. DIAGRAM OF SOUND LANGUAGE AND WRITTEN LANGUAGE

words but lacks understanding. (c) In the angular gyrus lies the visual center for memory pictures of written signs. Alexia occurs when this center is injured. In other words, we have word blindness. (d) Some consider the existence of a separate writing center in the foot of the middle frontal gyrus, but it is likely that this belongs to the motor center for the hand.

All these centers are memory centers for the representation of movements, of articulation, of acoustic and visual pictures and of signs of speech.

Figure 1 shows the connections between the motor and the sensory speech areas. When a word is spoken it reaches through the path a' and a memory picture for recollection or recognition is formed in the sensory speech area A. From here the impulse travels to the motor area which enables one to repeat the word. But as the child's musculature is unskilled and its attention restricted, the repetition at first is incorrect. Gradually, a memory picture for the expression of the word is formed and deposited in a definite area M, which becomes the motor speech area, so that later, in the repetition, the sensory impulses from A travel to the motor speech area M, and the memory pictures from this center set in motion the necessary coördinated movements

in the motor center *m* in the lower precentral convolution, and from here this impulse travels to the speech musculature by way of the speech path *m'*. The process, then, is *a'-a-A-M-m-m'*.

Now we can explain the concept formation in the child during speech development. Suppose the child sees a rose. From the eye an impulse travels to the visual cortical area where the rose is seen. Immediately a memory picture is deposited in the center *S* (Fig. 2), so that the child can recall form and color. As the child can also smell and feel the rose, memory pictures are deposited respectively in *R* and *F*. As these centers are connected by association fibers, upon seeing the rose, there are awakened in the child memory pictures of its smell and the feel of the petals; thus a concept "Rose" is formed out of partial representations. As the word "Rose" is spoken to the child an acoustic memory picture is deposited in *A*, so that the child recognizes the word as a sign for the flower. A composite representation is the result.

For the understanding, it is necessary that the speech center *A* enter into closer connection with those centers whose association makes possible the formation of a concept. To represent this graphically, it is necessary

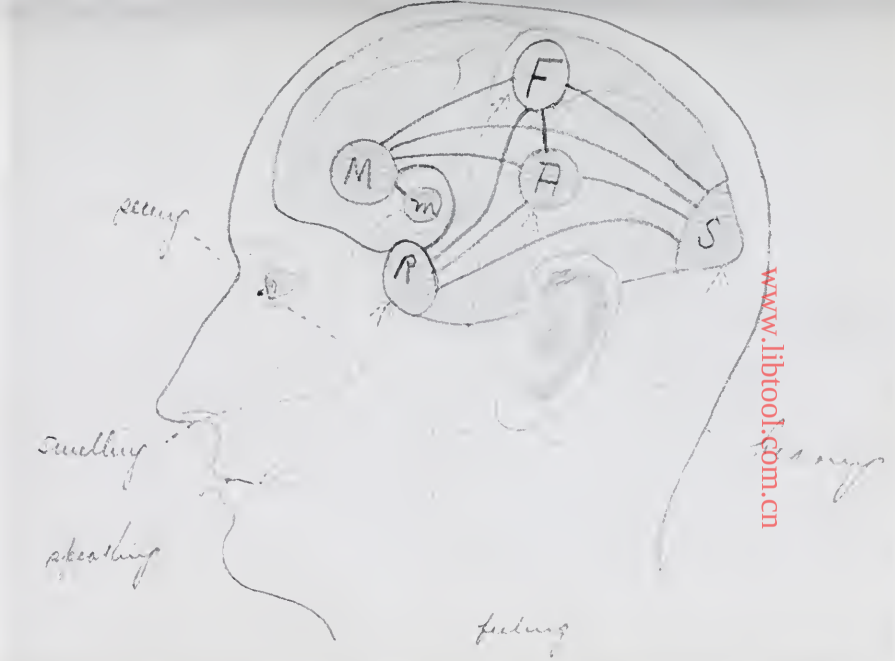

FIG. 2. DIAGRAM OF CONCEPT FORMATION

to inscribe besides *A* in Figure 1, the memory centers *S*, *R* and *F*, and their interconnections with the sensory speech center *A*. It is also necessary to insert a single "concept" center *B*. But it must be emphasized that concept formation is not to be represented as a simple event but one which is complex and depends on the integrity of numerous associated cortical areas.

Through the connection of the sensory speech center with the concept center, the foundation is laid for the possibility of speech understanding. Not only can the child, at this time, speak without understanding (*a'-a-A-M-m-m'*), but it can understand without speaking, (*a'-a-A-B*). From this is derived loud repetition of words with understanding—actual speech. At first, this follows on the path *B-A-M-m-m'*, and then, in consequence of late associations *M-B*, on the path *B-M-m-m'*.

Another step is needed for the understanding of speech disturbances. The first associations represent speech in the broad sense. This is broadened out by the hearing of written speech, reading and writing. The word signs are to be looked upon as signs of sounds and not concepts. Individual words are decomposed into letters and syllables and an optical letter-picture is associated with each sound,

vowel or consonant. In Figure 1, *O* represents this memory center for written signs to which impulses from the eye travel by way of *o* to the visual center *O*. As the child learns to read aloud, a picture of the letter is deposited in *O*, and as it syllabilizes aloud a sound picture is formed in *A* and at the same time a memory picture of the necessary coördinated movement of the speech musculature is laid down in *M*. That is, the optical center *O* enters into connections with the sensory center *A* and motor center *M*. For writing, a further association is necessary of *O* with the center for the movement of the hand *H*. At first, the child copies mechanically (*o'-o-O-H-h*), through which graphic-motor memory pictures are developed in *H*. Later when the child writes from dictation, it softly repeats to itself each word (*a'-a-A-M-m-m'*), wherefore, in consequence of the connections *A-O* and *M-O*, the picture of the letters is awakened in *O* and the impulses from here to *H* make possible writing from dictation. Finally, the connection between *H* and *M* and *A* becomes direct and voluntary or conscious writing becomes possible.

From the diagram of sound and writing speech, the following may be derived:

Repetition..... *a'-a-A-M-m-m'*

Speech understanding... *a'-a-A-B*

Spontaneous speech.....	B-A-M-m-m' B-M-m-m'
Reading.....	o'-o-O-(A)-B (M)
Reading aloud.....	o'-o-O-(A)-B-M-m-m' (M)
Copying.....	o'-o-O-H-h
Writing from dictation..	a'-a-(A)-O-H-h (M)
Spontaneous writing....	B-(A)-O-H-h (M) B-M-H-h

According to the diagram in Figure 1 the speech disturbances may be divided as follows:

It is assumed that the speech mechanism is built out of corticipetal, intercortical and corticifugal pathways. The main inlet is the hearing pathway. As has been shown, visual and touch pathways play a secondary rôle. The trapezoid form in the figure shows the intracortical association of the speech centers to form a speech zone. The speech zone is connected with the concept center. The main outlet is the motor speech pathway from the motor speech center to the speech musculature.

In disturbances of the hearing pathways, speech cannot ordinarily be developed since no center can be developed for the sound of

words. The same may be true of a high grade of hardness of hearing. In this case we deal with hearing mutism. Speech is unintelligible even in those who have become deaf in a later period.

In disturbances of the central or intracortical pathways, we have two kinds of central or cortical speech disturbances. First, those disturbances of intelligence in mentally diseased or mental defectives are known as dyslogias or dysphasias. Second, lesions in the sphere of the speech centers are known as aphasias.

Aphasia may be defined 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.

Cortical motor aphasia depends on a lesion in the motor speech center *M*. The patient cannot speak spontaneously and cannot speak words. Reading, spontaneous writing and writing from dictation are also encroached upon as both reading and writing depend upon the integrity of the motor and sensory speech centers. Copying is possible. Cortical sensory aphasia (Wernicke's) is caused by a lesion in the sensory speech center *A*. Understanding is lost, also repetition, reading and writing from dictation, while spontaneous writing, copying

and speaking are retained. The latter shows itself in paraphasia.

Subcortical Aphasias.—Motor subcortical or pure word mutism occurs in a lesion between Broca's center *M* and the speech muscle center *m*. Inner speech remains intact; reading, writing and speech understanding are retained, while speaking, repetition and reading aloud are lost. In the case of subcortical sensory aphasia (pure word deafness) which is very rare, the lesion is in the path from *a* to *A*. Inner speech is intact; reading and writing are possible, speech understanding is not. Total aphasia means destruction of both centers *M* and *A*. Transcortical motor aphasia means a breach between *M* and conception center *B*, and shows loss of voluntary speech and writing. Transcortical sensory aphasia means a breach in the path from the sensory center *A* to the conception center *B* and shows a loss of understanding of speech and writing. Conduction aphasia means a lesion in the paths between *M* and *A*. Repetition is affected while the understanding of speech and writing and copying are possible. Speaking and writing are retained but in the form of paraphasia and paragraphia.

Aphasias of individual senses.—Optical aphasia where the patient cannot tell objects

by seeing them but can through the other senses. Alexia is a symptom complex in which the patient is blind to letters and words and may be combined with agraphia when a lesion is in the neighborhood of the optical speech center. Amusia, or inability to sing or comprehend melodies, and amimia, which shows loss of ability to give mimic expression, are due to lesions in the main centripetal path from the motor speech center which leads also to disturbances of articulation: dysarthrias. Such disturbances may be organic or functional and if they are dependent on congenital or acquired defects or lesions of the external organs of articulation and their motor nerves, they are known as dyslabias.

CHAPTER III

SPEECH DISTURBANCES IN THE CHILD

STUTTERING.—Villiger says that stuttering occurs in from 1 to 1½ per cent. of school children and that this defect is peculiarly a disturbance of speech and is seen usually in children who are attending school. He described two types, one in which a tonic spasm of the speech musculature occurs, resulting in an extreme difficulty in initiating the word, beginning with a prolonged pause with stumbling before the word can be started. This initial prolonged stumbling having been overcome, the rest of the word is said quickly; for instance, in pronouncing the word "day" there is a prolonged tonic struggle with the letter *d* followed by a more rapid pronunciation of the rest of the word. The other type of stuttering is caused by clonic spasms in the speech musculature resulting in a frequent repetition of the initial letter or syllable of the word; for instance, if again we use the word "day" as an illustration, the *d* would be repeated half a dozen times in short spasmodic gasp-like sounds. The difficulty seems

to be mostly with consonants although vowels at times are also spoken with stumbling Stutterers can usually whisper or sing without impediment. Numerous associated movements occur in the stutterer during the tonic or clonic movements, such as wrinkling of the muscles of the face, twitching of the extremities of the body, wrinkling of the forehead, blinking, movements of the lower jaw, etc. There may also be disturbances in breathing or swallowing. The mechanism of the causation of this is ascribed by this author to a stage in the development of speech in the child when there is a dissociation between the child's desire to speak and its skill in producing sounds. The child, in attempting to overcome this, often blurts out sounds and repeats them in the attempt to speak quickly. If such a child is not taught to speak slowly, the defect grows until it cannot be removed. Slow speech and repetition in children should not be too hastily condemned, for it may be an attempt on the child's part to speak correctly, and should be encouraged in most cases. It is very important in this stage of speech development not to destroy the child's self-confidence. This period of disproportion between the desire and the ability to speak usually begins about the third or fourth year and grows much worse

throughout the school period; or it may be brought out for the first time when the child attends school. Bright children are more apt to be the victims of stuttering because they are conscious of the natural defects of their speech and more sensitive to ridicule. Expressions of amusement or actual ridicule on the part of adults at this time increase the fear and self-conscious feeling in the children and lead to a loss of attention and interest and often to backwardness. Villiger says that masturbation may occur coincidently with stuttering and is injurious to the proper development of speech because the nervous system is unfavorably influenced and feelings of anxiety concerning the habit add to the difficulty. Some children gain stuttering by imitation. This defect may be hereditary and alcohol may predispose. Boys more naturally suffer from it than girls. Injuries to the skull, excitements and infectious diseases predispose. Kussmaul says that stuttering is a spastic coördination neurosis of functional character. The treatment recommended for overcoming this defect is systematic speech exercises, psychic treatment, removal of anxiety and awakening of self-confidence.

Another form of stuttering for which the proper English equivalent could not be named

but which Villiger calls "poltern" is characterized by over-haste and swallowing of words or syllables so that language is unintelligible. This distinction is drawn between stuttering and these cases of "poltern." The defect is overcome if the child's attention is called to it whereas in stuttering it is made worse by this procedure.

STAMMERING.—This is ascribed to a faulty development of speech during the lalling period when the child is learning imitation. Individual sounds are spoken incorrectly and repeated by the child who mixes some of the sounds which it cannot speak with others or leaves them out entirely. If this defect is not corrected, we have functional stammering which is different from the stammering due to organic disease of the organs of speech; that is, the larynx, palate, pharynx, jaw, teeth, tongue and lips. Villiger insists that stammering is a functional disease and is an arrest of speech development. Stammerers vary usually in their manifestations of speech defect. When pronunciation of the letter *r* is disturbed, it is technically known as rhotacism; when the letter *r* is interchanged with other letters, particularly *l*, *w* and *g*, it is pararhotacism. Faulty construction of *l* is lambdacism, whereas the substitution

of *l* for other sounds, usually *d*, *t*, *s*, *j* or *n*, is paralambdacism. Faulty expressions of *s* and its interchange with other sounds are seen exceptionally and are known as sigmatism and parasigmatism and in certain forms as lisping. Difficulty with the expression and interchange of the letters *g* and *k* with *d* and *t* are known as gammacism and paragammacism. To summarize, stammering then is faulty expression. Stuttering consists in getting stuck and halting at certain sounds. A sufferer from stammering speaks fluently but does not express himself correctly. This defect often disappears in the first school years without treatment. A congenital form of stammering is recognized, the cause of which is unknown and is usually associated with other central developmental retardations or defects of intelligence.

Agrammatism.—When the child first learns to speak he, of course, does not speak grammatically, but if this persists after the fourth year, a real speech defect must be said to be present. Such children cannot speak sentences at all and use no inflection or connections; for instance, "soup eat, garden go." This is found in idiots and greatly retarded children, such as deaf mutes or stammerers. The defect may be in optic, acoustic, tactile or motor

spheres, or in disturbances of attention and memory. Sense perceptions leave few traces in the memory, and these children show want of skill in the motor system of the body besides the speech musculature. It will be noticed in this connection that Villiger gives no, or only a very meager suspicion, as to the clinical defects from which these children suffer. In other words, we are not told whether the difficulty is in the cerebral motor functions or in the cerebellar motor functions or in both. A mild form of agrammatism wherein the memory and attention are better developed but in which psychic defects can still be said to be present, shows itself in deficient inflection and the absence of syntax, and a peculiar arrangement of words. In still another form, the child can speak spontaneous sentences but one has difficulty in making them out. These patients are likened to foreigners who are learning a strange language. However, they can repeat correctly simple sentences. This type occurs in children between the ages of ten and fifteen years and depends on articulatory disturbances of long duration in part organic and in part functional.

Hearing Mutism.—This may occur despite the presence of normal hearing and normal

speech apparatus, together with a sufficiently well developed psychic function. Coën in 1888 called attention to this, although it was formerly known as congenital aphasia and mutism without deafness. This condition depends on a central retardation of development of speech; the pathology and etiology is little known. The presence of neuropathic taint in the families in which this defect occurs is postulated. Alcoholism is again suspected as being a baneful influence in the production of this defect. Adenoids and tonsils may contribute, together with inadequate development of collective motor functions. According to Gutzman, this symptom complex is due to a mal-development of the associative speech mechanisms. It will be recalled that the incidence of imitation shows itself in general movements and in special speech movements. In some children this incident appears rather early, in others late. These last also learn to walk late and are awkward, and often want of skill shows itself also in the movements of the muscles of speech. This want of skill is overcome by constant usage and exercise. Villiger again reminds that understanding produces actual speaking and that the desire to speak may not be in proper relation to the ability of the child to speak and so repeated efforts to deliver words

result in failure, the child becomes discouraged and gives up and remains dumb through fear. Hearing mutes are said to be normal mentally, but it has been shown that the intelligence has suffered and that in these cases attention and memory are faulty. Out of Villiger's own words, therefore, we have evidence which would tend to show that the entire development of the speech mechanism goes hand in hand with the development of intelligence in the child and that it may be perfectly possible for their evolution to occur at the same time and may even possibly be an identical process. As will be brought out later, there seems to be abundant evidence to support this view, for in a great number of clinical states wherein speech is affected, there is a corresponding defect in the mentality or intelligence. It will be noticed, too, that those children whom Villiger calls hearing mutes, have speech defects despite the presence of absolutely intact hearing mechanisms which Villiger, together with others, have insisted were the most important pathways for the proper development of the speech function. In some cases, which are more probably cases of deaf-mutism, acoustic defects are observed or visual defects may occur together. For instance, with the eyes shut, these children do not well differen-

tiate sounds which are heard and may not be able to localize them. Pictorial representations and the memory for form and color may also be defective. Spacial representation is faulty and defects may be found in the motor and tactile spheres. Hearing mutes cannot tell with the eyes shut whether things placed in their hands are light or heavy, dull or sharp, and on the motor side they are awkward in general movements, particularly those of the tongue and lips. The memory is poor and they are not able to reproduce from memory movements which have been shown them only a short time previous. It may be relevant here to call attention to the fact that these cases are also called apraxia by some authors. Taste and smell are better preserved. It may be that those cases which Villiger has called hearing mutism correspond to types of ideational and motor apraxia occurring in defective children as we understand them in this country. The author continues by classing these cases of hearing mutism under the heading of aphasia. First, the motor form which is the most common and corresponds to motor aphasias. Such children speak nothing or single words only, but they understand everything. Second, the sensory form corresponds to cortical sensory aphasia. Such children hear but do not under-

stand. This is psychic deafness. Hence, they do not learn to speak although they can imitate a little and repeat. They understand gestures and obey commands. These cases are comparatively rare and may be mistaken for deaf-mutes. A third form with both motor and sensory defects shows itself in children who understand single words but not complete sentences. Children with hearing mutism should be carefully studied so as not to mistake them for deaf-mutes because of the difference in the prognosis. In deaf-mutes the prognosis is not nearly so good as in cases of hearing mutism, who can, with careful training, be greatly benefited.

Congenital Word Blindness.—This consists in the ability to read single letters and figures but the inability to combine them into words, and exists in one out of every 2,000 school children. It is more common in boys. The lesion is placed in the angular gyrus. It is usually not severe and is due to a retarded development of the visual speech centers. The verbal alexia which results is comparable to that found in pathological cases in adults. The condition is first recognized at school and is not detected earlier, because they learn to read from memory and often copy well, but when

told to read backward or to write down the names of familiar objects they are unable to do so. Repeatedly, one finds striking forgetfulness, such as the inability to carry out orders for purchases. Here, besides the inability to read words, there is the added inability to read letters which are in a vertical plane; notes made in a continuous horizontal line can be read. Early recognition and persistent teaching may bring about great improvement. It is on record that men born word-blind have composed scientific works, though, to be sure, the manuscripts had to be revised and corrected. These children should not be put in special classes for defectives. Assistance outside of school hours may be given besides the regular school work. Blocks may be employed in the study of writing and reading. Marburg suggests the training of the left hand for writing in order to develop the reading center in the opposite hemisphere.

Deaf-mutism and Hardness of Hearing. —Congenital or acquired deafness or hardness of hearing leads to corresponding deaf-mutism. Deafness acquired before the seventh or eighth year leads to mutism. Boys are more naturally affected. Congenital cases occur more commonly in the female, acquired cases in the male.

Hereditary defects affect more the female children, and children's diseases more commonly strike the auditory apparatus of the male. The United States shows fewest and Switzerland the most, deaf-mutes. Mountainous countries where endemic cretinism is common show a great number of deaf-mutes. Jews also show a large proportion. Heredity plays a part, but most often not direct, but collateral, heredity. Several children of one family are often affected. Consanguinity may be a cause. Alcoholism, syphilis, mental diseases, weak-mindedness and squint or weakness of sight in the ascendants are said to be causes. Scarlet fever and meningitis account for most of the cases of acquired deaf-mutism. Other infectious diseases, otitis media, or fracture of the skull are responsible for individual cases. Those diseases which affect hearing usually occur during the first or second year. In the congenital form only a few pathological changes have been shown in the cochlea, while in the acquired forms distinct disturbances have been observed. Deaf-mutes also show other bodily defects; mental defectiveness and retinitis pigmentosa have also been found. Bezold has shown that in many congenital and in some acquired, forms of disfunction there is still some hearing left. He showed that only a limited part of the

scale is necessary for hearing speech (*B—G*). On enunciating clearly and loudly directly into the ear, he found that deaf-mutes can learn to speak; in addition to this, he has the patient watch the mouth. The better to do this, a mirror is placed in front of the deaf-mute and he is asked to observe the movements of the lips of the teacher as he speaks into the patient's ear. Children who have learned to speak but have become deaf before the age of seven, may become deaf-mutes. Deafness or hardness of hearing acquired after the age of seven leads to unclear and monotonous speech. Such children learn to depend more and more on signs and gestures and their vocabulary becomes limited. It has also been shown that in high grades of deafness, there is an accompanying mental retardation. On the other hand, in mental defectives who are also hard of hearing, it is difficult to say whether the mental defectiveness or the hardness of hearing is the cause of the mental retardation.

CHAPTER IV

FALLACY OF THE DIAGRAMMATIC CONCEPTION

Lest it should seem that the abstract of Villiger's work already given has been in any way a definite statement entirely or even partially satisfactory regarding speech disturbances or speech development, we must proceed to show the fallacy of such a belief.

Head[9] starts his introduction to Hughlings-Jackson's work on aphasia and affections of speech by saying:

"It is generally conceded that the views on aphasia and analogous disturbances of speech found in the text-books of to-day are of little help in understanding an actual case of disease. 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."

The dissatisfaction which Head shows by this statement concerning the type of conceptions of aphasia and speech disturbances as described by Villiger was shown even more forcibly by Pierre Marie[22] who in 1906 set about entirely rearranging our conceptions of this subject.

Before this, Hughlings-Jackson[15] had stated that "whilst I believe that the hinder part of the third left frontal convolution is the part most often damaged, I do not localize speech in any such small part of the brain. To locate the damage which destroys speech, and to localize speech are two different things. The damage is in my experience always in the region of the corpus striatum." This statement of Jackson as pointed out by Head is strangely in sympathy with Marie's localization of the lesion in aphasia in the quadrilateral area bounded by that author. Another worker on this subject, James Collier[5], expresses his dissatisfaction with the handling of this subject in the following words:

"The many schemes with their accompanying diagrams of the localization and intercon-

nections of the speech-centers, that have been brought forward by high authorities upon this subject, have been founded more upon theory than upon trustworthy pathological evidence; upon these schemes classifications of the clinical varieties of aphasias have been based, which from the first have been open to the criticism that varieties of aphasia were included and described which were purely hypothetical and which had not been met with clinically. These were types of aphasia which should have resulted from lesions in certain situations, had the scheme which gave them birth been a correct representation of the physiological mechanism of speech. The multiplicity and complicity of the schemes and diagrams that have been brought forward are in themselves a proof of the difficulties that have arisen in the attempt to apply them to clinical cases."

Clinical studies of this subject, together with correlated pathological research, have failed to clear up the many problems involved. The area for motor speech in the third left frontal convolution, the auditory sensory speech area of Wernicke in the temporal lobe, and the visual psychic area in the angular gyrus, together with areas added later for writing in the second left frontal and an area embracing

both frontal lobes as the seat of the concept center, all of them have been upheld by various writers reporting clinical and pathological material to sustain the individual views of the presenter.

Nevertheless, the concepts were so unconvincing, and the clinical and pathological material failed so signally to clear the situation, that Pierre Marie[22] demanded a revision of the whole subject of aphasia. He desired to sweep away the majority of preëxisting ideas upon the subject, and he railed against the conceptions advanced that speech memories are registered in the cortex as visual word memories, auditory word memories and motor word memories. He denied the existence of any of these centers, including the visual speech center in the angular gyrus or the supramarginal convolution, the Wernicke center in the posterior two-thirds of the left second temporal convolution, and he denied absolutely that the left third frontal gyrus had any function whatsoever in connection with the speech mechanism. He recognized no distinction between motor and sensory aphasias, and he argued that there was only one speech center diffusely localized in the left temporoparietal lobe and that this center is a region of intelligence specialized for language and not solely a

center of sensory images. He insisted that the so-called word blindness and word deafness are defects of a special intelligence for speech. He recognized only one variety of aphasia from a lesion of the cortex and this is the type which is usually called the sensory aphasia of Wernicke. This type, he stated, always resulted from a destruction of the region of specialized intelligence for language situated in what he called Wernicke's zone.

The only other types of aphasia which existed for Marie were anarthria and pure alexia, which were always due, according to him, to subcortical lesions. The motor aphasia of Broca he regarded as a combination of aphasia with anarthria, due to a double lesion, one in the special speech intelligence area above mentioned and the other occurring within a quadrilateral area surrounding the lenticular nucleus, which area determined the anarthria. This quadrilateral area is limited anteriorly and posteriorly by vertical frontal planes which are level with the anterior and posterior limiting sulci of the Island of Reil. The outer limit is the surface of the insula and the inner limit is the wall of the lateral ventricle. Above, this quadrilateral area was prolonged to include the overlying gyri of the convexity of the cortex, while below it included the subthalamic region. The motor

aphasia of Broca which he designated anarthria is usually due to a lesion in the upper two-thirds of this quadrilateral area which is in connection with the third frontal convolution, but contains no part of it and is in connection with Wernicke's zone also. That Marie certainly had sufficient reason to call for a revision of the whole situation as it touched our conceptions of aphasia, would appear from the highly important review by his associate Moutier[23], whose statistics are worth quoting:

"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."

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. Indeed, workers in this country, Frankel and Onuf, analyzed 104 cases of aphasia with autopsy and met with only 4 cases which seemed to confirm Broca's localization; but at the same time they examined 5 cases of Broca's aphasia (motor aphasia) in which the left third frontal convolution was absolutely intact and the fibers going to and coming from this part of the cortex showed no trace of degeneration. A surgeon, Burckhardts[2], operated on a patient removing 5 grams of the gray matter from the foot of the first and second left temporal gyri and no word deafness (sensory aphasia) resulted. Eight months later, in the

same patient, he resected the top and foot of the left third frontal convolution and no aphasia or agraphia resulted. In another case, the same surgeon removed a part of the left supramarginal convolution and destroyed a considerable portion of Wernicke's zone and no aphasia or any sort of speech defect resulted. In this last patient, the left third frontal convolution was also resected.

The operations in both these cases were performed with the deliberate intention of producing auditory and motor aphasia to counteract auditory hallucinations and over-activity of speech in two insane patients. In this connection, during the summer, I had an opportunity to examine a patient operated on by Guadiani at the Italian Hospital for a supposed abscess of the brain. The left temporal lobe was bored and punctured in several places. In the course of the convalescence some cortical tissue necrosed and three pieces, one as large as a five-cent piece, sloughed away at subsequent dressings. For a few days following the removal of the largest slough the patient was somewhat paraphasic and made mistakes, occasionally choosing the wrong word during conversation. He had no alexia, no agraphia, spoke fluently and spontaneously and from dictation, and could recognize, number and

name objects and write their use without difficulty. The only trouble from which he suffered was that occasionally in the course of a conversation he would misuse a word or would stop for several seconds attempting to find the right word to express his meaning properly. The paraphasia in this individual lasted about three weeks. Just before he left the hospital I saw him again and could find no defect whatsoever. Despite all this, there is considerable feeling, even amongst the recognized authorities, that Wernicke's area and Broca's area and the area for psychic vision are intimately involved in the mechanism of speech production, as the following quotation from Dejerine[6] will show:

"Dejerine, Ladame et von Monakow, Liepmann et Quensel, Mahaim, Dejerine et Thomas—ont montré que cette circonvolution faisait bien partie du centre cortical du langage articulé comme cela était admis jusqu'alors, et la même démonstration a encore été faite sur le vivant par la chirurgie cranio-cérébrale. Ce que l'on discute toujours et ce que l'on discutera encore longtemps, c'est de savoir is le centre du langage articulé est étroitement limité á la circonvolution de Broca ou si, ce qui pour moi est plus que probable, il n'emiéte pas sur les régions voisines."

Enough has been shown even in the above quotation from Dejerine, to destroy any faith whatsoever in the conception which relegates the whole or any considerable part of that special skilled act, which we call speech, to definitely or diffusely circumscribed localizations in the cortex. It appears possible, however, that on the motor side the center for the innervation of the actual muscles concerned in the production of sounds for the service of speech, is situated in the third left frontal convolution near the areas for the larynx, pharynx, lips and mouth, and that they serve for the discharge of motor impulses cotemporaneously with the necessity for the expression of thought, which is speech.

A clinical report is hereby given of injury to the left temperosphenoidal lobe with recovery of speech function after slight temporary loss which may have been due to fatigue, to shock following operation or to the diaschisis occurring according to the theory of von Monakow, but surely not to loss of cortex substance as the absolute recovery seems to show. The questions used in testing are the work of Dr. Tilney and are employed by him in the Vanderbilt Clinic and in private work. A copy follows. The first report is of the examination ten days after the injury caused by operation and

the second about three months after the injury:

Question 1—Can the patient speak spontaneously in ordinary conversation? (What is your trouble?)

Question 2—Can he enumerate and denominate, number and name objects? (Can you count your fingers aloud? Can you name the members of your family or the days of the week? Can you name these objects?)

Question 3—Can he speak from dictation? (Can you repeat, "Now is the time for every good man to come to the aid of his party?")

Question 4—Can he read aloud?

Question 5—Can he read to himself and understand what he reads?

Question 6—Can he recite what he reads or what he has previously learned?

Question 7—Can he write spontaneously?

Question 8—Can he enumerate and denominate in writing? (Can you write the numbers from 1 to 10? Can you add up this column of figures? Can you write the days of the week? Can you write the names of these objects which are shown to you?)

Question 9—Can he write from dictation?

Question 10—Can he copy printed or written matter?

Question 11—Can he hear and understand what he hears?

Question 12—Or sees?

Question 13—Is he aware of such errors as he may make in speaking or writing?

Question 14—Has he a concept of his own speech?

Question 15—Does he misuse words? Is he ungrammatical out of proportion to his education? Does he perseverate in fixed phrases? Does he speak a jargon?

J. de B., male, 46 years of age, Italian. Married, has five children.

He had an ordinary grammar school education in Italy and while there went one year to high school. He spoke and read Italian well, and was a prolific reader of Italian newspapers and novels. Since 14 years of age he has read no literature or scientific works of any kind. His education, therefore, is that of an average Italian youth of the middle classes. He speaks and writes English poorly and has never been educated in American schools, although he gets along fairly well with English. The examination has been conducted entirely in Italian.

Personal History.—He is right-handed and all his family are right-handed. Three years ago he suffered a small chancre for

which he had nothing but local treatment. He has received no constitutional treatment for this condition since.

On April 10, 1917, he had a severe ache in the left ear with a slight fever. A day or two later the drum was incised and the ear discharged continuously for two months. At the end of this time the discharge ceased for three weeks, and his earaches and all the ear symptoms disappeared entirely for this period. At the end of July, 1917, he had another attack of earache in the same ear, together with which he had a pain in the head about the left ear within a radius of two or three inches. The headache continued with increasing severity, and finally his whole head pained him keenly. By the first of September, he had daily fever, never more than 101. He vomited for the first time during the month of July and this vomiting was attributed to some medicine which was being given. Toward the end of July, he had internal strabismus with diplopia in the left eye. He had at no time, until just following the operation, any disturbance of speech whatsoever. He was brought to New York to see his physician, Doctor Gaudiani, who considered that he might have an abscess in the brain or a brain tumor.

I was called in consultation August 29,

1917, and the examination at this time showed a double choked disc with swelling to the extent of 4 diopters in the left eye and 3 diopters in the right, left internal strabismus, tremendously severe headaches with tenderness, localized only poorly over the left side of the head, particularly marked on percussion over the left parietal bone and the occiput on that side. There was diplopia but no limitation of vision. There was slight deviation of the tongue to the left, and slight weakness in the muscles of the left side of the face. Taste in the anterior two-thirds of the tongue was preserved and there was no disturbance of function in any of the other cranial nerves. Sensation in the 5th distribution was normal. The superficial and deep reflexes were all active and equal, although to muscle testing at the wrist, elbow, shoulder, hip, knee and ankle on the right side as compared with the left, there was a marked weakness. Later on, this weakness became more marked and there was distinct pyramidal tract involvement with diminished superficial reflexes on the right side and more active deep tendon reflexes on that side. The headaches continued. There was no vomiting, no ataxia, no adiadochokinesis, no tremor, no convulsions, nor irregular, involuntary movements. The sphincters were well controlled.

Mentally, the patient was clear and there was no apraxia or aphasia. Despite my diagnosis of possible meningitic luetic process at the base of the brain, an operation was performed because of the antecedent ear condition. At the operation, no abscess or tumor was found, although a considerable amount of fluid gushed out when the dura was incised. Several deep punctures in the temporosphenoidal lobes in each of the convolutions exposed were made, and at subsequent dressings of the wound, three sloughs, two small ones and a large one about the size of a quarter, came away from the middle third of the second and third left temporal convolutions. Immediately following the operation, the patient's speech was absolutely well preserved, but one week later, September 18, 1917, he became paraphasic and an examination of aphasic status at that time, according to our scheme of 15 questions, is detailed as follows:

1. "I have a pain in the head. It is much better since the operation. I am talking foolishly. I use the wrong words although I know what I want to say. It is laughable."

2. Counts aloud correctly up to ten. Does simple and even fairly complicated arithmetical calculations. Numbers and names objects

correctly although on three or four occasions he said, for instance, "bed" for chair, and "writing" for fountain pen, and used the word "doctor" for nurse (dottoressa), which in English means female doctor. This was said in answer to the question as to who the nurse was. At times, too, he said his oldest daughter's name for his wife's name.

3. Repeats any number of test phrases accurately which are read to him from newspapers or will repeat long paragraphs fairly correctly, excepting that he will say "guerra" (war) for "battaglia." At times he becomes confused and will cry, and as a result his utterances for a few seconds will be very paraphasic. Words are misused and interchanged and ungrammatically arranged, but he soon recovers and speaks again with only an occasional misuse of a word.

4. He reads aloud correctly and without defect.

5. He reads to himself and gives a fair account, in his own words, of what he has read, occasionally only transposing one word or two in several long sentences. For instance, the last syllable of the word "presidente" is used alone or before the first syllable so that he will say instead of presidente, "dentoprese," or as he did on one occasion, "presidere." This does

not happen very often and at times he will carry on conversation for several minutes without making any mistakes in speech. He often forgets his wife's name. I was reminded at the time of the similarity of his speech disturbances to those which are seen in fatigue. It must be remembered that this patient had suffered a great deal of physical and mental stress for several months and was undoubtedly exhausted, not only physically but mentally.

6. He recites perfectly what he reads, or test phrases that have been taught him.

7. He writes well spontaneously, except that he might use the same word several times in succession, and his grammatical construction is poor. Nevertheless, there is no jargon and the sense of what he wishes to say is very evident. As his writing is entirely in Italian I am not giving a sample of it.

8. He writes the numbers from one to any denomination. Adds up columns of figures correctly, writes the days of the week correctly, except that Sunday is written "domani" (tomorrow) instead of "domenica" which is proper. He wrote without any trouble or mistakes the names and uses of the objects shown him.

9. He writes well from dictation.

10. He copies perfectly printed or written matter and understands it.

11. He hears and understands what he hears and one must remember, particularly in connection with this entire examination, that this patient is very deaf as a result of his organic middle ear trouble. One is not always absolutely sure that he has distinctly heard what has been said to him. This seems to be proved from the fact that he obeys much more promptly and correctly written commands and answers written questions more correctly than he does spoken commands or questions.

12. He sees perfectly and understands everything that he sees and recognizes the use of objects shown him.

13. He is aware of such errors as he makes in speaking (in writing he makes none or exceedingly few), and becomes greatly confused and mortified and extremely emotional when he does make mistakes. He seems to feel that fun is being poked at him and will cry copiously after a mistake.

14. The number of syllables and letters in any number of words are perfectly recognized, particularly if written. The patient realizes that he makes an occasional mistake in speech and hastens to explain that he does not hear at all well and is tired and confused and has

a profound headache, although he says, too, that the headache is not so bad as it was before the operation.

15. He misuses words in a paraphasic manner, although only occasionally, and in spoken speech he is much more ungrammatical than he is in written speech. There is a tendency to repeat phrases in writing or speaking, but in neither is there a jargon.

His few difficulties in speech cleared up remarkably rapidly. I felt at the time of his examination, and feel even more strongly now, that a great deal of his paraphasic trouble was due to fatigue, pain, exhaustion and actual difficulty in hearing. The ensuing examination made December 15, 1917, seems to bear this out:

1. "I feel pretty well excepting for an occasional slight headache. I still see two objects. I am very much hard of hearing. I can hear better than I did before the operation, particularly in my right ear. My left ear is the worse, but I can hear a little bit in it, whereas before the operation, I could not hear at all. My dressings (he still wears a bandage over the site of the operation) are always wet with fluid which comes from my head."

2. Gives correctly the names of his wife and five children, numbers and names objects in

the room, gives the days of the weeks, counts the fingers on both hands, aloud, absolutely correctly.

3. Answered perfectly.

4. Answered perfectly.

5. Answered perfectly.

6. Answered perfectly.

7. Done perfectly: "Nel giornale della parola del Dottore, ho trovato che parlano di fare qualche cosa di benificienza pel Hospedale Italiano." *Translation*—In the Italian Medical Journal I find that they speak of attempting to aid in some way the Italian Hospital. Correct.

8. Well done.

9. Done perfectly. "Io sono de Benedictis chi ha sofferto molto nella malattia dell'orrechio." *Translation*—I am de Benedictis who has suffered so much from sickness in the ear.

10. Well done.

11. Hears and recognizes watch, obeys perfectly spoken commands and many complicated directions of various kinds.

12. Sees well, has no hemianopsia and no limitation of vision, but has suffered from diplopia due to paralysis of the external rectus of the left eye.

13. He makes no errors either in speaking or writing except as a result of his profound deafness.

14. Lichtheim's test (Presidente, 10 letters, 4 syllables), correct. Ospedale (4 syllables, 8 letters), correct. He offers the information voluntarily that he is so hard of hearing and is so sensitive about it that sometimes he will not wait to make sure of what he has heard, but he is extremely intelligent and quick to answer when he hears well.

15. He does not misuse words, except that once or twice during the conversation at this examination when the title of the periodical which was used in the test, "La Parola del Medico" was mentioned, he said: "La Parola del Dottora." Dottore means just exactly what Medico means, so that this may not be a paraphasic manifestation. Before today he had never heard of the periodical mentioned. He speaks fairly grammatically, as he always did, never having been a perfect user of Italian. He does not perseverate, although quite naturally his thoughts as expressed in speech and writing are apt to turn towards his own difficulties, particularly his hardness of hearing. He speaks quite fluently and lucidly and not in a jargon.

CHAPTER V

APHASIA AS AN INTELLIGENCE DEFECT

Hughlings-Jackson[12] always insisted that speech "is a part of thought and that in conditions of aphasia thought processes of all kinds were involved." Von Monakow,[34] in illustrating his theory of diaschisis, assigns a different interpretation to those defects of speech which are seen after a lesion and which he says are slight in degree and soon pass off, and those which remain well marked and permanent. The early symptoms, which are transitory, are due to a lowering of functional activity in a distinct part of the speech mechanism caused by the upsetting of balance between the several parts of the mechanism produced by the destruction of one of the integral parts by the lesion. He insists that the nervous system acts as a whole and so with the speech mechanism. That is, if in a case of motor aphasia, Broca's area is destroyed, the independent areas which von Monakow believes exist, according to the old diagrammatical scheme of localization, also suffer, and so a lesion in the motor speech area would produce

a depression of function in the visual, auditory and other speech areas and thus cause the resultant pronounced aphasia. Von Monakow believes with Marie[22], Grosset and others that the aphasia is simply a defect of intelligence as originally held by Trousseau. In fact, Marie lays great stress upon the intellectual defects shown by patients with aphasia and insists that those who deny this viewpoint have neglected to use even the simplest of intelligence tests. He states that he has been able to prove beyond doubt the obvious lowering of the intellectual level in every case of aphasia under his observation. This author cites numerous cases, not of speech intelligence loss, but of actual defects of general intelligence which can be found in every aphasic. We have as illustrative of this point the following clinical cases:

M. A., female, aged 33 years, Italian. Married, has two children. On August 31, 1917, while at home at her work of dressmaking, she suddenly found that she had lost her power of speech. She did not fall and was assisted to bed. There was no weakness of the extremities but her mouth twitched and was drawn towards the left. She slept for three or four hours, then arose and found that she could say only "Dio"

and "che cosa." Her husband asked her whether she knew him. She shook her head and could not respond.

She came to the Vanderbilt Clinic September 19, 1917, and it was thought that she might have a psychosis of some kind. On account of her inability to speak English she was turned over to me and after due study, the proper diagnosis was made. The only point of importance in the history is that she has suffered from valvular heart disease for ten years and has had to have two induced abortions on account of this condition. She had felt perfectly well physically before the onset of her illness excepting that her heart continued to give her trouble. Since her illness her mood has been depressed; she seems to be bewildered, dull and indifferent, will sit for hours doing nothing, and will not speak unless spoken to. Her thought content is hypochondriacal. She says that she is never going to get well, that she is unhappy and wants to die, and she refers frequently to her desires about returning home to her parents and children in Italy. She repeats continuously: "Here in this country there is plenty, but there in Italy my parents and children are starving." She will not, or cannot, do any housework but eats very well and sleeps uninterruptedly.

The aphasic status taken on September 21st, is as follows:

"Io mi chiamo Ansaldi Marguerite e son de trenta-tre anni il mio marito lavoro come cuoco nella cuilina di un hotel Americano."

Answer: "Io mi chiami Anan ansaldi margurrtr e nari di—" A hopeless jargon—does not understand what she reads at all. Cannot copy from writing.

Question: "How many letters are in the word 'Presidente'?" She answers 9, and is not able to answer at all unless she is allowed to look at the word. Asked to write what is the trouble with her, she writes jargon: "Silisena lare serlal nesse el nosilanenessto nelano—" (Jargon).

Asked to subtract 7 from 10, she answers 6, even when shown the problem on paper. Asked to subtract 5 from 10, she says 5, but when asked if she is sure she hesitates and says "I do not know." Asked to write the days of the week, she writes as follows: "selene, nestani, nerteni, lenerti, selani, enonenito—" (Writes all words wrong).

October 19, 1917.

Question: "What is your trouble?" Answer: "I feel weak in the heart. I can't read much and do not understand what I read." Counts fingers and gives the days of the week. Reads

mechanically and with many mistakes. Does not understand what she reads and is not able at all to give in her own words the substance of any written or spoken paragraph. Cannot write spontaneously, and makes many mistakes in writing from dictation and from copy. She hears and understands a great deal of what is told her but often makes a mistake and is not able to correct it. Has to be told several times to get up and open the door, to sit down, etc., before she understands. Lichtheim's test is well done. She can tell the number of letters in the word but occasionally will fall down on the syllables, although she insists that she knows what syllables are. Grammatical mistakes are made only in writing or reading, very rarely in speaking spontaneously. She remembers and names objects. There are alexia and agraphia.

November 28, 1917.

She is asked to read the headlines in an Italian paper which show that the Italians are fighting bravely on the Piave line and are holding the Brenta sector although they are outnumbered five to one. The article deals with the heroic resistance that the Italians are making successfully against their enemies. In asking her, first, to speak spontaneously her conception of what has been read to her, she

gives a satisfactory, although a very meagre abstract, but in writing, although I waited patiently for her to answer for more than a half hour, she never got further than the phrase "Gli Italiano," which is incorrectly spelled and means The Italians. She said that she was able to understand thoroughly what she wanted to say and she made the motions of writing perfectly, as can be seen in her broken phrase just mentioned, but found it absolutely impossible to reproduce what she wanted to say. This, despite the fact that there was no paralysis in any part of the patient's body at any time during the long period she was under observation. It is truly remarkable the extent of the damage which was done to this patient's brain without producing paralysis. She continues to improve in her spontaneous speech, making fewer errors, but occasionally finding it very difficult to pick out the word. On such occasions she often substitutes an irrelevant word or phrase for the one she wishes to use. A steady improvement is taking place which is most noticeable in her speech and in reading. She reads a good deal better but still makes a great number of mistakes which, however, she occasionally recognizes. She makes but poor progress in writing and still is as bad as she ever was.

A.M., male, aged 19 years, single. Italian.

The patient had bronchopneumonia three years ago. The left iris was operated on for cataract when he was a baby and on three subsequent occasions. Aside from these two incidents, the patient had been perfectly well until April 14, 1917, when he was operated on for appendicitis at St. Catherine's Hospital, Brooklyn. Three days later he became paralyzed on the right side of the face, in the arm and leg, and became unable to speak. About two years ago, the boy is said to have had an epileptic seizure. The attack was very severe and was repeated about one year ago. These two epileptiform convulsions were not followed by speech defects or by paralysis, and have not been repeated to date.

The patient is the eldest of eight children; all of the seven are well and the father and mother are both living and well. The mother had two non-induced miscarriages.

In order to understand the aphasic status of this patient, it is necessary to know that he went to school at six, attending the public school until he was 16 years of age. When he left he was in 7-A, and, although 16 years of age, he did not graduate and would not have graduated until six months later had he stayed in school. He was not promoted on three

occasions, partly because he did not attend regularly, and partly because of some trouble with his left eye, which was operated on and kept him out of school at various times almost continuously for three or four months. Aside from this, however, the boy was a truant and was unmanageable in so far as his attention to school duties was concerned. He was always bright and keen and picked things up easily, read a great deal of boys' fiction and newspapers, but nothing serious. He wrote a fine hand and was able to compose a fair letter to his friends and relatives. He spoke Italian fairly well, but English was always the language of choice and was spoken much better than Italian. He never could write Italian and, of course, cannot do so now. In fact, this boy can be said to have had the average education of a boy who leaves school in the grade known as 7-A. The average age of these children is 12 years. It will be remembered that he was 16 when he left.

Four years ago he began to work in a shoe factory where he stayed for three months and then went to a marble works for seven months, and back to the shoe factory until recently. This patient, and also the one above reported, are right-handed, and have come from right-handed families.

The aphasic status, following the order of our fifteen questions, is as follows:

1. The patient cannot speak spontaneously in ordinary conversation.

2. Can be done with a defect. The defect appears to be mostly in naming objects.

3. He cannot speak from dictation.

4. He cannot read aloud.

5. He can read to himself and seems to understand what he reads.

6. The patient cannot repeat aloud what he has read, cannot say his prayers aloud, but he can sing "My Country, 'Tis of Thee" all through without a mistake in the words, but when asked to repeat the words of the song, he cannot do it.

7. He can write spontaneously but uses wrong words. For instance, when asked the question, "Please write down what troubles you have," he puts down the phrase, "I am England." When asked what this means, he replies, "I had appendicitis." When asked to write my name, he wrote, "Dr. Frisco," Teddy Roosevelt, was written "Tippery."

8. The patient can write the numbers from 1 to 10. He can add up simple columns of figures and can write the days of the week and can also write the names of various objects which

I showed him, but will often make mistakes which he frequently corrects himself.

9. The patient can write only simple things from dictation, but leaves out letters and quite often words, and in Question 10, he will do the same thing, i.e., he can copy from print but will omit words or letters.

11. The patient hears and understands what he hears.

12. He sees and understands what he sees. A peculiar reaction which should be noted is one in which I asked him to look at my watch and tell me what time it was. He could not do so in words, but he drew a diagram of a watch with 13 numbers on it, there being 2 tens, and then drew the hands of the watch on the blackboard, so that the result of the diagram presented the correct time, which happened to be five minutes to four.

13. He is aware of errors in both speaking and writing, but is not able to correct them.

14. The patient is able to tell the number of letters in a word, but does not seem able to tell the number of syllables, although he assures me he understands what syllables are. July 2—After a long explanation he finally demonstrated that Lichtheim's test is positive. He was able to pick out the number of letters and syllables in many words.

15. The patient is practically wordless. Therefore, no ungrammatical uses of words could be made out. Whatever utterances he has, and they are very few indeed, he perseverates.

History after the Onset.—This patient at first was able to say only "Yes" or "No." Soon thereafter, however, he began to say words, but spoke a hopeless jargon and was only able to give his own name and that of his father and several of his relatives. He used "Yes" and "No" properly for assent and dissent, but aside from this could not be said to have very much spontaneous speech. He has always had the power of mimicry very highly developed and can make himself understood by various means in this way. He has learned to use his left hand very quickly in writing and does fairly well. He has improved a great deal, particularly on the sensory side, being able to understand a great many more things that he sees and hears than he did at first. He still speaks and writes a hopeless jargon, however.

This patient indulges now (9 months after onset) in much silly behavior, and is the butt of a great deal of ridicule at the hands of other clinic patients because he spends most of his time trying to use his charms on the clinic

nurse. He comes to see her a great many days when it is not necessary for him to attend the clinic and has made himself so ridiculous in his attentions to her that other patients laugh at him. His grasp on school knowledge and current events is poor and he indulges in much foolish rhyming and grimacing.

J.M., male, 36 years of age, married. Irish.

History.—On Friday, December 16, 1916, this patient went to sleep at night apparently perfectly well and woke up the following morning complaining of weakness in the right arm. There had been prior to this no headache, no dizziness or numbness, and the weakness in the right arm was not accompanied by any of these symptoms. Later in the day the patient became drowsy and finally unconscious. His wife in the meantime came home from a shopping trip and found him on the floor paralyzed on his right side. His eyes were open and he seemed to recognize her, but could not speak. She states that she had been gone not more than one hour and that when she left he was not unconscious. The patient says that he was unconscious during this hour that his wife was gone, but we have no means of being absolutely sure of this point.

We are certain, however, that he was not unconscious more than one hour, if at all.

Previous History.—On March 19, 1916, our patient was struck by a street car over the right ear, contusing it and wounding it severely; the right side of the face was also contused. Immediately after this injury he complained of deafness in both ears, worse on the right side. Following the injury, he did not bleed from the nose, from the ears or into the eyes, and is said at the hospital where he was observed not to have fractured his skull.

The patient has led a very active life, has been a soldier in the English army, and has been all over the world, most of the time in tropical climates. He has been admittedly a very hard drinker. Venereal infection is denied.

In order to understand the aphasic status in this case, it is necessary to know that he has been practically illiterate, that he could read very little, and could write less, before the onset of his trouble. He was able to read simple articles in the daily newspapers with difficulty and could only write the simplest of letters. The deformity of the right pupil is congenital and has been diagnosed as a cataract. The family history is negative, the patient

not knowing the cause of death of his parents and other relatives.

1. The patient cannot answer this question.

2. These questions were answered well although there is a slight defect in the naming of the members of his family and the days of the week.

The patient failed in answering the 3rd, 4th and 5th questions.

In the 6th, he failed also. Although he can carry a tune perfectly well and gets many phrases in the song, he cannot be said to sing the words intelligibly.

In answer to question 7, the only thing the patient can do is to write his own name, and very frequently only his last name.

8. The patient can write from 1 to 10, but cannot add, cannot write the days of the week and cannot write the names of objects shown to him.

9. He cannot write from dictation. In considering these last two tests, it must be remembered that this patient is possessed of a very poor quantity and quality of education and some allowance must be made for this lack.

10. The patient practically fails in doing this test, but can copy his own name and his wife's name and very little else.

11. He hears and understands what he hears

perfectly. He recognizes the watch when placed close to the left ear, the right one being deaf. The patient's eyes were closed in the watch test. He obeys very well all spoken commands, provided they are not too complicated.

12. He recognizes everything that he sees; his answers are all correct.

13. He is well aware that he makes mistakes in speaking and writing.

14. The patient's idea of his difficulty is that he understands everything and has the mental picture of what he wants to say but cannot say it. Lichtheim's test could not be made on this patient because of the lack of education. He does not know what syllables are and, therefore, naturally could not be expected to perform this test properly, although he is able to tell the number of letters in most words, particularly words and names with which he was previously familiar before the onset of his trouble. If this question must be answered, we should have to say that the patient has a correct conception of his speech.

15. The patient occasionally misuses what few words he does utter, speaks a jargon and is prone to repeat.

This patient is subject to quick changes in mood, shows great lack of interest, drinks a good deal and is very untidy and careless about

personal habits. He is bad tempered at times but usually smiles and shows an utter disregard for the seriousness of his condition.

M. G., female, 20 years of age, married, 1 child. Italian.

Family History.—The patient's father and mother are living at 45 and 39 respectively. The father is very alcoholic and beats his wife and children, especially when he is drunk. There are seven brothers and sisters, all living and well. The patient's child seems to be normal. The husband denied any venereal infection whatsoever. The patient has been known to her husband for 19 years, and while she may never have been very bright or intelligent, she was considered a normal girl of her class and education. She had a normal, jolly and open disposition and there are no seclusive symptoms in her make-up.

History of the Onset.—On June 11, 1917, the pregnancy terminated and labor began. Several hours afterward the attending physician insisted on a consultant, because forceps were deemed necessary. While she was under anæsthesia, two doctors extracted the child and after the labor both mother and child were apparently in normal condition. Suddenly,

two and one-half hours later, the patient was seized with a generalized convulsion, particulars about which are impossible to obtain. For instance it is impossible for us to know whether the convulsions consisted of generalized tonic or clonic spasms, or were limited to twitchings to one side. It is said that the patient was unconscious during this convulsion, which lasted about one-half hour or more. A second convulsion seized her five hours later, and during the night several others; altogether the patient is said to have had five convulsions. She was practically unconscious for three weeks and during this time, had one or two more convulsions.

She was treated during her unconscious period for eclampsia, and in applying hot water bags for the relief of the supposed kidney condition, horrible burns of a second degree were inflicted upon the patient practically over the whole back. At the end of three weeks, when the patient regained consciousness, it was found that she was paralyzed on the right side, including the face and both extremities. It was found, also, that the patient was practically wordless, her utterances being limited to the words "Yes" and "No," which were not always used properly, as a means of assent and dissent. She did not seem to understand

what was asked her, and it was considered that she had suddenly become demented. She was taken to St. Michael's Hospital in Newark and remained there for six weeks. By this time the paralysis on the right side of her body had cleared up considerably, being reduced to a hemiparesis. The speech condition, however, was practically not at all influenced, and the patient's condition has continued as it now presents itself since then. The aphasic status is as follows:

There is no answer at all to Questions 1, 2, and 3.

There is complete failure in answering Questions 4, 5, and 6.

7. She can only write her first name "Mary" and makes the first letter of her second name (maiden name) which is T., but goes no further.

Complete failure in answering Questions 8 and 9.

10. She is only able to copy her written name. This should be considered a complete failure.

11. Patient does not stand up when asked to do so. When a watch is held to her ear, a look of intelligence and recognition spreads over her face, but she is not able to tell what she hears or describe, write or indicate in any manner the name or use of the object.

Complete failure for Questions 12, 13, and 14.

15. The patient is wordless except for the words "No," "Yes" and "All right," which are not always correctly used.

It must be remembered that this patient had an ordinary public school education, that she was able to read and write commensurately with one in her position, both in Italian and English, but now can do neither.

This patient was sent to me for commitment to a hospital for the insane, it being considered she was demented. There is no case that I can remember to have seen or read of which can approach the depths of loss of all intelligent speech function which was reached in this case. She had absolute aphasia and apraxia. She did nothing purposeful with either extremity of both sides, in the absence of sensory or motor paralysis and ataxia. She could obey no command, either by the aid of mimicry or imitation, or in any other manner. The simplest and most primitive suggestions given in various ways were not perceived. She simply laughed immoderately, and if she thought the examiner was displeased, would cower in fear or would attempt to placate by guttural and throaty sounds of pleading.

All four of these patients had this in common, namely they were untidy, slothful

and often allowed nasal and salivary secretions to soil their faces unnoticed and were in other respects very careless in their personal appearance and habits. This was definitely not a characteristic of their condition prior to the onset of their trouble. They all indulged in much silly conduct, characterized by immoderate, often causeless laughter and seemed to show no proper appreciation of their sad plight. The emotional reactions of all of them were entirely inadequate and they were quite dull and indifferent, having apparently lost interest entirely. While the woman M. A. showed what was apparently a depression, this mood was not consistent, her husband assuring me that she indulged in much foolish smiling and grimacing and in much impulsive and silly behavior. She wandered aimlessly around the house and would not read or cook, both of which things she liked to do a great deal before she became sick. She took to her reeducation with much antagonism and I had a great deal of difficulty in getting her interested. She was much like a petulant spoiled child. This patient had her interest revived, however, and her mood became more stable as she began to improve. Hand in hand with the improvement in the aphasic status, her behavior became more nearly normal,

until eventually she cooperated very well and showed considerable interest in her work. The other patients, however, continued their silly conduct and became hardened clinic hangers on, begging for money each time that they were asked to come for treatment, examination or lecture. There is little doubt that if every case of aphasia is carefully inquired into with this object in view, that disturbances in the volitional, emotional and intelligence spheres of their mental life can be found in practically every case.

CHAPTER VI

APHASIA AND APRAXIA

Marie[22] located the areas for speech, together with those for apraxia, and considered that together these areas were centers for the ideas of highly skilled and conventional acts. According to him, lesions in areas causing apraxia result in a defect of intelligence, as a consequence of which the apraxic patient is unable to perform purposeful acts in the absence of any paralysis, and he also considers that the apraxic patient suffers from aphasia of low grade.

That the destinies of speech and apraxia are intimately bound seems to be undoubted, despite the fact that cases of apraxia have been reported which seem to be limited to the left side only, pointing to lesions in the right hemisphere. The following quotation from Wilson's article, "A Contribution to the Study of Apraxia with a Review of the Literature,"[35] will illustrate this particular point, i.e., how closely bound aphasic and apraxic manifestations may be found in the same patient:

"There is a case* of left hemiplegia in the National Hospital at present which presents certain features of unusual interest for our subject. It is a case of mitral stenosis with an ordinary embolic lesion of considerable extent in the right cerebral hemisphere. The clinical picture is one of complete flaccid paralysis of the left arm and leg; the left face is also affected, but to a much less degree. On examining the patient one is at once struck with her immobility and lack of spontaneity. She rarely speaks unless spoken to; she still more rarely makes any voluntary movement of her non-paralyzed right limbs. When requested, however, she can perform any movement with her right arm in an eupraxic way, and there is no agraphia. Her face is more or less 'fixed' in an expression suggesting anxiety; her eyebrows are symmetrically elevated, and her eyes look straight in front of her. This expression never varies. She is quite incapable of making any grimace, of smiling, of laughing; when asked to try, her reply is 'I can't.' She is incapable of closing her eyes on request, or, rather, of sustaining the contraction, for she will close them momentarily, opening them again without the slightest pause. She is unable to look voluntarily to the right or the left, and

* A. P., under Dr. Risien Russell.

it is only with difficulty that she can be tempted to gaze in either of these directions, by the stimulus of a sound or otherwise. Yet there is no paralysis of conjugate deviation at all. She can protrude her tongue, can show her teeth, bite, etc., well enough. Her voice, since the stroke, has been peculiarly monotonous and rather shrill; she is incapable of modulating it. There is a fixity of tone more or less in keeping with the fixity of her facial expression, but I have the authority of Sir Felix Semon for the statement that the larynx is normal. She is incapable of making the movements of swallowing, though with food in her mouth she swallows well, if slowly; she cannot cough, or sigh, or yawn voluntarily."

This case of Wilson's, then, illustrates quite definitely that apraxia may occur with lesions in the right cerebral hemisphere. That this cannot in any way be used to separate the problems of aphasia and apraxia is seen from the fact that many cases of aphasia have occurred with lesions also of the right hemisphere, and that, too, in right-handed individuals of right-handed stock. Two personal cases will be mentioned later.

A systematic presentation of the subject of apraxia as it relates to aphasia is extremely difficult, because of the many contradictions,

disputes and uncertainties of psychological, anatomical and pathological knowledge adduced in the elaboration of both subjects. The attempt made by so many sharply to delineate and separate apraxia and aphasia as definite parts of general intelligence is perhaps responsible. Here no such attempt will be made, and the idea followed will be to correlate aphasia, apraxia and general human intelligence closely without particular regard to specific cerebral localization of these functions.

Wilson states that he has seen ideational apraxia and agnosia in alcoholism, senile dementia, delirium, postepileptic confusion, etc. Here "under the influence of some toxin or some general pathological process, the function of the whole cortex must be seriously at fault." We shall also see that Liepmann's[19] original case also presented profound mental changes such as are seen in advanced senile dementias.

While the views of Wilson are very clearly set forth, he seems to have followed other workers in the field of aphasia and apraxia in so far as he has also made reference to various claims of localization, and quotes diagrams purporting to explain the mechanism of the production of apraxia and its localization. As previously with aphasia, the statement is

again made in the case of apraxia, that in right-handed individuals the lesions causing both of these conditions are to be found in the left hemisphere. This writer carries the analogy further by recognizing also in apraxia, as in aphasia, cortical, subcortical and transcortical apraxias. A great point is made of the fact that apraxia is always supracapsular and may exist without any sort of paralysis at all, the capsule escaping responsibility in all cases. Wilson draws a close analogy between motor aphasia and apraxia. Indeed, he says that "in motor aphasia we have a form of apraxia of the speech musculature." He ascribes the loss of motor speech to the inability to articulate auditory, visual, and, most important, kinesthetic word images. Agraphia and the instrumental type of amusia are also special kinds of apraxia, and he ascribes the loss of function in these cases to lesions of the centers for graphic kinesthetic images, or the visual image of writing, or to a loss in the image of the movement for writing or playing instruments. In other words, his ideas of apraxia follow very closely the various theories of localization as set forth by Broca, Wernicke and Villiger in aphasia, and these theories are absolutely dependent on the conception of more or less circumscribed centers connected in each hemisphere and intercon-

nected between hemispheres for the production of voluntary motion. This, in spite of the fact that it has been impossible to show by anatomical or pathological investigation that any such centers exist either in apraxia or aphasia. Wilson, however, restricts the term apraxia to the motor side, following Liepmann in this respect, for he says in the same article that "in regard to the lesions which underlie ideational agnosia, or ideational apraxia, for that matter, there is little to be said that is definite." In this connection, the pathological anatomy of the subject of apraxia will show by reference to the literature of cases reported by Stransky[32], Marcuse,[21] and Pick[24] that in a great many of these cases the lesions are too widespread to be of any localizing importance. Only in the matter of motor apraxia does there seem to be any ground for the suspicion that the supramarginal and angular gyri and the first and second left frontal convolutions have anything to do with this difficulty.

The original case of apraxia described by Liepmann[19] was one of motor aphasia and right-sided apraxia. At postmortem, it was found that the whole corpus callosum was destroyed excepting the splenium, that there were two foci of softening in the white matter beneath the first and second convolutions of the left

hemisphere, also that there was a large subcortical cyst beginning under the lower third of the postcentral gyrus, spreading back through the parietal lobe beneath the supramarginal gyrus, and ending at the posterior part of the angular gyrus. Besides these lesions in the left hemisphere and in the corpus callosum, there was a focus of softening in the right hemisphere and in the internal capsule, and another in the neighborhood of the supramarginal and angular gyri. So that this case, which formed the basis for the original conceptions of Liepmann of motor apraxia, is very unsatisfactory from the viewpoint of pathological anatomy in relation to localization.

Most patients suffering from apraxia have arteriosclerosis. Von Monakow[34] declares that generalized cerebral vascular sclerosis plays an important part in interrupting association tracts and also in causing deficient nutrition to all parts of the cortex. So that areas of softening in any one of several spots, due to vascular change in the vessels of the brain, may not entirely explain the symptoms; for it is inconceivable that the defective nutrition caused by the arterial changes in all parts of the cortex distant from the lesions found, may not also help to explain some of the trouble. For this reason, a statement by Liepmann in

regard to apraxia and by others regarding aphasia, that these conditions are more common in lesions of the left hemisphere, is not borne out by pathological investigations, because single lesions in either condition are rare and seldom are the lesions found limited to either hemisphere alone. Clinically, also, the statistics of Liepmann seem to confirm this statement. These statistics show:

Of 89 patients, 42 had left hemiplegia and 41 had right hemiplegia, 5 had aphasia but no hemiplegia and one was purely apraxic only. In describing the left hemiplegias, Liepmann says that nearly all were able to execute given movements with the right non-paralyzed arm. Of 41 right hemiplegics, 20 were apraxic and there were 14 also aphasic, which demonstrates rather conclusively the intimate relationship between these two conditions, although these figures also demonstrate that the conditions are not necessarily associated. It seems rather to be deplored that most of the cases of apraxia which have been investigated with pathological findings have been patients who were either old dementias of senile or arteriosclerotic origin or sufferers from alcoholic dementia. This is especially true of the work of Bonhoeffer[1] and Pick[25]. While it seems to have been fairly definitely proved that the great majority of

cases of aphasia and apraxia occur in left hemisphere lesions in right-handed individuals, it has not been so definitely proved that the remaining cases of aphasia and apraxia necessarily occurred in individuals who were left-handed, and who, therefore, may have had a specially developed right hemisphere. We will be able to show several cases which will clinically illustrate this point.

It is not absolutely essential to our conception of speech disturbances or voluntary movements that they be localized in either hemisphere alone, and it would seem that caution should be used in adhering strictly to the viewpoint that these special skilled acts, of which speech is one, have their correlating centers almost exclusively in the left hemisphere. In nearly all of the reported cases of apraxia with autopsy, and in a great many cases of aphasia autopsy, with lesions are found in both hemispheres. It may be assumed with fairness that various individuals in attempting to fit the findings with their theories have unduly emphasized the findings in the left hemisphere. It seems that the most sensible view to take of the entire matter is that the whole cortex is indivisibly associated with the development of the function of speech and its expression, and also with the development of

the function of specialized purposeful movement and its performance. True, in certain individuals, the left hemisphere acts as the leading or dominating organ for the inception of these functions; but in a great many cases, the conditions may be reversed. In this connection, Liepmann has always denied that either one of the hemispheres is necessarily the dominant factor in the production of apraxia. He also denies the existence of an apraxia center in either the supramarginal or angular gyrus. His contention is that any such center or group of centers cannot exist independently of the motor sensory area grouped around the fissure of Rolando. The main point in this discussion for him is the function of the corpus callosum, particularly that portion through which fibers come from the anterior central convolutions, namely, the middle third of the corpus callosum. In illustrating this point, Liepmann calls attention to the fact that in most cases of apraxia due to lesions of the corpus callosum, the posterior 4th or 5th is intact. Secondly, cases have been repeatedly observed in which the anterior portion was destroyed without apraxia being present, and thirdly, there is a case on record in which apraxia has occurred when the anterior portion of the corpus callosum was entirely intact.

Bearing on the point of stock-brainedness, that is, the tendency in families to have the left or the right hemisphere predominating for speech and voluntary movement, as is shown by the right- or left-handedness of such individuals, the work of Kennedy[14] may be of interest. Before referring to this author, it might be well to state that according to Liepmann's researches, 95½ per cent of all individuals are right-handed, and, therefore, their left hemispheres lead in the production of purposeful movements and in the utterance of speech. Kennedy asserts that while certain individuals of a right-handed stock may be left-handed, in these individuals the speech center is always in the left hemisphere. Kennedy also applies the same rule to individuals of left-handed stock who are right-handed, the leading hemisphere in such individuals being the right one. He calls on these observations to explain the existence of crossed aphasias, that is, aphasias occurring in right-handed individuals with left hemiplegia and in left-handed individuals with right hemiplegia. The facts of stock-brainedness are purely speculative and have never really been proved by any extensive statistics; nevertheless, Kennedy reports seven cases as a basis of his conclusions, which it might

be worth while to set down in short abstract form.

The first was a French sailor who suffered a very extensive destruction of the right hemisphere with a resulting left hemiplegia, hemianæsthesia and hemianopsia. This sailor was left-handed in everything but writing, which he was able to do, however, with either hand. Although the lesions in the right hemisphere were very extensive, there was no evidence of aphasia at any time. Kennedy states that this man was the only member of his family who was left-handed. Nevertheless, the fact that he was able to write with his right hand is overlooked, and also the further fact that his ambidextrous accomplishments may have been acquired, and that, after all, the left hemisphere may have been the leading one, despite the author's conception.

A second case is a young man of 36 who had a right-sided hemiplegia and was aphasic. He was always left-handed except in writing, and was the only member of his family who could use his left hand for skilled acts. Here the same remarks apply as in the foregoing case.

A third case was a left-handed individual who developed a left hemiplegia after a fracture of the right occipito-temporal skull and middle meningeal hemorrhage. He was the only left-

handed member of his family. No aphasia developed. Again, the remarks made in the first case are pertinent.

The fourth case is a right-handed girl who had severe left-sided attacks of epilepsy followed by severe but transient hemiplegia and aphasia. This girl's mother and father were left-handed and the inference is that her speech centers were in the right hemisphere which, according to Kennedy's theory, should also have been the leading hemisphere for all sorts of skilled movements. Whether these cases are illustrations of the theory of stock-brainedness or whether they are merely examples of the point Hughlings-Jackson so often made, that no particular hemisphere has the exclusive function of the speech mechanism, is a salient point.

The fifth case occurred in a young man who suffered from complete aphasia for ten days following a right subtemporal decompression for a right-sided subcortical Rolandic glioma. This patient was right-handed, but two brothers were left-handed, and it is presumable that Kennedy wished to imply that in this individual the right hemisphere was the leading one for speech.

The sixth case was a woman of 67 who, following a cerebral hemorrhage, had a left

hemiplegia with mixed aphasia. She was right-handed, but her mother, brother and a daughter were left-handed; so that presumably the right hemisphere in this individual was the leading one for speech.

The seventh case was one of left hemiparesis, hemianæsthesia and hemianopsia with no aphasia. He had been left-handed all his life and was ambidextrous in regard to writing. The rest of his family were right-handed and so presumably the left hemisphere should have been dominant for speech in this individual.

The fallacy of this sort of observation can readily be seen when attention is called to the fact that a great many cases of right hemiplegia occur without any sort of speech defect whatsoever, regardless of whether the individual is right-handed or not. We have all seen this a number of times. Some support is given, clinically at least, to Jackson's conception that it is not essential for either hemisphere to be the dominant one for the production of speech or purposeful voluntary movements. The following cases will tend to illustrate, clinically, the difficulty and the dangers of ascribing too much importance to the accurate localization of the speech mechanism in either hemisphere alone:

E. C., male, 51 years of age, single.

History.—He was taken on the 14th of November, 1917, at 10:30 A.M. from a saloon by an ambulance surgeon from the Kings County Hospital. While standing at the bar he had suddenly become unconscious and had fallen to the ground. He had had no prodromal signs of any description. When he was brought to the hospital, he was unconscious, temperature 99, pulse 96, and respiration 18. He remained unconscious for two days and the blood count and urine taken immediately were negative. The blood pressure was 125 systolic and 70 diastolic. The outlines of the discs were blurred and showed some edema and the vessels were congested. He had no convulsions during this time, and at the end of the second day he could be aroused only with extreme difficulty, would open his eyes, but could not speak a word. He moved his right arm and leg, but the left arm and leg were completely paralyzed and flaccid. The Marie-Foix sign was present on the left side and there was an Oppenheim and Babinski sign on the left side with clonus. The left abdominals were gone and the left side of the face was paralyzed. The tongue deviated to the left and the uvula somewhat to the right. The pupils

were normal in every respect although somewhat dilated.

On the evening of the second day he had a nosebleed and the sight of the blood started him speaking, indistinctly however. He spoke a senseless jargon which could not be understood at all. A Wassermann taken of the blood at this time came back negative. This was repeated three times and found negative each time. There is, however, the history of a chancre twelve years ago with treatment at Hot Springs by mercury and inunctions, for a period of about two months. Further questioning brings out the fact that he had an attack of palsy on the right side of the body three years ago from which he recovered entirely at the end of two weeks, having had at no time any speech defect whatsoever, although the attack was initiated by a period of unconsciousness lasting several hours.

This patient played ball and did his mechanical work always with his left hand and favored his left hand in doing all sorts of skilled acts, except writing. He always wrote with his right hand and wrote a very fine hand. Spinal puncture was denied.

When the patient said his first words at the end of the second day he could not make himself understood at all. A day or two later

he could use "Yes" and "No" for assent and dissent and could give his name. Shortly after this he tried to give a description of his accident in halting, stumbling phrases with great difficulty and made many mistakes which he, however, immediately recognized and tried to correct. He wrote a good account of his whole trouble without difficulty; heard and understood what he heard, saw and understood what he saw, and did Lichtheim's test well, both for letters and syllables. He could read spontaneously and from dictation, understood what he wrote and could write from copy. He understood perfectly what was read to him or what he himself read, but became lost in a meaningless jumble of phrases when he attempted to speak. He was very much excited and very much upset. He was encouraged to write and to read aloud, and the improvement in him grew quite rapidly, so that at the end of a week he could make himself understood although his vocabulary was still quite limited.

On November 27, two weeks after the accident, he had, to all intents and purposes, recovered entirely. He could read and speak well, and the paralysis on the left side had improved synchronously with the improvement in speech. The physical signs on the left side

had disappeared entirely, and the patient was discharged apparently perfectly well.

R. B., male, aged 29, married.

He came to the Vanderbilt Clinic May 11, 1917, complaining of paralysis of the left side of the body, including the face, arm and leg, the loss of memory, and, as his wife expressed it, "foolishness." This condition had lasted three months and has remained stationary.

History.—On December 17, 1915, one and a half years before the onset of his present trouble, he was hit by an automobile in the lower spine. He was thrown to the ground and was unconscious for two hours. He was taken to Bellevue Hospital where an *x*-ray and blood examination showed nothing in the way of fracture, dislocation, or disease. He was discharged from the hospital with a diagnosis of contusion of the spine, and was returned to his home practically well, excepting for a soreness in the lower dorsal region when he bent forward. This pain in the back persisted for several months and finally disappeared. For two months before the actual onset of this trouble, the patient complained of headaches and vomiting nearly every day, usually at night after his supper. During this time, too, his general

disposition seemed to undergo a change. He was unusually irritable and restless, and gave way to explosive outbursts of temper. He did not complain at any time of dizziness, nor did he have perversions or subjective changes of sensation in any of his limbs. There was no numbness or weakness and never any disturbance of speech during this prodromal period of two months before the onset of his aphasic condition.

The patient's wife says that he has never been ill, and he says he has never suffered from any venereal infection and that for a number of years he had been in the habit of drinking about one pint of beer a day. They have had two children, both of whom are well. The wife never had any miscarriages.

It is well to note the early history of this patient. He was born in New York City, began school at the age of six and graduated at the age of 14. He was bright and was promoted regularly, but was never very fond of attending school. After graduating he went to La Salle Academy for one year and quit soon after his father died in order to go to work. The only foreign language which he ever tried to study was French for one year at La Salle Academy. It cannot be said that he ever really knew any French, and he forgot what he did

know long before the onset of his trouble. He was always considered a fairly good singer. He never had any musical education, either vocal or instrumental. He was always rather wild, and preferred to dance and play pool rather than study while he was at high school. He always preferred mechanical work to study of any kind. He never read anything except newspapers, and can be said to have had only an ordinary public school education. Even mechanically, however, he was never very skilled and was only a laborer in the highways department of this city when attacked by his present trouble. He and all his family are right-handed.

Onset.—This patient was in the habit of going to work at night as a watchman. At 1 o'clock his actual work was finished and from then until 7 o'clock in the morning it was his custom to sit on a chair or pass the time as best he could. It is definitely known that until 1 o'clock on the morning of February 25, 1917, he had been in his usual health; in fact, for a few days before the onset he had had no headaches, no vomiting. Nothing is known of what occurred to him after 1 o'clock on this particular night, and he himself is not able to explain, excepting that he sat down to rest

and to sleep until morning. He was not seen again and nothing is definitely known about him until a chauffeur found him unconscious on the floor about 7:30 A.M. It is known positively that he was unconscious for an hour thereafter. When he regained consciousness he did not speak for more than a week. He was absolutely wordless. During the second week he began to call his wife by name (May), then his boy Willie, and finally his sister Hannah. He could say "Yes" and "No," but did not always use the words properly; and he could ask for a drink. During the second week he was very bad tempered. He remained in Bellevue Hospital for three weeks and during this period he could say no more than what has just been mentioned. He was five weeks in the City Hospital and here he improved physically and was able to get up and walk, but mentally he has remained practically the same as now. He has added but very little to his speech and the condition has practically continued the same since the onset.

The aphasic status, in the order of our fifteen questions, is as follows:

1. "Cold—none—stroke—speech." "I do not know." In other words, there is no spontaneous speech.

2. Counts all fingers, then says "Two." Asked how many fingers on both hands, says "Four." "Willie, Annie," says "Maggie" then "May," and then mentions the name of his niece, "Genevieve." He was asked to limit his answer to the members of his immediate family. He included his sister and niece (Hannah and Genevieve), and made a mistake at first in mentioning his wife's name, so that we may consider this a defective answer.

Asked to name the days of the week, he said "August" and the second time "September," which, of course, makes this an absolute failure.

Asked what an inkwell was, he responded "ink." Pointing to a yellow sheet of paper and asking for the color and the name of the page, he answers, "paper." Pointing to a magazine, his answer is "book." Pointing to a rubber stopper in a bottle, his answer is "celluloid." Pointing to a blotter, he says "boiler."

When asked to name the number of chairs in the room, he gets up to close the door. When asked the color of his tie, he says "blue," then says, "I don't know." (The color of his tie was black.)

3. He cannot speak from dictation.
4. He cannot read aloud what is printed.
5. When asked to read to himself from a

newspaper article, he gets up to look for a piece of chalk and finally asks for the chalk by name. The only thing which he can read is his own name, that of his wife and child and very little else. He does not understand what he reads, even though the article is a simple one.

6. He cannot recite his prayers, with which he was previously very familiar. Cannot sing his favorite song, which was "River Shannon." Cannot even hum it or whistle it tunefully. Tone and cadence are missing from the whistling and humming.

7. He cannot write spontaneously except his own name and his wife's name. He occasionally attempts to write to friends and the result is a hopeless jumble and jargon. It is interesting to note that when he wrote to his wife from the hospital he always addressed her by her maiden name.

8. Writes from 1 to 10. He cannot write the days of the week nor the date. Does incorrectly even simple sums. Cannot subtract and cannot multiply or divide.

9. Cannot write from dictation.

10. Cannot copy printing or writing. When asked to copy a simple sentence from a magazine he answers by writing his name.

11. He hears but does not understand what he hears.

12. He sees but does not understand what he sees.

13. He is apparently not aware of errors made in writing or speaking. He seems unable to say definitely whether he makes mistakes or not. Certainly he does not correct them when made.

14. He appears to have no conception of his speech as tested by Lichtheim's test. He cannot tell how many syllables or letters there are, by any means of indication, in any words, not even his name. Occasionally he can tell the number of letters in his name and in the name of the president of the United States, but he is not always correct. He has at no time been able to tell us the number of syllables in any given word correctly several times in succession.

15. He misuses words; he is ungrammatical out of proportion to his education. He repeats fixed phrases and when he does speak or write, it is a hopeless jargon.

The family history in this case is very interesting. His father is dead, cause not known. The mother died at the age of 43 from an attack of apoplexy, after an illness lasting only three days. The onset in the mother's case was very sudden and she never regained consciousness, so that the supposition is that she had a cere-

bral hemorrhage. The rest of the family consists of two sisters. One of them is perfectly well physically and mentally. The other, aged 32, had a right-sided apoplectic seizure two years ago without a defect in speech of any kind whatsoever. She did not make a good recovery from this, although she improved somewhat until three weeks ago, when she had a second similar right-sided attack of hemiplegia. The Wassermann is negative in both the sister and patient, the patient having had numerous spinal fluid and blood Wassermanns performed at Bellevue Hospital and here, all of them negative.

The physical examination shows the usual symptoms of a profound left hemiplegia.

NOTE: In both of these cases the ascendants were right-handed.

CHAPTER VII

THE PSYCHOLOGY OF VOLUNTARY MOVEMENT AND SPEECH

The conceptions of the development of speech and the mechanism of its production are intimately bound up with the problem of the cause of voluntary movements and the development of purposeful skilled acts. Psychologists have taught, and clinicians have followed their lead in teaching, that the idea of a movement, whether it be the idea of a movement of speech or any of the other purposeful movements, exists in consciousness and precedes the execution of the act. This is particularly brought out by William James [13] whose conception is that among the sensations produced by a movement there are some which are caused by stimulation of sense organs not located in the moving members. These sensations are called by James remote sensations, and they are not the constant and variable results of the movement. For instance, "the eyes may close or turn to one side and the ears may be filled with the noise of other things." But what he calls resident sensations are the muscular and cutaneous sensations which

originate in the moving member and are constantly necessary for a given movement. It is these kinesthetic sensations, according to the psychologists, which give us the knowledge of our movements and constitute the ideas of movement. In other words, to will a movement is to bring into consciousness the kinesthetic memories of such a movement. The idea of the movement must be present before the will can work on it and the voluntary act follow. Therefore, given the kinesthetic or sensory image of the movement and how it is going to feel, the next step, according to this psychological conception, is the performance of the actual movement itself. This idea is brought out in the words of William MacDougall:[20] "The kind of idea that tends to issue most directly in action is the idea of a movement, the kinesthetic idea." This is illustrated by the author in the case of the beginner in golf who is coached in addressing the ball by an expert. We quote: "The expert commands a readjustment of this and that limb, of the trunk and head, until the proper attitude is struck, and it is the learner's task to combine the kinesthetic impressions which this attitude yields to a single percept that can be produced on future occasions. Frequent repetition of such a series of movements under

similar conditions results in their becoming what is called secondarily automatic."

Many authors, particularly Liepmann, have persisted in placing this area for the reception of kinesthetic impressions in connection with speech production in the motor cortex; others, including Déjerine, as we have already seen, have placed the area for the reception of the kinesthetic images of speech in the left third frontal convolution. We hope we have, however, succeeded in throwing a great deal of doubt on the proposition that this area of the brain possesses this function, as applied particularly to speech. We have adduced considerable pathological and anatomical proof against this conception in the work of Marie, Moutier and others already quoted. Psychologically, the conceptions of MacDougall and other psychologists have been disproved by Woodworth.[36] In his experiments, the subjects used were young adults, two women and eleven men, nearly all of them persons possessed of psychological training. The experiment consisted in requesting these individuals "to make given movements with some preliminary hesitation, and to note the condition of mind that preceded the movement. He was to note particularly what imagery appeared; and in the case of motor images he was asked to compare them

with the sensations resulting immediately afterwards from the actual movement." Stress was laid on the necessity for the slow performance of movements so that imagery would be given more time to appear. Prompt movements were also made which, however, were less often accompanied by any sort of imagery. The movements consisted of opening the mouth, wagging the jaw, winking and closing the eyes, flexing or supporting the fingers, flexing the foot, opening and closing scissors, forceps or manipulation of the dynamometer. In some cases a choice of various movements was allowed; for instance, the hand was to touch any part of the body or an object in the foreground. The fingers were to be flexed or extended, or a reaction was to be made to a sound with either the hand or the foot.

"The results of 128 single introspections of the conscious preliminaries of voluntary movements showed:

27 gave kinesthetic imagery.
27 gave visual imagery.
17 gave imagery of other kinds.
30 gave only peripheral sensations.
27 gave an absence from the field of attention of all sensorial elements, whether external impressions or images.

"In 55 per cent some sort of imagery oc-

curred, but kinesthetic imagery occurred in only 21 per cent and it was marked or 'adequate' in only 9 per cent. In nearly half of the cases no imagery whatsoever occurred, and in presumably only one-tenth of the cases were the images 'fair representations of the actual sensations of movement.' "

Pertinent to this discussion are the experiments which Woodworth made on himself, as a result of which he reaches the conclusion that consciousness does not play an important part in the establishment of complete voluntary control. Woodworth was unable to detect any kinesthetic imagery in attempting to establish complete voluntary control of the isolated movement of extension and flexion of the great toe separate from that of the other toes. Quoting again from the author, he says: "I infer from the results of Bair combined with my own, that even in first getting control over a particular movement, at least in the case of adults, the kinesthetic image of that movement is neither a necessary nor a sufficient condition." The author calls attention to the fact that the child is equipped by nature with a large store of "definite reactions, defensive, locomotory, nutritional, vocal, ocular, facial, as well as with what seem more random movements of the arms and legs. These reactions necessarily

occur first without the child's foreknowledge or intention; but having thus occurred they enter into the developing system of his thoughts and desires; they become associated with other things, and this is the process by which the child acquires control of his motor inheritance. The question is whether the first associations are formed between the movements and the feelings and images of them, or whether the child's first desire for a movement is the desire to feel it again." That this is not necessarily so is evidenced by a number of observations, one of which is reported by Kirkpatrick.[15] A child who was 17 months old and had not learned to walk, one day became interested in a pair of cuffs which had been left by her father on a table. Creeping to the table the child pulled herself up, reached for a cuff with one hand and put it on her other wrist, thus standing alone for the first time. Now she placed the other cuff on her other hand and walked across the room. When the cuffs were taken from her, she refused to walk any longer and it was only after a pair were given her that she resumed the new-found act of walking, which she continued to use even after the cuffs were taken away from her a few days later.

Instances of children who have learned to

walk suddenly are numerous; for instance, my own first boy did not walk although he was 15 months old. Suddenly one day he straightened up from the floor and walked unaided for fully 16 feet. His aunt screamed with surprise and pleasure, and the boy immediately sat down and did not walk again until two weeks later. On this occasion some noise in the street attracted him and he lifted himself up by the window sill, stood there looking into the street and then turned around and walked across two rooms to his mother. These cases certainly show that kinesthetic images are not always the first thing to be associated with movements, and that they do not necessarily precede them. These acquired skilled acts, however, having once been mastered, the interest passes on to others. There probably is not much doubt that the initial acquiring of movements becomes the property of the child accidentally.

Dexter[7] observed the progress made by a child in the coördination of movements. He noticed that this child one day happened to put his hand into his mouth, seemed pleased and attempted to do it again. The following day he succeeded a number of times and in a few weeks had the movement completely under control. Woodworth, in detailing this case,

feels that it is impossible to conceive that the movement of the child's arm, when it had accidentally put its hand into its mouth, was accompanied by any particularly different sensations or impressions from other movements which it had attempted prior to this of a similar character. Later, the sensations in the mouth having given pleasure, they became associated with the motor innervation and thus the movement finally became a voluntary one. This may be the genesis of a number of voluntary movements. The same argument is applied to the matter of visual and auditory images, the author insisting that the eye movements illustrated by stimulation of the visual cortex and the movements of the head in hearing due to stimulation of the auditory cortex may occur quite independently of the motor area. For instance, it is a fact that if the coördinating center for eye movements is stimulated, even if the motor area for the eyes has been cut off, the eyes will turn in the direction of visual stimulation. This would seem to preclude the necessity of these movements depending on kinesthetic images. This line of reasoning also applies to the turning of the eyes towards a source of sound, this movement also occurring on stimulation of the auditory area when the

motor and visual areas are destroyed. Important for the production of voluntary movements, however, according to Woodworth, is the sensation of the initial position of a member, since the movement will differ according to the initial position. If a hand is in the position of flexion, that position must be sensed in order to determine the initiation of movement in response to stimuli. If the knowledge of the position of the hand were lacking, the result would be indecision. Sherrington[28] has shown that by cutting the afferent fibers from the muscles in monkeys, indecision and ataxia of movement occur. There will be over-reaching or premature stopping. This special sense of the position of the limb prior to the initiation of movement may not come into consciousness and it may be in a large measure reflex, but in deliberately preparing to make a movement this sensation of position is absolutely essential. Woodworth's conclusions are, therefore, first, that a kinesthetic image does not occur as the adequate determinant of voluntary movement. Second, he denies the existence of any image at all as such a determinant, and third, he insists that the field of attention just prior to the movement is often inadequate as a "distinguishing mark of the movement." He further states that the "intention" is not always

present and is seldom felt in the field of attention at the moment just preceding the innervation of the movement. According to this author, then, the proprioceptive sensation of position of a limb is vital to the occurrence of voluntary movement. In passing, attention is called to the fact that the cerebellum is the great proprioceptive organ. Referring to his experiments, Woodworth found that in a great majority of cases the condition of mind preceding the movement was almost blank. He says that the "complete determinant of a voluntary motor act, that which specifies exactly what act it will be, is nothing less than the total set of the nervous system at the moment. The set is determined partly by factors of long standing, instincts and habits, partly by the sensations of the moment, partly by recent perceptions of the situation and by other thoughts lately present in consciousness. At the moment, however, these factors, though they contribute essentially to the set of the nervous system, are for the most part present in consciousnsss as a background or fringe, if at all, while the attention is occupied by the thought of some change to be affected in the situation."

All this is quoted in refutation of the theory set forth by Wilson in explaining apraxia.

On page 192 of the 31st Volume of *Brain*

appears the following exhibition by this author of the theory of the production of apraxia which is taken from Kleist.[16] The instance, as quoted by Wilson, is that of the sight of a rose, "suggesting the idea of my taking it and put-

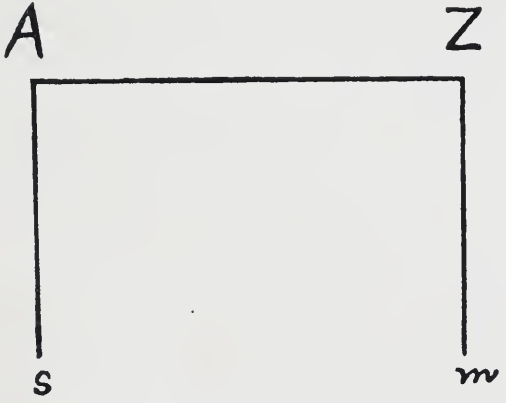

FIG. 3. DIAGRAM OF WERNICKE

ting it in my buttonhole." In this scheme the diagram of Wernicke is used. "The *s* stands for a visual impression, the *A* would stand for the intimately associated idea, namely, the rose. The *Z* is the ideational connection suggested by the idea of the rose which is to put the rose in my buttonhole. Finally, *m* is the idea of the movement which is to work out

the aim or object of the idea of plucking the rose." Now, the author says, follow the actual innervation of the motor sensory area and the completion of the act. In the light of Woodworth's experiments and the views which he lays down as a result of the same, one can see that this generally accepted scheme or diagram explanatory of the production of purposeful movements, is untenable.

CHAPTER VIII

THE MODERN CONCEPTION OF SPEECH

We are now ready for what we believe should be the proper conception of speech. Having as we think proved that speech is a form of skilled voluntary movement and that it is a function of the entire intellect, it follows that the development of what we know as general intelligence or, psychologically, the mind, is inseparable from the development of speech and skilled voluntary movement. Speaking clinically, therefore, disorders of general intelligence, aphasia and apraxia are inseparable and indivisible. In order to prove this clinically, we must show that all cases of aphasia and apraxia are at the same time deficient in intellect. We must prove that will, memory, judgment, emotions, perceptions, impressions, everything that we include as a subdivision of the concept "mind," are affected in cases of aphasia and apraxia. We do not anticipate a great deal of difficulty in doing this. Not only must we show that intelligence is defective in cases of aphasia but we must point out this defect in all clinical entities wherein speech

is affected; otherwise, our conception breaks down entirely. We have already illustrated this point in our cases of aphasia; later we shall bring to our aid cases of Friedreich's ataxia and Marie's heredo-cerebellar ataxia, cases of multiple sclerosis with speech defect, chorea, paralysis agitans, and the other clinical conditions in which speech is affected, not leaving out the so-called functional conditions. In keeping with our conception we are prepared to show, clinically at least, that the entire brain takes part in the control of the mechanism of speech, not excluding the cerebellum, for in a number of these clinical conditions, this great proprioceptive organ is involved, if not primarily, at least secondarily. For instance, in chorea, Hughlings-Jackson, as far back as 1864 in the clinical reports of the Lincoln Hospital, pointed to the case of a patient suffering from chorea who for several years had been in the habit of saying quite involuntarily, "bloody." In recounting this instance, Jackson states that the patient was frightened by a man shouting the word. The fright "produced chorea, and, if I may use the term, chorea of his mind too; as for three days he said the word 'bloody' and little else, and now he ejaculates it occasionally. The mental process for saying that word is as little under his

control as a few of the muscles of his face are, for the twitching of which he is now attending at the Lincoln Hospital." While we are on the subject of chorea, it might be well to state that Poynton and Holmes[26] found vascular and nerve cell changes in every region of the brain and brain stem in this condition.

It need hardly be mentioned that it is not difficult to point out the coincident development of dementia, speech defects and apraxia in Huntington's chorea. Even in simple Sydenham's chorea the great irritability, mental instability and sensitiveness which occur, at times amounting almost to a psychosis, are often associated with speech defects. These children speak in a quiet, tired, monotonous, colorless manner, or in jerky, sharp, explosive, syllabic stammering, particularly if the choreic movements are severe. According to many authors, choreic children are nervous, irritable and excitable, even before the movements make themselves manifest, and some show marked grades of feeble-mindedness. In the cases which afterwards develop Huntington's chorea, these characteristics are even more marked.

With this digression we will proceed to our discussion of the presence of mental defects in aphasia and apraxia. In the discussion of apraxia in relation to various forms of dementia,

Pick[25] states that dementia is a mosaic of partial dementias localized or localizable, and that this has an obviously important bearing on the causation of apraxia. We have seen, too, that Bonhoeffer[1] in his work with cases of alcoholic delirium in connection with apraxia found that acute exacerbations in alcoholic delirium may produce clinical symptoms referable to various areas of the cortex, and that he is thus able to correlate clinical symptoms in alcoholism with lesions in various parts of the cortex, as confirmed by autopsy findings. In all forms of the psychoses, particularly general paresis and dementia precox, aphasia and apraxia may occur. A number of cases of apraxia studied by the German workers in this field were in the presenile, sclerotic and senile dementia types. Most of Pick's[25] cases were arteriosclerotic and senile dementias. Other workers have pointed to the effects which the general destructive processes in dementia precox have on speech and voluntary movements of various kinds. Southard[29] showed in his work entitled "Study of the dementia precox group in the light of certain cases showing anomalies or sclerosis in upper brain regions," that the symptomatology in the cases studied might be explained by reference to the cortical localization of the most

important disease processes found. In the pre-Rolandic group, this author places nine cases with injury more or less severe to the whole of both upper frontal convolutions. The symptomatology of this group is embraced in the terminology catatonia and paranoid dementia precox. The pathological condition found is gliosis and satellitosis; in one case (No. 1358), the symptoms were exclusively catatonic. In this case the greater part of both frontal regions was wholly spared from microscopic lesion. The only lesion found was one in the left inferior frontal gyrus. In the other eight cases, the entire brain underwent a mild generalized sclerotic process characterized microscopically by widely distributed glioses affecting principally the frontal and occipital lobes and including the cerebellar cortex and even the pons. In case No. 1062, the patient showed various disturbances of speech. He was abusive, delusional, noisy, obscene, and also had a prolonged period of mutism. In all of the other cases the clinical descriptions are meagre, but frequent reference is made to the presence of catatonic symptoms or paranoid delusions with various speech disturbances. Therefore, speech, it can be inferred, was found correspondingly affected with the intellect in each case of this group.

In his post-Rolandic group, there were seven cases, four of which were alike in developing catatonic symptoms. In one case, No. 1137, with a sensory misinterpretation of stimuli with catatonia, the pathological findings were in the right supraparietal region and Southard states that "microgyria and the dense local disposition of fibrous tissue form a striking local lesion in this case." There were also, however, general gliosis and satellitosis. Another case, No. 1298, with catatonia and depression, which had at one time been diagnosed as manic depressive insanity, disclosed on autopsy a double postcentral gliosis in the white matter and visible atrophy of the right postcentral gyrus, also a peculiar focus of "durapial adhesions over the left middle frontal gyrus." Taking the whole of this group of cases, Southard says that they "show lesions which lead to the suspicion, if not the proof, of congenital anomalous symptomatology. These cases have catatonic symptoms in common." In an occipital subgroup of this post-Rolandic group, the author places three cases in which the occipital regions were more markedly sclerotic than the rest of the brain, and he points to the well defined "constantly recurring, voluntarily executed, visual episodes (communications with God) as a clinical anatom-

ical correlation." However, in all these cases there were changes also in the frontal lobes. In one case of this group, No. 1294, the hallucinations shown by the patient were auditory rather than visual, and the brain showed frontal, cerebellar, and pontine glioses in addition to the occipital. Interesting to note is the fact that no temporal gliosis was made out macroscopically, but there were foci of leptomeningitis, both of the temporal and frontal types. In another case, No. 1317, although there was occipital microgyria, the hallucinations were auditory. There were some religious delusions. This would seem to break down the paranoid correlation which may possibly be made out in some of the other cases.

In an infrasylvian group, Southard places one case, No. 1319, with paranoid delusions. He was not able to show any anatomico-clinical correlations in this case.

In the cerebellar group, besides the cerebellar changes, there were profound and widespread changes throughout the whole brain in most of the cases. In one case, No. 1034, there was macroscopically pure sclerosis of the dentate nuclei of the cerebellum. Clinically, this patient had flexibilitas cerea. He began with paranoid symptoms and in six weeks became catatonic. He also had auditory

hallucinations and persecutory delusions, was mute, reticent, impulsive, showed echopraxia and automatism. Although macroscopically there were no palpable changes excepting in the cerebellum, microscopical examination showed a widespread gliosis in various areas of the brain. There was also a satellitosis of the frontal region. In another case, No. 1168, besides the postcentral hypoplasia on both sides in their upper two-thirds, there was a sclerosis of the olives and of the right dentate nucleus and also of other portions of the cerebellum. Clinically, there were hebephrenia, mutism, resistiveness, impulsive reacting movements and attitudes. The largest group of cases reported by Southard was a group of fifteen which were clearly dementia precox, but macroscopically no lesions were found. Microscopically, these cases showed satellitosis of the deep cortex layers and a gliosis of the dentate nuclei. The author claims that a number of these cases might with propriety be classed with the cerebellar group.

In a summary of this material, Southard states that 68 per cent of a series of dementia precox cases, from which there were carefully excluded frank arteriosclerotic and coarsely wasting processes, showed macroscopical atrophies and aplasias. In 86 per cent of the entire

series, or in 24 out of the 28 cases studied, either coarse or microscopic lesions were found. Quoting the conception of Wernicke that insanity is a disease of the association limits of the brain, Southard says, "Even in the field of aphasia, one observes with recent writers a tendency to replace the focal destruction theory with some theory more consonant with modern ideas as to the tremendous breadth and complexity of the cortical areas attending speech."

In explaining convulsive movements, catatonic attitudes, etc., the author says: "Neither a convulsion nor a catatonic impulsive movement is adequately explained by lesions of the muscles involved, by lesions of the motor nerves or spinal cord, by activities in the central gyrus, or even necessarily by any post-Rolandic or pre-Rolandic brain mechanism or combination of mechanisms. Perhaps, indeed, the reason lodges back of the central nervous system." For our purposes, it is well to quote the author's 12th and 15th conclusions:

"Several groups of cases were classified from the distribution of macroscopic lesions, although the focal purity of these cases can often be brought in question from the results of microscopic examination (infrastellate gliosis and satellitosis also in macroscopically 'normal' areas).

"I. Pre-Rolandic group, including a superior frontal-prefrontal subgroup of paranoidal trend.

"II. Post-Rolandic group, including (a) post-central-superior-parietal (sensory-preceptual) subgroup in which katatonic features are the common factors; (b) occipital subgroup.

"III. Infrasylvian group (too small for clinical correlations).

"IV. Cerebellar group (katatonic features).

"The cerebellar-katatonic correlation is doubtless in line with some contentions of the Wernicke school, and obvious comments might be made in connection with the proprioceptive functions of the cerebellum (Sherrington)."

A résumé of the deductions which we can draw from this study of Southard's would again seem to bring us confirmation of the fact that speech functions cannot be separated in any of their elements from the development of the mind or intelligence as a whole. It would appear that the author himself has not been able to show conclusively that the apraxia and aphasia and other speech defects seen in dementia precox are due necessarily to lesions in any particular focal area in either hemisphere. For the first time we have an indication of the part which the cerebellum may play as the chief proprioceptive organ for the coördination of voluntary movements, includ-

ing speech. In all the catatonics of Southard's series, there was some degree of mutism, stereotypy, echopraxia or negativism of speech and movement. In a number of his cases, the cerebellar cortex and its dentate nuclei were seriously affected.

Southard, in conjunction with Caravan,[30] presented two cases of chronic lesion of the left angular gyrus which received the clinical diagnosis of dementia precox. One was a cyst of softening and the other a solitary tubercle. It is well to remember, however, that these authors did not report microscopical examinations of any other portion of the hemispheres, brain stem or cerebellum. Both the lesions found were in the left hemisphere. According to the summary, both patients at one time or another showed hallucinations of hearing and seeing, disorder of consciousness, confusion, incoherence of speech, mutism, impulsivity, fixed attitudes, mannerisms, violence and catatonic stupor. A sample of Case A's speech is "Sure I am somebody. I must be somebody. Can't you see there is no bed? As there is no bed there is no room." Case B showed grotesque shifting of ideas with some play on words. "There is your knuckle, bite it and its pinochle. Look at the Spanish-fly blister." Together with the speech disorders

of these cases, there were also emotional changes. These were volitional defects, and it is particularly stated by the writers that the patients showed little or no intellectual disorder. The conclusion is drawn that these two cases with findings in the left angular gyrus tend to correlate the clinical symptom complex of dementia precox reactions with catatonia with lesions in the left angular gyrus. This conception of these cases with no microscopical report available for study of other parts of the brain other than the immediate focal lesion found, is unfortunate, particularly in view of the further fact that Southard's larger series of cases do not by any means seem to warrant the correlation of clinical symptoms of catatonic mutism, etc., with focal lesions anywhere in the brain. It would appear plainly evident from a consideration of this autopsy material that the apraxias and aphasias and other speech disturbances occurring in dementia precox are not susceptible of correlation with pathological findings in any so-called centers for speech or for voluntary movement. It is noteworthy that Kraepelin[17] separates a large group of his dementia precox cases with marked apraxic and aphasic symptoms and calls this group "schizophasias."

Charles I. Lambert [18] reported some clinical

and anatomical studies in Alzheimer's disease and related conditions. Five cases were reported, one of which may have been a case of Korsakoff's syndrome. In all five of them, the author states that "among the more striking symptoms of these cases were the outstanding symptoms of agnosia, aphasia and apraxia. An exact analysis of these focal symptoms presented considerable difficulty, since to an impaired recognition, word understanding and expression, there were added a general dementia and an unresponsive resistive attitude." The author further adds, however, that there was "a complete loss of spontaneous speech without gross paralytic symptoms developing, so that we might probably conceive of the aphasic symptoms resting largely on the marked dementia present and somewhat in the nature of a mixed sensory-motor-transcortical aphasia. When apraxia symptoms occurred they were mainly of an ideatory nature. In relation to the aphasia and apraxia disorder, the absence of paralysis or even limited impairment of motor function is of interest and should be remarked in relation to a differential diagnosis." In one case there was a right hemianopsia. It does not seem necessary, in view of these statements by the author, to give illustrations of the aphasic or apraxic

status of these cases; but a close study of the clinical findings leaves no doubt that both these conditions were present.

The anatomical findings in these cases are again brought to bear as an aid to our contention that intelligence in its widest sense, apraxia and aphasia are one and the same process, that they develop together and disintegrate together. Quoting from the report of the autopsy findings, particular note is made of the fact that generally distributed or circumscribed brain atrophy is present with the complete absence of arteriosclerotic change in the vessels to account for it. "The central convolutions in all cases appeared almost normally full, the frontal convolutions atrophic, the temporal, parietal and occipital convolutions rather strikingly atrophic in two cases, particularly so in three of them." No areas or foci of softening or gliosis were seen or felt. Sections of the vessels shown by the author present no changes in any of the cerebral arteries or their branches of an arteriosclerotic nature. There were changes in the muscularis and in the perivascular lymph spaces. Microscopically, gitter and granular cells were found. No mesodermal cells were found in these spaces or in the pial spaces in the cortex. In the various layers there were changes obscuring the normal

layer formation, reduction in the number of cells and an increase in the neuroglia elements, closely corresponding to the degree of atrophy present in the macroscopical examination. The fewest changes were found in the central convolutions; the largest degree of change was found in frontal, temporal and occipital lobes and in the cerebellar cortex. In the nuclei of the cerebellum the changes were also abundantly evident. Plaques of degeneration were also found in the spinal cord, in the brain stem and in the basal ganglia. The plaques were found evenly and symmetrically distributed throughout the two hemispheres in two cases, and asymmetrically distributed in two other cases. In one case (the author's case No. IV) it is said that the left parietal lobe in the angular gyrus shows a maximum degree of macroscopical and microscopical change. In this case the first layer of the cortex in the area mentioned showed changes which were quite striking. It is of interest to note that in this case the first evidences of disease appeared in the sixtieth year "with a change in his personality and declining memory. He failed to recognize old friends, was vacillating in his mood, sometimes agreeable, again irritable and obstinate. He ate ravenously and wet and soiled his clothing." In talking, the patient repeated

many words and at times was unable to finish what he was saying. His talk was a jargon or gibberish. This case had a right hemianopsia but no paralyses. Further examination shows that this patient "paid no attention to simple commands. He shook hands when the physician held out his hand, put out his tongue when told to do so, tried to open a knife, and when given a clock said '20-100-100-20'." This patient was aphasic and paraphasic, but not distinctly apraxic. That he had agnosia, however, is shown by the fact that one day he put his hand into the hot stove without appearing to know what he was doing. He did not know what food was for, had difficulty in carrying it to his mouth and had to be spoon fed. The inference is that he tried, however, to carry the food to his mouth. He is said to have had difficulty in handling a pencil when asked to write, but no mention is made of the fact as to whether he could write before his trouble. On occasions he put on a sheet as a shirt and would pull and haul at the bedding by the hour. The autopsy in this case showed more particularly a lobal atrophy involving the left parieto-occipital lobe, although there were brain changes in practically all parts of the brain. One can notice even with this author, who seems to have made a very careful anatom-

ical study of these cases, the tendency to correlate a clinical finding with what he considers the most important pathological lesion without due regard to the conditions in the rest of the brain.

Without going any further into the pathological and anatomical studies of these cases, we have, therefore, five patients suffering from Alzheimer's disease characterized chiefly by aphasia, apraxia and agnosia, which, instead of confirming the presence of focal and diagrammatic cortical localization areas for speech and voluntary movement, rather seem to confirm our contention, which we wish to repeat, namely, that the function of speech and voluntary movement is an integral part of the development of the functions of the entire brain, and is incapable of being anatomically, physiologically or psychologically separated into centers.

CHAPTER IX

DISTURBANCES OF SPEECH IN THE FUNCTIONAL CONDITIONS, EMOTIONS AND FATIGUE

The disturbances in speech seen in the various functional conditions, such as the neuroses, the hysterias, fatigue and the emotions, are of great interest in linking up this function with that of the general intelligence of the individual. Trigant Burrow[4] has attempted to correlate the development of hysterical manifestations in a phylogenetic way with the development of speech. He develops the conception that the origin of language was mimetic and that the language of primitive races was essentially instinctive and gesticulatory. The connection between inner thought and outer action is inherent and inseparable, and language, as it developed, was used to disguise whatever was unpleasant. He also draws an analogy from this for the mechanism of the production of hysteria.

In hysteria, the symptoms develop as a disguise of unpleasant conflicts, desires and strivings. At any rate, it is certainly true that very few cases of profound hysteria exist

without some sort of speech defect. Whether we believe with Freud and his followers that hysteria is a conversion mechanism by which special subconscious conflicts are converted and enervated into somatic symptoms, or whether we believe that the symptomatology of this condition is due essentially to a psychical dissociation and an oversuggestibility to inner or outer influences, is, for the purposes of this discussion, not important, nor is it important to believe with Burrow that hysteria is a phylogenetic reaction, or with others that it is essentially a mechanism of reaction to fear and other unpleasant emotions. Whatever the mechanism of the production of this condition, it is nevertheless true that all sorts of speech manifestations occur which vary from complete aphonia or anarthria to stammering, explosive or halting speech or even slight dysphonias and inconstant aphonias. It is also important to note that hysteria occurs in individuals with unstable emotional control possessing also defects in the volitional and inhibitory spheres. Two cases illustrative of speech troubles seen in hysteria are given as follows:

The first was a man 40 years of age, single, a carpet layer by occupation, who on June 2, 1917, entered a carpet beater to extract some carpets. This machine is a large steel cage

which is whirled around by electricity, so that the carpets contained therein may be mechanically agitated and thus freed of the contained dust. In some manner he was locked in the cage, the machine was started and he was unable to get out. He was whirled around inside of this cage and knocked from side to side, for about half an hour, the carpets saving him from being mashed into a pulp. All the time he was in the cage he yelled and screamed in terror for help. Finally he was released, and bleeding from a scalp wound on his head staggered from the cage supported by some fellow workmen. He trembled and quivered with fright, stuttering and stammering fearfully. Suddenly, breaking from his rescuers, he ran out into the street; he was caught and brought back with great difficulty. He was finally calmed and taken to a hospital where his scalp wound was stitched; he did not speak at all for three hours after the accident. Following this, he returned home trembling, fearful and very much upset. That night he lost his voice entirely and did not speak for several days. The neurological examination disclosed no palsies anywhere and absolutely no evidence of paralysis in the distribution of the 9th, 10th and 12th nerves, so there was no physical reason why he could not speak. There was no

evidence of a motor or sensory aphasia, no paralysis, and absolutely nothing organic to explain his condition. At the end of the third day when he began to speak he talked in a harsh whisper. He suffered from generalized tremor. Eventually, he was able to speak more strongly, but he has continued to this day with a defective speech characterized by harshness, quick changes in tone, pitch and cadence, halting, explosiveness and tremulousness. He talks like one who is in the throes of a terrible fear, and speech is associated with an increase in the generalized irregular tremor throughout the body.

Another case is that of a young man of 20, who fell from a scaffolding while painting, a distance of about 25 feet, and sustained what was said to be a concussion of the brain. He was not unconscious following the injury, but was stunned and dizzy. He went home unassisted and several days later became affected by an impairment in his speech. There were syllable stuttering and tonic and clonic stammering. At times, however, when the patient's confidence was gained and he became greatly interested in a conversation, he would speak several sentences without any trace of stuttering whatsoever. On several occasions he made attempts to return to his work. Not only did

his speech defect become worse, however, but he trembled and became so nervous and excited that he had to give up working. Treatment in both of these cases was of very little benefit, and neither one of them has improved very much.

Everyone who has been fatigued will notice how slow all his mental processes become. Associations are awakened slowly, recollections are delayed, and active thought production is difficult. So also the expression of thought will suffer, the speech becomes slow, colorless, lacking in emotional tone, and there is a slurring and sliding in the speech with paraphasic inability to choose the proper words and phrases. In this connection, it is pertinent to refer to the five cases of pseudoparesis developing in young soldiers reported by J. Ramsay Hunt at the November, 1916, meeting of the New York Neurological Society. Hunt described five young individuals who broke down under the intensive training, and the physical and mental stress incident thereto, in a cantonment where he had them under careful observation. The symptomatology suggested paresis. There was tremor in the muscles of the face and tongue, sluggish reactions to light in both pupils, elision and slurring speech and syllable stuttering, marked memory and atten-

tion defects and a general slowing of the mental faculties of rather rapid development. The serology was negative, and he was able to exclude syphilis, to the satisfaction of himself and everyone who heard him. All these five cases recovered under rest and proper treatment, the defects in speech and intelligence cleared up hand in hand, and all of them returned to their duties perfectly well at the end of a month.

Hoch[11] calls attention to what he terms the lowering of mental tension in fatigue and the aphasic and paraphasic characteristics of fatigue thought processes and speech. An amusing incident is brought to my mind of the suspicious and alarmed actions which an intern on my service in one of the large hospitals in this city showed when he heard me speaking about a case which we were discussing at the bedside. I had been under a very prolonged physical and mental strain and was very much overworked. The journey to the hospital had been very tiresome, and I was extremely fatigued. I had anticipated an easy hour of round-making, but case after case of interest had been called to my attention, and we finally arrived in the ward where a patient lay in whom the intern was very much interested. He asked me to make the examination with

him and help him in the diagnosis. The patient, who was a paretic, presented a very beautiful illustration of perseveration of speech and echolalia. In using the test words to bring out also the typical paretic speech defect, the intern was very much horrified to see that I butchered the test words in a much more striking manner than did the patient himself. Everyone who has been fatigued has had an experience of this kind. Public speakers with a reputation for clear diction and beautiful enunciation will often notice the difficulty they have, when fatigued, not only in thinking but also in speaking. Famous public orators often complain that the last speech which they make at the end of a hard night of campaigning is always the most unconvincing, dull and poorly enunciated. One such speaker has described very vividly to me his feelings at such times. He recounts how he starts out after dinner at his first meeting full of vim, the thoughts following one another in quick association, with logical sequence and telling effect on his audience. Toward the end of the evening, at some times earlier than at others, as he gets tired, although he repeats with very little variation the same speech which he has delivered earlier in the evening, the thoughts expressed are not so logically and clearly put

and do not follow each other with the facility by which his earlier talks are characterized. He has on a number of occasions become panic-stricken when confronted with the difficulty of pronouncing words which he ordinarily speaks with great distinctness and clearness. Words are slurred and mispronounced and he finds it impossible to give them the proper emotional emphasis. Often on such occasions he has to cut short his discussion and give up for the evening.

The emotions also play an important rôle in the development and expression of speech. The inhibitions and accelerations of speech and thought production which occur under emotional stress are known to all of us; even on the motor side of speech the element of emotional tone plays an important part. Indeed, it is almost impossible to separate this element from intellectual speech. It is inconceivable that any motor formula alone can be responsible for speech when one considers only this single influence of the emotions in speech. The simple words "No" and "Yes" can be delivered so that a variety of meanings may be implied and understood. Skilled actors are able to throw into the mechanical production of speech feelings of all shades, and one may often be thrilled to passionate heights merely

by listening to the clever play of emotional tone which such artists are wont to use in their delivery. The emotions, therefore, together with the rest of what goes to make up the mind, are inseparably connected with speech, not only on the intellectual side but also in the motor expression of that function. Hughlings-Jackson [12] seemed quite satisfied that the right hemisphere controls the production of emotional and automatic speech. He and others have pointed to the fact that even with extensive destruction of various parts of the left hemisphere, aphasic and some apraxic individuals were able to perform involuntary movements or make involuntary utterances which could not be repeated voluntarily. Such individuals will use an explosive "No" or "Yes" in assent and dissent, or will swear volubly when confused or angered but will not be able to repeat the swear words when asked to, even from dictation. One of my patients, J. M., used in Chapter III, to illustrate the presence of intelligence defects, when exasperated one day, swore a blue streak in perfect English but was never able to repeat the swear words.

In this connection, it is well to call attention to the emotional lack of control of certain aphasics. There are two patients now in the wards of the Kings County Hospital who

show this very beautifully. One is an old man with a diplegia who is prone to prolonged attacks of involuntary laughter or crying, leading almost to syncope from exhaustion. The proper emotional responses are seldom or never given correctly. For instance, one day when he was quite sick his wife came to console him. She was very much touched and cried as she spoke comforting words to him. All during her visit he laughed very forcibly and uncontrollably, his face becoming suffused and his breath coming in gasps, until complete exhaustion caused his involuntary laughter to cease. On another occasion, as a test, I told him some supposedly very amusing stories. He immediately began to cry, the tears rolling down his cheeks, and did not stop for a half hour or more. When questioned, he explained very carefully that he did not desire to cry when he cried nor to laugh when he laughed, that he really felt like doing neither but was unable to control his emotional responses. Sometimes when he simply desired to smile, the smile soon developed into a loud, gradually increasing guffaw and finally into convulsive laughter. This patient was almost wordless. He could say only "Yes" and "No," using both these words properly for assent and dissent. He could read print and could write from dictation and

from copy, and could explain in writing very well what he had read. He could write with his (left) paralytic hand and showed a very beautiful mirror writing. He saw and understood what he saw. There was no hemianopsia. He heard and understood what he heard. He recognized the names and uses of objects, and could do simple sums of addition spontaneously. The difficulty seemed to be chiefly on the motor and emotional side of speech. The patient was right-handed.

Another patient, in the same ward, was a Portuguese sailor with a left hemiplegia. This patient never cried regardless of what was said to him. He was always ready for a smile which soon grew into a fit of uncontrollable laughter. The history in this case is not reliable because he was brought to the hospital wordless and paralyzed and no relatives have ever been found. We do not know whether he was right-handed or left-handed. He speaks a senseless jargon spontaneously. He uses properly several simple phrases. For instance, he will say in French or in Spanish, "How do you feel?" "I feel good," "Yes," "No," "I feel fine," "I thank you." In English he can say, "I am all right," "Yes," "No," and "My name is Emanuel; I am a sailor man." This is as far as his spontaneous speech production will

allow him to go. There is no hemianopsia, but there seem to be visual and auditory agnosia and paraphasia. He is unable to recognize the names of various objects seen and heard. He cannot write, and attempts at getting him to read are met merely with guffaws of laughter. He performs simple imitative movements, can light a cigar properly, will wind a watch on command, will extract a match from a box. There is no tactile agnosia. This patient has never been known to cry during his entire residence in the ward. He will laugh even if things are stolen from him or if he is berated and scolded. These two cases show quite clearly the intimate relations between the emotions and speech.

In conditions of great emotional stress, speechlessness is very often seen; fright, anger and great joy will very often impede speech, particularly in women. These emotions may have a contrary effect on speech; but whatever the individual reaction is, speech is always somewhat affected in conditions of stress. Some persons who are habitually accustomed to speak quickly, under the influence of rage will talk quietly, slowly and incisively. Others will speak rapidly, ungrammatically, stumblingly, with stuttering or stammering of various types.

CHAPTER X

THE INFLUENCES OF MIMICRY ON SPEECH

That auditory word images, visual word images or kinesthetic word images are not essential in producing speech, can be seen, aside from the psychological considerations mentioned, from the following points. People who have the faculty of expressing themselves by means of changes in tone and modulations of voice developed to a high degree, find it possible to transmit meanings by such manipulation of spoken words which the utterances themselves may belie. For instance, talented actors, by voluntary control of speech, are again and again able to impart to the simplest phrases importance which others cannot do. This, of course, does not altogether depend on the quality of the receptive auditory apparatus of the hearers. Whether one catches the meaning imparted to these words, other than the words themselves, depends almost entirely on the attention and degree of insight which the hearer possesses. As an example, it is possible for a talented individual, and for many others, uttering the words "I hate you," even in the

dark, where associated gestures cannot be called upon to aid in giving a contrary meaning to this phrase, so to say these simple words as to convey to the hearer exactly the opposite state of feeling on the part of the speaker. It can be seen, therefore, that the hearer must be exercising in the fullest possible way all the functions of the intellect in this particular case, especially the ability to interpret the emotional elements of speech, in order to obtain the intended meaning of the speaker.

Gestures, of course, are a great aid in speech. Calling again on examples seen in everyday life for illustration, all of us will readily recall occasions when a shake of the head or movements of the eyes, of the face, or a general attitude of the body during a conversation, meant more than the actual words spoken. Indeed, it is possible to convey a great many thoughts in this form of speech without the actual utterance of a single word. Shall we exclude, therefore, this form of thought communication and expression from the category of speech? It seems, rather, more logical to incorporate this method of communication between humans as an integral part of the speech mechanism.

In analyzing the processes through which the individual conveys thought by means of gestures or changes in tone and cadence of

spoken words, one cannot say that kinesthetic memories, visual memories or auditory memories of words are being used to help either the user of these forms of expression or the hearer in the understanding of them. It is impossible to analyze separately these various images in speech because they are not, according to our view, analyzable into separate components. They are all part of the general functions of the mind.

It has often occurred to us to ask of the psychologists who would have us believe that various sorts of images are necessary or essential in diagrammatic operation for the proper perception or expression of thought, how they explain the understanding of difficult abstract or scientific propositions as laid down, for instance, by teachers in giving instruction to their pupils. A professor, in expounding the theories of the physiological principles involved in the action of the glands of internal secretion to a group of medical students who have never heard anything about them before and have never seen them, is able to give an accurate exposition of his own thought so as to make clear to them the knowledge which he wishes to impart. It is impossible to say beforehand whether such students are by preference so-called visualists or auditifs. The impressions

received by the students and the amount of understanding of the matter which they have of such impressions received, depend entirely on the degree and quality of their general education or general individual intelligence, and not on the calling up of any specific images. All the faculties of the mind must be used in receiving these propositions as they are being laid down by the teacher. It is inconceivable, as some would have us believe, that certain individuals arouse auditory word memories of what is spoken to aid them in the understanding of speech, or that others arouse by preference visual images of these matters. It would seem more in keeping with the probable truth that all possible sources of mental activity for the reception and storing up of impressions are used in such a case. If this were not so, the understanding of thought as it is expressed in speech could be made a simple matter, reducible to a measured capacity for imparting knowledge and for its reception, by means of the stimulation and production of various sensory images, whereas, as a matter of fact, we know that the ability to impart knowledge by means of speech and to acquire it by means of thought depends entirely on the individuals who are teaching or receiving this knowledge.

All these considerations would seem to point to the fact that general intelligence is the basis of speech and not any separate faculty of the mind. As a corollary to this, it must of necessity follow that no particular part of the brain and no group of centers can be said to control the mechanism of speech production in its broadest sense, although the control of the motor part of the speech function may very well be limited in a poorly circumscribed manner to a region adjacent to the motor area in the cortex for the muscles of phonation, voice, lips, etc.

CHAPTER XI

CEREBELLAR SPEECH DISTURBANCES

As examples of speech disturbances due to injury, disease or maldevelopment in the afferent cerebellar tracts, we have the difficulties of speech seen in Friedreich's ataxia. As illustrations of defective speech seen in injury, maldevelopment or disease in the efferent cerebellar tracts, we have the heredo-cerebellar ataxias and the cerebellar types of catatonic dementia precox. Speech disturbances seen in the cerebro-cerebellar conditions are best illustrated by the cerebro-cerebellar diplegias and in some cases of multiple sclerosis. As evidence of speech troubles, using the word speech in the widest sense always, we also have the conditions which depend on interruptions in the tracts known as the extrapyramidal, namely, those connecting the corpus striatum and the red nucleus and, therefore, indirectly, the red nucleus and the cortex by means of rubro-cortical or cortico-rubral tracts, and the cerebellum by the superior peduncles. Illustrative of this are the speech disturbances seen in paralysis agitans and Wilson's disease.

Friedreich's Ataxia.—Here the disturbance seems to be, in the beginning, at any rate, one of loss of coördination explainable on the same grounds that the ataxia in the muscles of the trunk and extremities is explained; that is, that the afferent impulses to the cerebellum are cut off in the case of speech from the muscles of phonation and articulation. When these muscles are set in motion during utterance, ataxia occurs, resulting in the so-called "hot potato" speech. One of my patients described her difficulty by saying that she did not seem to be able to get her tongue and lips in the right position to speak properly, and at times she felt that it was impossible to speak at all because of this difficulty; but if she exercised a great deal of care and voluntary effort in speech and spoke slowly, she was able to speak better. The speech disturbances in this case consisted of a peculiar, slow, colorless, unemotional character in her speech, with changes in tone and pitch, occurring often in the same sentence, but nearly always in the course of speaking several sentences. It was quite customary for her to speak in a fairly even, slow, monotonous tone the first few words of the sentence, and then to speak rapidly and in a different tone and to end finally in an entirely different pitch. There would be considerable volume to the

speech at first, but this gradually tapered off as though the patient were tired. The most important thing seemed to be the entire lack of color in the speech, there being no emotional manipulation of speech possible whatsoever.

Another patient in the same ward had a speech which is described as halting, distinctly ataxic, with remarkable changes in pitch and tone occurring in the same sentence. Her voice also grew very weak towards the close of a long sentence, and words were spoken almost in a whisper at the end. Her speech was also colorless so far as emotional tone was concerned.

While both of these patients were advanced cases of Friedreich's ataxia, the peculiar character of the speech disturbance when it occurs early in this condition was seen in a boy of 12 years whose symptoms were only of three years' duration. The emotional tone in this boy was well preserved, and it was possible for him to show in his speech the quality of the particular mood in which he happened to be while talking; but throughout the speech there was an unsteadiness in holding sounds, more particularly vowels, as in Ah and A. These vowel sounds were pronounced in a very tremulous and unsteady sort of way, with changes of definite character in tone and pitch.

It is interesting to note that in the first two cases, which were advanced, there was an associated secondary mental defect due quite possibly to the narrow institutional life which they led. In the boy, however, this was not observed; he was bright and did well at school. The first two, however, were distinctly retarded mentally in all intellectual spheres.

One other case of Friedreich's ataxia with speech disturbance is that of a young man of 26 whose symptoms began 18 years ago. He has the typical "hot potato" speech, talking as if something disagreeably hot was being held inside the mouth which had to be pushed from place to place during speech. Aside from this defective feature his speech is also slow, halting, with a tendency to unevenness in tone and is absolutely colorless emotionally.

Three other cases of this condition observed by the writer showed no disturbances of speech whatsoever. They were very early cases, the symptoms being of not more than three years' duration.

Heredo-cerebellar Ataxia.—Three cases which I have personally observed of this condition occurred, one in a child of nine and the other two in brothers who were more than 40 years of age. There was also a sister who suffered from the condition, but she had no trouble

with her speech mechanism. All three of these patients had the characteristic monotonous, halting, colorless type of speech, which is a great deal like the speech of the paralysis agitans cases. No matter how great the distress or the joy of these patients, they seem unable to convey an inkling of their emotional status through manipulation of their utterances. The words are pronounced slowly with a halt between each word or phrase, but there are no changes in pitch or tone. There is a tendency to slurring of sibilants and a strange lack of expression, not only in their voices but also in their faces. In this respect, I have been able to see very little difference between the speech disturbances of paralysis agitans and cerebellar ataxia. In the two brothers, there was a considerable degree of dementia of the organic type, defects being present in all mental functions.

Cerebro-cerebellar Conditions.—In cerebro-cerebellar diplegias, in dementia precox and also in multiple sclerosis, it has been definitely established that disease processes may occur in both the cerebrum and the cerebellum. In cerebro-cerebellar diplegias of profound type such as one sees in cases of accidental feeble-mindedness following difficult labor or other injuries or diseases soon after birth,

there is very evident agenesis in both the cerebral and cerebellar cortex. It is rather common in these cases to have as an expression of the profound mental retardation an entire absence of speech. In milder types where the agenesis or defective development due to injury is not so profound, speech is retarded in direct proportion to the degree of brain atrophy or injury. The following case is illustrative of the former type and is reported in full from the records of the case taken by Dr. O. S. Strong, not only because it serves as a good example of the condition mentioned, but also because it is of interest owing to the fact that speech has not developed at all even though the child hears perfectly.

A. A., male, five years old. Is one of twins. After a normal labor the child appeared to be all right. When fifteen days old, it began to have attacks during which its face would become blue and its body would be bent in a position of opisthotonus. These attacks occurred for two months; they were in the nature of generalized convulsive seizures and always began with a sharp cry. The child's body would be contracted tonically and then would bend backward, resting on the back of the skull and pelvis; the face would become very blue and the whole attack would last

about ten minutes. Occasionally the attacks consisted merely of spasmodic crying with blueness of the face without convulsions. He had altogether three severe convulsions and many crying spells with blueness. Thereafter, the child appeared to develop fairly well, although he did things awkwardly and performed voluntary movements with a great deal of tremor and trembling. He did not creep until he was two years old and did not walk until he was three. Investigation shows that deliveries of both this boy and the twin were normal in every respect. The first teeth appeared when the child was about sixteen months old, three months after the normal child had his first teeth. The affected boy began to make sounds when he was four years old, but never distinctly, so that his vocal sounds were never understood as words. There has always been more or less slobbering, and the child has been irritable and hard to manage.

On the back of the child's head there are two large bony prominences, the one on the left side being much larger than the one on the right. The head is distinctly microcephalic and asymmetrical. The affected twin is in every way smaller and less well developed than the normal one.

The neurological examination shows that all the cranial nerves are normal. The fundi are normal. The pupillary reactions are physiological and hearing is normal to both air and bone conduction. The boy walks in a very insteady manner with the legs widely spreads and with unsteady, jerky movements of his upper extremities and trunk. In stopping there is a tendency to fall to either side. He also stands very unsteadily when his feet are put together, even with his eyes open. All the superficial reflexes are normal; the muscle strength seems to be normal, although coöperation was so poor on account of his mental condition that nothing definite can be said about this. There is a tremor in both upper and lower extremities of the intention type; irregularly ataxic and choreiform movements are brought out very easily. There is distinct equilibratory and non-equilibratory coördination in both upper and lower extremities. There is marked adiadochokinesis. There are no sensory changes. Guttural sounds are made but no understandable word has ever been spoken. In other words, there is a very low grade of imbecility. The child is extremely emotional, bad-tempered, and not amenable to discipline, persuasion or correction.

The speech disturbances in dementia precox

have already been mentioned. Reference will be made here only to the fact that Kraepelin [17] has recognized a distinct form of dementia precox, which he calls "Schizophasia," in which there are very marked changes in speech with aphasic and paraphasic characteristics. Kraepelin also dwells on the fact that speech disturbances may and often do occur in all types of dementia precox. The cerebellar findings in this condition by Southard and others should also be borne in mind.

Multiple Sclerosis.—In this condition the speech defect is probably a cerebellar disturbance. Indeed, in the paraplegic types of multiple sclerosis with the difficulty limited fairly definitely below the bulb, speech disturbances never occur. The opposite, however, is true where the sclerotic plaques are located either in the medulla or in the cerebrum and cerebellum. The speech disturbances range from absolute anarthria or aphonia to varying degrees of dysarthria and dysphonia. In these two types, the lesions are undoubtedly situated either in the cortico-spinal tracts and their afferent fibers to the cranial nerve nuclei, or, very rarely indeed, in the nuclei themselves; in the former case the speech is a spastic difficulty. The typical speech of multiple sclerosis, however, is not of the bulbar type but has

more of the cerebellar characteristics; namely, it is monotonous, peculiarly ataxic, colorless and emotionless, and shows frequent changes in tone and pitch.

F. W. Scripture [27] makes a study of the records of speech in disseminated sclerosis by the graphic method. In his conclusions on the nature of sclerotic speech, he notes the peculiar vibrations, irregular rhythm, monotony, etc., and attributes these qualities of the sclerotic speech to ataxia. When thickness and explosiveness occur in the speech, these symptoms are attributable to spasticity in the muscles of enunciation and are in no wise different from the speech defects seen in infantile cerebral diplegia or ordinary hemiplegia. In brief, Scripture attributes all the difficulties of speech in multiple sclerosis to ataxia or disturbance of tone or a disturbance of breathing, which might result in "staccato speech." He insists that scanning speech never occurs and explains that the disturbance of rhythm in the speech of multiple sclerosis which simulates scanning is explainable by an anataxia of breathing. Scripture distinctly states that syllables are not separated by pauses in this condition. This author feels that the laryngeal ataxia responsible for most of the speech disturbances in multiple sclerosis occurs also in Friedreich's

disease and in some of the other conditions not yet thoroughly studied.

Paralysis Agitans.—In this condition there is characteristically found the same dull, monotonous, slow, but not halting speech, void of all emotional coloring, that is seen in cerebellar conditions without the true ataxia already spoken of in the speech disturbances of Friedreich's and multiple sclerosis. The speech is more apt to be stiff and is probably not wholly cerebellar and indeed may be in most part a spastic speech. Differentiating it from the speech of Friedreich's ataxia, is the fact that changes in tone never occur. There is a true monotony, with no halting or slurring, such as is seen in the heredo-cerebellar conditions.

In Wilson's disease, a report of a case of which is herewith appended, the speech is evidently gradually lost as a part of the general mental deterioration with final dementia. The motor quality of the speech is undoubtedly spastic. This girl spoke through her teeth, very stiffly, and the only sounds uttered were throaty, guttural noises, only a word here and there being recognizable.

S. F., female, aged 22, single.

Family History.—The mother, who is 45

years of age, is neurotic and suffers from headaches and metrorrhagia. The father died of tuberculosis (?) at the age of 30 and was a shiftless man who would not work. The mother of the patient finally deserted him and after his death married again. She had by Sadie's father three children, of whom the patient is one, the other two being a girl, 24 years of age, and a boy, 20 years of age, who are both said to be physically and mentally normal. The mother has had no children by the second marriage, and she states further that there is no history in any of the branches of her family of any illness such as exists in our patient. She knows very little about her first husband's family, and is not even sure that he died of tuberculosis, although she heard that he died from some wasting disease. The patient's mother states that the pregnancy as a result of which our patient was born was perfectly normal and uneventful and that the labor was easy and no untoward incidents occurred during the labor or the puerperium. The child walked at one year and progressed satisfactorily. She was bright at school and left when she was a little more than 12 because a cousin who brought her up thought it best that she go to work. She worked as a messenger girl in a dry goods store for several years and

then stayed at home to keep house for her mother.

History of the Onset.—The girl, having previously been perfectly well, when about 15 yeard old, suddenly fell to the floor and was carried unconscious to the bed bleeding profusely from the vagina or the rectum, her mother did not know which. It is said that the patient passed large clots of blood and that the hemorrhage lasted several hours. At the end of this time it stopped completely, but she lay in bed and was so extremely weak that she could not move her head and had to be fed. Several days later when she finally began to talk and move a little, she complained of a severe pain in her throat and in the upper part of the chest. This pain was so severe that she was doubled up, her head and knees touching during the paroxysms. She still remained in bed, but gradually the pain subsided somewhat only to return at the end of several weeks with increased severity. She was taken to the Long Island College Hospital at the end of two or three months. Soon after this, she became drowsy and would lie in bed for months without moving and in a semi-conscious state. During this period, the pains in the throat and chest were not evident, but

at the end of each drowsy period, they would return with increased violence, and during the attacks of pain, she would not be able to talk at all and was doubled up in a knee to chin posture, which was the only position in which she could obtain relief. During one of these attacks, she completely lost her voice and has never since regained it, not having spoken a word distinctly for more than four years. The dysarthria and dysphonia have remained stationary, there being no remissions whatsoever.

The tremor began three or four years ago. The grimace or spastic smile which the patient has, began about seven or eight months ago. Of late years, the patient has occasionally lost some blood from the rectum. On several occasions, her legs have become so extremely spastic that they have swelled considerably, although, as the patient's mother expressed it, "there was no dropsy and the skin did not pit."

The mental deterioration has been very rapid. The mother says that she is definitely sure that until seven or eight months ago the girl was mentally as keen as she ever was, despite the fact that she could not talk.

Inspection.—The patient is fairly well nourished, and lies in a spastic attitude, the

face being fixed in a fast grimace or grin, the legs stiffly extended, the head and trunk also being held rigid. Her movements are made with stiffness and tenseness in the muscles, which are quite noticeable.

Coördination.—On account of the great spasticity in the skeletal muscles, examination to bring out the coördinating status is not always satisfactory, but non-equilibratory tests, namely, the finger-finger, finger to nose, knee to heel and knee-heel tests, seem to be well done.

Gait.—The patient can walk a little with considerable spasticity and is apt to fall, especially when she attempts to run.

Skilled Acts.—Are very poorly performed because of the spasticity, and speech is extremely difficult. She cannot write because of the tremor. There is aphonia and anarthria, although at times she can speak one or two words and will hum songs, so they can be understood.

Reflexes.—All the reflexes are equal and active, both superficial and deep. There is no ankle clonus, although tapping of the patellar reflexes is sufficient to cause an extremely marked response with apparent clonus. The

superficial reflexes, including the abdominals, are present and equal. It has been possible, on account of poor coöperation, to examine this patient's plantar reflexes only during sleep; and on a number of occasions when this was stealthily done at night, there was the normal flexion of the big toe and no Babinski was elicited. There was no Oppenheim and no Chaddock. The abdominals were present and equal during sleep. Pupillary reflexes are equal and prompt, both as regards light and accommodation, although there is a weakness in convergence, the left ocular movements not taking part in this reaction, the eyeball seeming to remain fixed with the same degree of spasticity as the skeletal muscles. The corneal reflex is equal and active. There is no nystagmus.

Muscle Strength.—Does not seem to be impaired. The tendo achillis and the muscles of the wrist are held so rigidly as almost to constitute a deformity, and it requires great strength to overcome the spasticity. This is more marked at times than at others.

Associated Movements.—In talking or performing voluntary acts with the arms or legs or in attempting to rise from a prone posture, the whole body seems to take part

in irregular movements which are particularly marked in the face and in the arms and hands; these movements are athetoid-like.

Electrical Reactions.—Are normal.

Cranial Nerves.—There seems to be no palsy of the third, fourth or sixth, and none of the seventh or fifth nerves. Taste is normal. Sensation in the trigeminal distribution is normal. The optic nerve is normal and is not affected in any way. Hearing is acute, and there does not appear to be any paralysis of the palate, tongue or vocal cords, although it is extremely difficult to examine the throat, on account of the great spasticity and rigidity in the muscles of the mouth, face and throat.

Mental Status.—The patient is suffering from a real dementia and has reached the level of the low imbecile type. She is extremely emotional, cries and laughs involuntarily, although the elated mood is more frequent and constant. She is, at times, however, very vicious and will scratch and bite. She leads a practically negative vegetative existence.

When in complete rest, the tremor is observable and highly suggestive of the paralysis agitans type in the hands, but when the patient attempts to do something, she uncovers a generalized tremor, particularly marked in the

hands and head and trunk, of the intention type. The tremor is, at all times, rhythmical, has moderate force, and not very much amplitude and is very much increased by stress and emotion. The patient seems able, with coaxing, to control the tremor and at times is able to stop it altogether. It is not exactly of the intention type either, for the patient is able to drink out of a cup without spilling a drop. At the commencement of the act of grasping the cup and just before she is able to do this, the tremor is quite marked, but as soon as she grasps the cup and brings it to her lips, the tremor practically stops, and she is able to drink the contents without spilling them.

The roentgen-ray photograph is negative. Special eye examinations are also negative. Fluoroscopic examination of the liver has not been made because this method of examination is not available at the hospital. Blood Wassermann is negative.

Speech Disturbances in Tabes.—Walter B. Swift [33] brings up the subject of defective speech in tabes, which he differentiates from the speech disturbances seen in general paresis. The difficulties seen in tabes Swift ascribes to a "failure in the report to the central nervous system of the positions of the speech mechanism during utterance. In other words, speech

in certain utterances demands certain very exact and complicated coördinations in the speech mechanism musculature. Just these exact coördinations are impossible, due to a lack of sensory report of their position."

The speech in tabes which this author describes has a slurring, slovenly, inadequate quality, which vanishes when instructions are given to the patient to talk slowly so that enough time may be given for the report of the positions of the various muscles used in speech to the cortex. The author appears to be a little indefinite in his anatomy, referring the entire matter of coördination and position sense of the speech musculature to the cortex without especially mentioning whether he means the cerebral or the cerebellar cortex. Anatomically, we know that there are two connections, the direct or posterior spino-cerebellar tracts which are affected primarily in Friedreich's ataxia, and secondarily in tabes, and are only a portion of the afferent cerebellar connections of the middle lobe of that organ. An important portion of this pathway is received from the columns of Goll and Burdach, or at least, from their nuclei, and helps, with the direct spino-cerebellar tract and the vestibulo-cerebellar and the olivo-cerebellar connections to make up the inferior cerebellar

peduncle, so that not only in tabes, but also in the other conditions already mentioned, speech defects occur where the afferent cerebellar mechanism is disturbed, the position and muscle sense of the speech musculature being interfered with. It is needless to say how frequently, particularly late in the disease, mental defects occur in tabes—the taboparesis of authors.

All of the cases cited above serve to show that the cerebellum plays no little part in the proper coördination of the motor part of speech. It makes for plasticity of speech and for variableness of many of its physical characters. It helps and no doubt entirely directs the motor coördinations of the speech musculature, and the cerebellar speech mechanism when functionating properly enables man to speak quickly, to change the voice in tone and pitch, to impart emotion to the voice and, in fact, gives it its finer qualities. Is this all the cerebellum does? Following out our very broad conception of the speech mechanism, we feel that this is only a small part of what the cerebellum helps to do. We also feel that there is an anatomical basis for our views. In developing these views, however, we must have reference to our anatomical and physiological knowledge concerning the cerebellum.

CHAPTER XII

NEW CEREBELLAR FUNCTIONS

In abstracting what we already know of the functions of the cerebellum, and in recounting some of the anatomical points involved, reference is had to the chapter on the physiology of the cerebellum in Starling's [31] book, and to the fine little work by C. J. Herrick,[10] 1916. With that function of the cerebellum which has to do with the maintenance of equilibrium and other changes in the posture of the body not especially connected with visual and auditory stimuli, namely, the vestibulocerebellar mechanism, we are not at all concerned, at least not primarily. With the matter of coördination, however, we are entirely concerned. The coördinating mechanism of the cerebellum depends on the reception of afferent stimuli from the muscles, joints and ligaments of the body; but as we are only discussing speech, only the muscles of speech need be mentioned. This system of afferent nerves has been called (by Sherrington [28]) the proprioceptive system, because it is not excited by changes in the environment, but only by

changes within the body itself. Excitation o this system results in a regulation of the extent of motor discharge in the muscles and prevents over-action even though it may be evoked. It is a "compensatory reflex in an opposite direction to the reflex immediately excited from the skin." In this way, according to Sherrington, tone is maintained in muscles. Most of the speech defects, therefore, seen in tabes and Friedreich's ataxia are due to changes in tone and to disturbances of the regulative coördinative mechanism, resulting in ataxia and various types of dysphonia and dysarthria of ataxic character. The other proprioceptive part of the cerebellar mechanism, which really does not concern us in the discussion of speech, comes in through the vestibular tracts either directly to the nuclei of the cerebellum, or, first, to the vestibular nuclei and then through the inferior peduncle, to the dentate nucleus. This pathway also is mainly afferent.

The efferent pathway of this proprioceptive system is from the roof nuclei of the cerebellum, particularly the dentate, through the superior peduncles into the decussation of the peduncles which cross just below the roof of the aqueduct of Sylvius at the level of the red nucleus. The red nucleus is the first relay in this neuron; the second relay leaves the red nucleus and

crosses soon thereafter, forming by this recrossing the rubro-spinal tracts. The red nucleus, very evidently, therefore, is an important relay in this efferent cerebellar pathway, and has more or less important connections with the cerebrum which are both afferent and efferent, and with the corpus striatum by way of the ansa lenticularis. It is in the destruction by disease of this mechanism that we get the speech disturbances and other symptoms of those two extrapyramidal diseases known as paralysis agitans and Wilson's disease.

More important, however, than all these pathways is the enormous pallio-cerebellar pathway from the large association areas, particularly in the frontal and temporal regions of the cerebrum through the basis pedunculi into the nuclei of the pons, and thence by a second relay to the opposite cortex of the cerebellum. This pathway is known as the middle cerebellar peduncle, and in size is much larger than the area occupied by the pyramidal tracts in this region. The importance of this pathway is tremendous and its functions are shrouded in mystery. That it must be important is evident not only from its size but also by the fact, as given by Starling,[31] that there is a close association between the development of each cerebral and the opposite cerebellar hemisphere.

"Atrophy of one-half of the cerebrum brings about atrophy of the opposite hemisphere of the cerebellum." The tremendous size of the cerebellar cortices which are made up almost entirely (if we exclude the middle lobe of that organ) by this connection, also attests to the great importance of this pathway. No attempt has been made to go very thoroughly into the physiology or anatomy of this most interesting subject. Only those points which seem to bear on our argument are mentioned. The physiology of this region is also extremely interesting. Experiments by Ferrier [8] show that relatively much greater stimuli must be applied to the cerebellar cortex in order to get a motor response than is necessary to obtain the same degree of response by stimulation of the cerebral cortex. When such responses do occur, as a result of stimulation of the cerebellar cortex, they are always of limbs and large units of the body, not of single muscles or groups of muscles as when the motor cerebral cortex is stimulated. On the other hand, movements excited by stimuli from the nuclei of the cerebellum are not quite so widespread, and can be initiated with much slighter stimuli than is necessary to produce the same degree of movement when applied to the cerebellar cortex. It seems, therefore, that the functions of the

cortex of the cerebellum appear to be different from those of the nuclei of that organ. Horsley regards the cortex of the cerebellum as an afferent receptive center entirely, and the nuclei as efferent centers, which have to do with movements of the eyes and head, trunk and limbs, and are mainly concerned with the maintenance of equilibrium in response to alterations of the positions of the body.

While this gives us very little definite information about the functions of the cortex of the cerebellum, enough has been said to indicate that these functions are but little understood, and that they are for that reason probably of great importance. How important, may be seen from Herrick's [10] discussion of this matter. In correlating the importance of this cerebro-cerebellar pathway, we must turn first to this author's idea of the functions of the cerebral cortex (p. 298): "There is a specific localization of function in the cerebral cortex, 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.

"No cortical area can properly be described as the exclusive center of a particular function. Such 'centers' are merely nodal points in an exceedingly complex system of neurons which must act as a whole in order to perform any function whatsoever. Their relation to cerebral functions is analogous to that of the railway

stations of a big city to traffic, each drawing from the whole city its appropriate share of passengers and freight; and their great clinical value grows out of just this segregation of fibers of like functional systems in a narrow space, and not to any mysterious power of generating psychic or any other special forces of their own.

"The essence of cortical function is correlations, and a cortical center for the performance of a particular function is a physiological absurdity, save in the restricted sense described above."

Again on page 290, speaking of the association centers of the cortex, Herrick [10] says: "These alone are greatly enlarged in the human brain as compared with those of the higher apes. In the latter animals, the projection centers are fully as large as those of man, the much smaller brain weight being chiefly due to the relatively poor development of the association centers." Now we find that "the cerebral cortex is in direct connection with the red nucleus of the cerebral peduncle by a cortico-rubral tract, arising in the frontal region of the cortex, and by ascending fibers from the red nucleus to the same general part of the cerebral hemisphere. From the frontal, parietal, temporal and occipital association centers there arise large descending fiber tracts to

the nuclei of the pons (cortico-pontile tracts). These connections between the cerebral cortex and the red nucleus and pons put the cerebral cortex and the cerebellum into very intimate relations, but the exact way in which the cerebrum and the cerebellum coöperate functionally is obscure."

This pathway by means of the cortico-pontile tracts, or, as they are also called, the middle cerebellar peduncles, between these tremendously important and highly developed cerebral association centers and the mysterious cerebellar cortex, assumes a position of very great importance. In structure, the cerebellar cortex differs from the cerebral cortex in the form and arrangement of its neurons, the former being similar throughout in construction. The cerebral cortex, as is well known, shows marked differentiation in various places in the character, form and arrangement of its cells and neurons. We already know that the functions of the cerebrum are of the highest type, and, together, give us in man that momentous thing which we call the human mind, or intelligence, in the broadest sense. This much seems to be undoubted. The function of the cerebellum, from all its known physiology and undifferentiated anatomical structure, seems to be admirably adapted only to one thing,

namely, coördination and the maintenance of equilibrium. Why, therefore, should a different function be ascribed to the cerebellar cortex when considering its relations to the cerebral cortex? Why not the function of coördination, of cerebral mechanisms? Reasoning logically, therefore, if the cerebrum is the seat of the highest functions of intelligence, and if speech is nothing but the expression of this intelligence, then, of course, the function of the cerebellum in relation to the cerebrum is the coördination of the various mechanisms of speech and intelligence,—coördination, for instance, of thought production and the emotions, the disturbance and dissociation of which in abnormal states is best seen in dementia precox with all its attendant protean symptomatology. Stewart Paton is very fond of describing the mental defect in dementia precox as an intellectual ataxia, an illustrative phrase which may have in it more truth than convenience. Of course, this is entirely speculative. At least, there are anatomical reasons why this conception may be possible.

In the types of cerebro-cerebellar agenesis of cerebro-cerebellar diplegias involving injuries at childbirth, this conception finds clinical corroboration. These children show a disfunction in all the fields of cerebral and cerebellar

mechanisms, namely, of intelligence, motor and sensory disturbances, and gross defects or entire absence of speech. In the heredo-cerebellar ataxias, the same holds true to a more moderate degree. All writers on this subject, not only of Marie's heredo-cerebellar ataxia, but also of Friedreich's ataxia, comment on the regularity with which intelligence defects, amounting in many cases to dementia and imbecility, occur together with emotional and speech defects. In tumors of the cerebellum and of the cerebello-pontile angle, disturbances of intelligence of a profound character are often seen very early. In a great many autopsied cases of dementia precox reported by Southard,[29] previously discussed, there were marked changes in the cerebellar cortex and in the nuclei, so much so that Southard postulated a group of dementia precox cases on his cerebellar findings. In the great majority of all these cases, of course, the cerebrum was correspondingly severely involved. The evidence seems to point to a necessary correlation, therefore, of processes in both the cerebrum and the cerebellum, for the production of these grave speech-intelligence and motor-sensory defects seen not only in dementia precox but also in the other conditions above discussed. It is, of course, realized that nothing of a posi-

tive character has been contributed to this subject; the matter has consisted entirely of speculation on a clinical and anatomical basis.

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