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APHASIA

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APHASIA

by

S. A. KINNIER WILSON,
M.D., B.Sc. (Edin.), F.R.C.P. (Lond.),

Physician for Out-patients, National Hospital, Queen Sq.
Junior Neurologist, King's College Hospital
Consulting Neurologist, Metropolitan Asylums Board.

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PREFACE

To deal adequately with so large, intricate, and many-sided a problem as that of aphasia is impracticable within the limits assigned by the Editor for his series of "Psyche Miniatures." What will be found in the following pages is rather a general introduction to the study of the subject, which it is hoped may serve as a sort of *mise au point* for the physician who is interested in neurology, and for the psychologist who is not familiar with the clinical side of the question. An attempt has been made to indicate the separate and combined importance of the anatomical, physiological and psychological aspects of the aphasia problem; each makes its individual contribution, for which its own setting is appropriate, yet without a synthetic collocation of the data furnished by all three lines of approach no advance towards solution can be expected.

For the purposes of this book the author has in minor degree drawn upon his previous communications dealing with its topic, as follows :

1. "A Review of the Question of Aphasia," *Review of Neurology and Psychiatry*, 1909, vii, 151.
2. "Treatment of Disorders of Expression (Aphasia, Apraxia, etc.)," in *The Modern Treatment of Nervous and Mental Diseases*, edited by W. A. White and S. E. Jelliffe, New York, 1913, vol. ii, pp. 475–504.
3. "Aphasia and its Treatment," *The Practitioner*, 1915, xciv, 683.
4. "Aphasia," in *The American Oxford System of Medicine*, edited by H. Christian and Sir James Mackenzie, New York, 1921, vol. vi, pp. 243–259.
5. "An Introduction to the Study of Aphasia," *The Lancet*, 1921, ii, 1143.
6. "Discussion on Aphasia," *Brain*, 1920, xliii, 433.
7. "A Contribution to the Study of Apraxia," *Brain*, 1908, xxxi, 164.

INTRODUCTION

APHASIA as the expression of a diseased state occupies a unique position among clinical syndromes. No other is at once so clearly indicative of a partial outfall of intellectual processes and so definitely dependent on disorder of known cerebral physiological mechanisms. Its study ought therefore to be of precious value in throwing light on the interaction of the psychological and the physiological sides of brain activity. However marked the trend in some schools to investigate psychical function without reference to the organ of mind, this constitutes on the face of it a one-sided approach, and there should be no necessity for the veteran von Monakow to remind us that, as daily experience teaches, "every psychic process presents certainly a physiological effect in the brain, whether we will or not."

The field of aphasia should be a meeting place of the psychologist, the physiologist, and the clinico-pathologist, and great would be the

triumph were converging lines of approach to coincide with exactitude as they emerge on its common ground; but, unfortunately, the subject is one the study of which, complex enough in itself, seems fated to be rendered more confusing and involved by controversy and by terminological disagreement. Certain preliminary considerations are essential, not merely to an understanding of it, but also to knowledge of how to face it. Cases of aphasia are clinical commonplaces; the literature of aphasia is enormous; and theories to explain the phenomena are rife. Some, however, are mutually contradictory, while others are one-sided. The situation at present is perhaps one of a little uncertainty, for while 'classical' theories have been assailed, the views supposed by some to have replaced them have not received that general support which would indicate conviction of their truth. As an introduction to the aphasia problem we must take into account certain points, neglect of which has often proved a fertile source of misunderstanding.

I. Aphasia is not a disease, but a symptom. Like all symptoms, it is the expression of departure from a normal state, the external manifestation of disorder of function of a mechanism. When we speak of different types of aphasia, we do not refer to different varieties of a particular morbid entity or disease, but to certain groups of symptoms, all of which are disturbances of the function of speech—a mechanism in many respects, as we shall see, exactly comparable to other mechanisms with which the physician is familiar.

II. The function of speech is an intellectual function; the neural arrangements underlying its activity constitute a physiological mechanism; and the component units of the latter have an anatomical localisation or site. Thus the problem can be, and often has been, approached from anatomical, physiological, and psychological sides respectively and separately, and investigators have not always been careful to avoid inappropriate transference of terms or even of conceptions from one system to another.

For example, the term ' centre ' occurs with some constancy in descriptive accounts of aphasia, but its meaning has been so variable as often to lead to erroneous impressions. An anatomical ' centre,' an anatomical projection-area, may be exactly delimited, as in the case of the visual area in the calcarine cortex, the Betz-cell motor area of the precentral cortex, and so on. A physiological ' centre,' on the other hand, indicates the focus of functional activity of the particular system concerned, and does not of necessity correspond exactly with its anatomical correlate : a ' process ' is not localisable in the same sense as a morphological unit. As an instance, the cortical physiological ' centre ' for motor activity can scarcely be delimited as is the Betz-cell anatomical system. Abundant evidence goes to show that much more of the brain surface is concerned in motor functions than the anatomical strip in the precentral gyrus from which movements of the opposite limbs can be readily obtained by electrical stimulation, and from which the corticospinal or pyramidal paths,

whose function is certainly motor, start.

Again, the function of speech has a psychological aspect, and only too often has the writer on aphasia speculatively postulated psychical or ideational 'centres' which cannot possibly correspond to anything on the anatomical or physiological levels, but for which the same term is used.

For example, Lichtheim postulated a 'centre' for concepts apart altogether from the 'centres' of the physiological order (motor, sensory) concerned in speech, and Grasset[1] imagined a 'centre O' of the same psychological class, from which in his well-known 'polygon' diagram he drew lines to 'centres' of quite another sort. Yet Bastian, the author of one of the most logical and philosophical contributions to the study of aphasia, says, "after the most careful consideration of the subject, I believe that no cases exist which need for their interpretation the postulation of a separate centre for concepts."

[1] J. GRASSET, *Les centres nerveux*, Paris, 1905, p. 43.

It will therefore be seen how unsatisfactory are the various schemata devised by the ingenious to simplify the complexity of the cerebral speech mechanism. The plotting out of physiological centres on paper almost inevitably leads to the conviction in the student's mind that they stand for anatomical areas of the cortex, which is not precisely the case, while the drawing of lines of communication between these physiological centres and a 'psychical' or 'ideational' centre makes confusion worse confounded.

One need only scan the extraordinary collection of schematic diagrams representing the views of various writers on the question, happily gleaned and reproduced by Moutier[1] in his *Thèse*, to appreciate the force of the contention. The nomenclature is as bewildering as the schematisation, and one is reminded of the remark of Curnow, which Hughlings Jackson used to cite with approbation: "The tendency to appear exact by disregarding

[1] François Moutier, *L'Aphasie de Broca*, Paris, 1908.

the complexity of the factors is the old failing in our medical history."

III. While speech as a function of the intellect deserves and requires study along psychological lines, it must be clearly distinguished from thought. The two are not identical in the sense of being coterminous; and failure to appreciate this has proved another source of confusion. Pierre Marie and Moutier, for instance, whose contributions aroused the greatest interest at the time of their appearance, and since, are among those who seem throughout their work to identify thought and language. Now it is held, and with reason, that abstract thinking may proceed without utilisation of any of the 'symbols' of speech; one of the most convincing and interesting proofs of this has been furnished by the author[1] of *Mémoires d'un médecin aphasique*, as the following excerpt shows:—"Apart from a few words, such as 'yes,' 'no,' 'thank you,' 'if you please,' I could say and write absolutely nothing; not only could I say nothing,

[1] F. NAVILLE, *Archives de psychologie*, May, 1918.

but I had nothing to say, as far as the expression of words is concerned. Yet I had all my thoughts, all my conceptions; it was only my symbols that at that moment were lacking." In the literature of aphasia much is said of 'inner language' (*langage intérieur, innere Sprache, das innere Wort*). This latter German term is said to have originated with von Humboldt, who defined its meaning as the content and processes of consciousness, expressed by speech. Even so, 'inner speech' and thought are not synonymous; Henschen, to whose monumental work on the subject subsequent reference will be made, agrees with Hughlings Jackson in holding that "internal speech is not necessary to perfect logical thought." Further proof of these points has been well set forth by Mourgue[1] in a recent communication.

Notwithstanding the latest pronouncements by so prominent a psychologist as McDougall, who, in complete reversal of his position of

[1] R. MOURGUE, *British Journal of Psychology (Med. sect.)*, 1921, i, 97.

twenty years ago—when he held[1] that "each of these two bodies of doctrine, the physiological and the psychological, supplements the other . . . and the conjunction of the two kinds of research brings before us a number of problems of the deepest interest that remain hidden so long as we confine our attention to one or other of these sciences"—now maintains[2] that psychology must be studied without reference to the very organ which is essential for its existence as a science, I believe it is unwise, unprofitable, and, indeed, impossible, to divorce the psychological component of speech from its anatomo-physiological counterpart.

Speech is an intellectual process and behind the overt expression lies thought, but neglect of the anatomical and physiological basis of speech is largely responsible for the present uncertainty, for the psychologist is not interested in, or is tempted to ignore,

[1] W. McDOUGALL, *Physiological Psychology*, London, 1905.

[2] W. McDOUGALL, *An Outline of Psychology*, London, 1924.

the patiently acquired facts of cerebral localisation of function, and confines his attention to speech disorders as disorders of the psyche, any idea of attempting to 'localise' such being supposedly irrational. Yet when in actual fact a small area of softening in the left hemisphere renders a patient mute, when a similar small lesion makes it quite impossible for him to read, though he is not blind, the clinician must either forego all effort to correlate the lesion and the symptom, or, alternatively, and against those who conceive of psychical function only 'as a whole,' must hold and contend that local lesions of the brain may be, and often are, associated with partial disorders of a mental mechanism.

As we proceed we shall have evidence of the clinical occurrence of restricted aphasic symptoms, partial disturbances of that thinking in symbols which constitutes speech, and we shall see how they contradict the views of those who now assert, as does Head,[1] that no lesion, however local, can affect

[1] HENRY HEAD, *Brain*, 1920, xliii, 87.

speech and speech only, that alexia, agraphia, amnesia verbalis, etc., are "hypothetical conditions" which cannot be associated with limited destruction of any part of the brain.

Perhaps a preliminary word is necessary as to the term aphasia itself. It is obviously an unsatisfactory expression for many cases in which the patient still has speech of a sort; 'dysphasia' is scarcely if ever employed, but it is less open to objection, and the same remark applies to use of the terms agraphia and alexia. Dysgraphia and dyslexia are preferable. Further, inadequate attention has been drawn to the fact that, in Jackson's phraseology, an aphasic speaks badly "with what of his brain is left"; that is to say, aphasia is double-sided, symptomatologically speaking; the negative aspect is constituted by outfall of certain components in speech functioning, the positive by imperfect use of what is conserved. We have thus to take into consideration the mental level of the aphasic patient and the degree of dissolution of mental processes that has followed the lesion.

In other words, aphasics are not always easily comparable one with another; in fact, no case of the condition seems ever to be clinically identical in every respect with another, even though apparently of the same group.

Finally, by way of introduction, it is as difficult to define the limits of aphasia as a symptom, as it is to determine what exactly is to be included in the term 'speech.' It is the more necessary to mention this matter in our introduction to the subject because Head[1] in his recent studies of aphasia has adopted the position that the disorders of language produced by a unilateral lesion of the brain are never "exclusive affections of speech, reading, or writing." In another place he says that the function thus disturbed by an organic lesion "cannot be comprised obviously under the headings of speaking, reading, and writing; for not only may the loss of power to carry out any one of them be partial, but the disturbance extends beyond their limits and affects other mental activities." Now it may be granted at once

[1] HENRY HEAD, *l.c.*

that 'pure' cases of these disorders are doubtless rare, and that mixed varieties are the rule; for the sake of argument, one might for the moment admit that "inability to write is always associated with some other loss of function." It is, nevertheless, a clinical fact that one or other defect, in speaking, reading, or writing, may be by far the most prominent feature of a given case, so that while, for example, speaking and reading may be for practical purposes intact, writing may be all but impossible, as in a valuable case published in 1903 by Stanley Barnes,[1] and similarly with alexia from lesions in the distribution of the left posterior cerebral artery, as in a case of my own (unpublished), and with 'pure motor aphasia' as excellently shown by Dejerine. Even as regards the new terminology introduced by Head, he himself admits the terms "are not intended to define the limits of the disturbance," and in most cases "two or more of these

[1] STANLEY BARNES, *Review of Neurology and Psychiatry*, 1903, i, 531.

aspects of symbolic formulation are affected." In other words, since 'pure' cases of Head's varieties do not occur either, his argument for the abandonment of alexic or agraphic varieties on the ground of their never occurring in a 'pure' state cannot be sustained.

It is naturally impossible to say with exactitude where speech as a function of the mind begins and ends, but for practical purposes speaking, reading and writing, in their widest senses, do in fact cover most of the speech field. We shall see at a later stage that it is convenient in some ways to regard speech on the executive side as a form of the more general mental function of eupraxia, and on the receptive side as part of the more general function of eugnosia, and the corollary of this way of envisaging mental function is that aphasia as ordinarily understood is on the expressive side a form of apraxia, and on the receptive a form of agnosia. To this manner of approaching speech defects attention is directed below. Some of the ingenious schemes devised by Head for the study of aphasic

patients are clearly tests for the wider disturbances of apraxia or agnosia, and are included as tests for speech defects only by unduly enlarging our conceptions of what the term should connote. Neither their interest nor their value is thereby lessened, but their inclusion as criteria of the functioning of the speech faculty is an arbitrary extension of what is meant by speech, and one for which I can find no adequate justification.

A given aphasic symptom-complex may be looked at in three ways : What is the anatomical site of the lesion producing the defect ? What are the physiological mechanisms involved ? What is the nature of the psychological disorder ? No study of the subject can make any pretence at completeness if any of these three aspects is ignored, nor, on the other hand, can it be harmonised with clinical data if any one is elaborated at the expense of the others.

CHAPTER I

Anatomical Considerations

It seems superfluous to point out that by universal consent we are warranted in stating that in right-handed persons the lesions prone to be associated clinically with disorders of the function of speech are situated in the left cerebral hemisphere, and in a certain part or parts of that hemisphere. The simple fact that these lesions are largely unilateral (left hemisphere) is a truly remarkable circumstance, which fails to impress only because it is a commonplace of neurological knowledge. For here is localisation already, and of the most pronounced type; that the lesions underlying disorder of an intellectual function, that of symbolic thinking, are unilateral in the encephalon is the strongest argument that can be advanced for the à priori likelihood of

further localisation within that unilateral site. Head's view, that "the faculty of speech is not localised in any area of the cortex," is true only in the sense that psychical processes are not to be 'placed' by application of the criteria of physiological localisation, but if it is taken to imply the absence of a localised physiological setting for the arrangements that are essential to the faculty of speech, it is completely contradicted by the researches of Henschen,[1] who has collected and collated 1,337 cases of his own and from the literature, and the cumulative effect of this weight of evidence, pointing as it does to the existence of a cortical speech area, cannot possibly be ignored. Niessl von Mayendorf,[2] similarly, has analysed

[1] S. E. HENSCHEN, *Klinische und anatomische Beiträge zur Pathologie des Gehirns*. Vols. V., VI. and VII. Stockholm 1920, et. seq. The English reader will find a long and excellent resumé of Henschen's volumes in an article by Walter Schaller, *Archives of Neurology and Psychiatry*, 1925, xiii, 226.

[2] NIESSL VON MAYENDORF, *Die aphasischen Symptome und ihre kortikale Lokalisation*, Leipzig, 1911.

some 200 cases, and a glance at the ingenious charts published by him, in which the sites of the recorded lesions are indicated on a diagram of the left hemisphere ruled into squares, will suffice to show that lesions within a more or less well-defined cortical region, extending from the posterior part of the lower frontal lobe, by the island of Reil, to the temporal and lower parieto-occipital areas, are likely to reveal themselves clinically by aphasia in one or other form. In the so-called negative cases mere macroscopical examination is insufficient. The histological study of the cortex has reached so fine a pitch that no case can be called 'negative' if this has not been undertaken.

Whether localisation of lesions within this speech area, to correspond to clinical varieties, can be established is regarded in some quarters to-day as doubtful. The time-honoured division of aphasia into motor and sensory types—a physiological classification—recognises the existence of two differing groups whatever be the terminology adopted, and it is legitimate, physiologically, to distinguish efferent

from afferent impairment of function. Whether it is useful in the question of aphasia may be another matter. As far as speech is concerned the doubt is whether one type of symptom ever occurs without the other, though it be in varying proportions. According to this 'global' theory large lesions in the speech area are followed by gross aphasic disorders and small or limited lesions by limited aphasic disorder—in each case an aphasic disorder as a whole. The occurrence of so-called 'pure' cases of the condition—*i.e.*, of one or other clinical variety to the complete exclusion of the symptoms of another—is due, perhaps, to an unconscious trend towards schematisation ; the more thoroughgoing the clinical examination the less likely are such cases to be recorded. It is a fact, none the less, to which attention has already been directed, that in a given case disorder of speech may be largely limited to the receptive or to the expressive side, or indeed to but one aspect of reception or expression. I regard it as amply proven that lesions towards the anterior part of the speech region are

followed by aphasic symptoms in which defect of expression predominates, and those towards the posterior part by aphasic symptoms in which defect of reception is pronounced. Further, in many instances localisation within these general anterior and posterior areas has been rigidly demonstrated, illustrations of which will be found on a later page. There is justification, moreover, in seeking limited lesions for minor or minimal aphasic disorders, though no precise correspondence between size of anatomical lesion and amount of impaired physiological process is to be expected, for reasons already mentioned. The clinical symptom of astereognosis, or inability to recognise the qualities of an object by touch and so to name it, is a disturbance of a high-grade mechanism involving sensory appreciation and discrimination, exactly comparable to what is required for recognising objects by sight, and its underlying anatomical area in cortical cases is without question localised in the postparietal region (superior parietal lobule), as has often been proved

by operation or at autopsy. There is no reason whatever why analogous defects on a similar plane, such as those of failure to recognise words, or the notes of music, should not be equally the outcome of a lesion localisable with the same relative minuteness.

The task of assigning disorder of function to site of anatomical defect is none the less peculiarly arduous. A tendency to correlate symptoms with naked-eye lesions and to ignore the necessity for the exact determination of their extent by the microscope is to be deprecated; further, the effect 'at a distance' in linked neuronic systems of a given anatomical lesion must not be overlooked.

When, as remarked above, it is stated that the overwhelming bulk of evidence points to a general speech area in the left hemisphere, lesions of which are revealed clinically by varieties of aphasia, I do not ignore the exceptional cases, of which not a few have been put on record. Several valid explanations have been advanced in such cases;

perhaps the most significant is the possibility of left-handedness occurring, if not in the individual concerned, then in the other members of his family. Thus Henschen[1] has collected 85 positive cases of motor aphasia (aphemia) associated with definite lesions of the so-called Broca's area at the posterior extremity of the third left frontal gyrus; the contradictory cases he cites number 17; of these some ten or eleven are not, according to his analysis, conclusively contradictory, but six are left which are. The proportion, 85 to 6, is not very different from that of right- to left-handed individuals in the community. One of my own cases is that of a man of 55, with severe left hemiplegia and pronounced motor aphasia, who it was ascertained had never exhibited any trace of left-handedness previously; yet his own sister, and his father's brother, were definitely left-handed. There is something to be said for the 'stock-brainedness' theory, elaborated more particularly by Foster

[1] HENSCHEN, *l.c.*

Kennedy,[1] according to which the absence of aphasia in large right-sided cortical lesions in definitely left-handed individuals (of which he gives three excellent illustrations) can be explained by their belonging to a 'left-brained' stock, notwithstanding their left-handedness; similarly the occurrence, as in the case just mentioned, of aphasia from a right-sided lesion in a right-handed individual may be due to his belonging to a 'right-brained' stock in spite of his right-handedness. In other words, ectopia of the speech areas is a possibility in some right-handed members of a sinistral stock, and in some left-handed members of a dextral stock. This is not mere speculation, but a hypothesis which accounts satisfactorily for not a few recorded instances of 'crossed aphasia,' and which receives support from the pathological side, as has been well shown by Mendel.[2]

[1] FOSTER KENNEDY, *American Journal of the Medical Sciences*, 1916, cliii, 849.

[2] KURT MENDEL, *Neurologisches Centralblatt*, 1912, xxxi, 156.

It is desirable to bear in mind, as has already been stressed, that the mental level of the subject is not to be ignored when the question of a speech area in the left brain arises. Qualifications must be made for exceptional cases. Thus in the matter of illiteracy it is evident that the usual view cannot apply in its entirety. Ernst Weber[1] cites the instance of two right-handed illiterate Russians, who, after a lesion in the right hemisphere (involving of course the left half of the body) entirely lost their ability to read, though they could still write. As their normal powers of writing and reading were limited, the presumption is strong that the speech area was bilateral but poorly developed, so that a lesion involving the appropriate right cortical area destroyed the power of reading, such as it was, the left area being too feebly developed to undertake the work. That their power of writing with the right hand was not affected is explained, naturally, by its unique representation

[1] ERNST WEBER, *Zentralblatt für Physiologie*, 1904, xviii, 341.

in the left hemisphere. And there are cases on record where aphasia has not occurred, although the situation and size of the lesion might have led one to suppose its appearance probable, presumably owing to the illiteracy of the subject and the lack of full development of the specialised centres.

The rôle possibly played in the function of speech by the right hemisphere, in right-handed persons of ordinary education and culture, and the question of compensation or substitution by speech centres of the right brain in cases of left hemisphere lesions, has long been a matter of controversy, but is perhaps better discussed in our next section, since the matter is primarily physiological.

Chapter II

Physiological Considerations.

To understand the physiological mechanisms employed in speaking we may clear the ground by a brief sketch of how speech is acquired. Long ere the child has begun to articulate, his ears have become accustomed to many and varied sounds, included in which are the sounds of words, mere noises to the child, without any meaning. Some of these sounds are repeated in his hearing much oftener than others, so that he gradually learns to distinguish them and to associate them with certain objects, animate or inanimate. We have no reason to doubt that, before articulate speech begins, the infant is using both hemispheres for vision and both for audition. It is important to note that capacity for retaining impressions of a visual or auditory kind exists long before appreciation of their significance can possibly arise. The

child hears hundreds of words before he can himself utter one, yet it is quite certain they are leaving their mark in what are to become his speech centres. The famous "Frost King" episode in the history of Helen Keller demonstrates how the brain can store up what it does not understand, preserve the stored-up material, and utilise it years later as though it were original or intuitional, instead of having been in reality unconsciously absorbed. A striking illustration of this point is furnished by Bastian[1]; the case was that of a little boy of nearly six, who had never uttered a single vocal sound, and whose 'dumbness' had led the anxious mother to consult two eminent physicians in regard to his mental state. One day, on the occasion of an accident happening to one of his favourite toys, he suddenly exclaimed, "What a pity!" these being the first articulate sounds he had ever made. No clearer proof is required of the retention of sounds in the process of learning

[1] H. C. BASTIAN, *Aphasia and other Speech Defects*, London, 1898.

speech, sounds which eventually become associated with particular mental impressions. Development must have been taking place along these lines without there having been any previous evidence of it by the usual trial-and-failure method of the infant.

The next step is the child's endeavour to imitate by articulation the sounds he hears constantly repeated by others, and in this way the movements necessary for the articulation of these sounds, or words, are learned. Again we must believe that both hemispheres are being utilised for speaking movements; before he learns to read or write, therefore, his 'inner language' consists of the memories of certain sounds closely linked to the memories of certain movements, and the former of these is also intimately bound to the visual ideas of certain objects. When his nurse says 'bow-wow' he will look for or point to his little toy dog, and when it is shown him he will say 'bow-wow.' Thus as early as he begins to think at all, he is developing his 'inner language.'

When the child reaches the stage of

learning to read and write, his 'inner language' becomes considerably more complex. He learns to write by letters and by syllables, which are the signs for sounds. In this way he acquires a visual picture for each sound, made up of a letter or combination of letters, and so he reaches the stage of a visual knowledge of letters, syllables and words.

He learns to write, however, with one hand, and that the right hand. Hitherto, as we have seen, the presumption has been that both hemispheres have been utilized for hearing sounds, for articulating sounds, and for reading signs that stand for sounds. When he writes signs that stand for sounds, we must suppose he uses largely, or entirely, a part of his left hemisphere only, as far as the physiological mechanism is concerned. Now if both hemispheres have thus shared in the acquisition of speech, how comes it that in the average individual it is only necessary to destroy a limited portion of the left cerebral cortex for a complete aphasia to result? The received explanation is the general right-handedness of *homo sapiens*,

but the inadequacy of this solution can be seen when it is realized that the difference between the daily work of the two arms is vastly less than the corresponding difference in function between the right and the left speech areas. It is fortunately not necessary to allude here to the various ingenious, not to say bizarre, speculations that have been advanced to account for right-handedness in the man of to-day as compared with the ambidexterity, or, indeed, left-handedness, postulated, on grounds more subtle than convincing, for our prehistoric ancestors; in historical times, certainly, right-handedness already predominates, as witness the following passage from the Book of Judges, chap. xx :—" And the children of Benjamin were numbered at that time out of the cities twenty and six thousand men that drew sword. . . . . Among all this people there were seven hundred men chosen left-handed; every one could sling stones at a hairbreadth, and not miss." Thus the percentage of left-handed fighting men was not more than about 2.7. Assuming to-day that, say, 95 per cent. of individuals are

in fact right-handed, we must ask the question whether this has any bearing on the location of speech centres in the left hemisphere. It will be agreed, I think, that the acquisition of writing is more laborious than that of learning to speak and read, and almost invariably postdates the latter ; what is still more significant, physiologically speaking, is that a unilateral mechanism is employed for the first time, as opposed to the bilateral mechanisms of articulation and of vision. The teaching of children to write with the right hand is universal, and any relative left-handedness or ambidexterity they may previously have exhibited tends to disappear under the influence of training, especially, for some reason, in the case of girls, but the contrary process—a right-handed child becoming left-handed—never occurs. It has been maintained by Weber[1] that the epoch-making discovery of Broca in 1862 became possible, although right-handedness had existed in man for hundreds of years, only because in the fulness of time

[1] ERNST WEBER, *l.c.*

the dissemination of the accomplishment of writing among all classes determined the lead of the left hemisphere over the right, and at the same time finally established the localisation of the speech centres in the left cerebral cortex. The argument is certainly overstated; in Abercrombie's *Pathological and Practical Researches on Diseases of the Brain and Spinal Cord*, 3rd edition, 1836, I find 20 cases of the association of right hemiplegia and aphasia, and 4 cases of left hemiplegia and aphasia. But a physiological concentration of this kind must play a rôle in leading to preponderance of the left hemisphere in speech mechanisms. By analogy, one would suppose the musical functions to be more bilaterally and equally represented, and certain researches (Probst, Mingazzini) go to substantiate the contention.

We believe, then, that there exists in all normal individuals who have had a regular education a potentiality of development of speech centres in both halves of the brain, and that as the growing child becomes definitely

right-handed, and is trained to write, this potentiality weakens, though whether it ever entirely disappears is questionable. Vicarious action of the right hemisphere must in a general way be admitted as a feasible explanation of some otherwise obscure phenomena of aphasic cases. Henschen, for example, has collected 28 cases of *bilateral* destruction of the foot of the third frontal gyrus, and in every one of these mutism (aphemia) was complete and permanent. On the other hand, Charcot and others since his day have noted the transience of the aphemia in some third left frontal cases, a condition more readily explained by substitutive function of a twin centre in the right hemisphere than by any taking over on the part of neighbouring fields on the left side, or by functional recovery on the part of Broca's area itself, the proof of this being restitution of speaking although the Broca lesion has persisted and been found at autopsy. In other instances, however, the right hemisphere has been unable to compensate.

Our hope of improvement in certain

cases of aphasia depends to some extent on the belief that the right cerebral speech centres can be induced thus to take up functions hitherto incompletely exercised, if not entirely neglected ; at the same time it should be clearly understood that the degree of possible utilisation of these centres cannot be accurately foretold. Some individuals are more right-handed than others ; to some people the left limbs remain peculiarly *gauche*, so that on the personal factor much must depend. Speaking generally, however, no case of aphasia should be abandoned as therapeutically hopeless without a serious attempt to awaken dormant activities in the opposite hemisphere. If these facts were better known the cultivation of ambidexterity would receive that impetus which it assuredly deserves. The suggestion has been actually made[1] that in cases where hemiplegia is a possible future event the patient should be encouraged to develop a graphic centre in the right side of the brain by practising writing with the left hand.

[1] COLEY, *The Practitioner*, August, 1909.

Still speaking physiologically, we are to bear in mind that a spoken word and a written word are movements, while on the sensory side a seen word is no more than a seen object, and a heard word than a heard sound. Before the child can write a single letter correctly he will scribble with his pencil and say it is 'writing,' and if to us it is an unintelligible scrawl it is identical physiologically with an intelligible scrawl. The infant's laryngeal noises may be unintelligible but they do not differ integrally from the laryngeal noises that we call words. All must, accordingly come under ordinary physiological rules for motion and sensation; and disorders affecting both projection-systems, afferent or efferent, and physiological cortical centres for motion and sensation, may impair the reception or the execution of speech elements exactly as of any other function elements.

The projection-systems of the physiologist are those which pass out of and down from the cortex to motor centres of the lowest level, in pons,

medulla, and spinal cord (for muscles of face, throat, limbs), and those which proceed from peripheral sense organs to a series of cortical centres (for vision, hearing, touch, etc.). As far as the function of speech is concerned, the motor cortical centres for articulation, phonation, and movements of the hand and fingers in writing are localised and delimited ; so are the reception centres for auditory and visual impressions belonging to the speech category. Both sets alike are liable to the same disturbances of function as those of any other, non-speech, projection-systems. When the function of the motor efferent paths to tongue, lips, palate, vocal cords, etc., is impaired the condition is called dysarthria by the clinician ; if the interruption of function is absolute, a state of anarthria exists. It is a simple matter to make a diagnosis of dysarthria or anarthria, for the muscles concerned must in the case given be paralysed for all movements, *e.g.*, for those of swallowing, whistling, etc., as well as for the movements of speech. Analogously, if a hemiplegic patient cannot write

because of a projection-system paralysis, no more can he use the same muscles for playing the piano, shaving, or any other movement. It is matter for regret that there is no word except agraphia to express this inability to write from lesions of the projection-system, for the term, in the parlance of aphasia, applies to impairment of writing from involvement of systems that do not belong to the projection group.

As every one knows, there are large areas of the cortex to and from which very few or no projection fibres pass; they are called association areas, for they are linked to each other by important sets of fibres which never leave the cerebrum. *Inter alia*, these association fibre-systems join projection-centres to each other, and their course, therefore, is transcortical. Applying this generality to the particular mechanism of speech, we say that a complicated series of association fibres impinges on motor and sensory projection-centres concerned with the reception or execution of speech components; and the point of significance in the

present connexion is, that disorder of these transcortical association-systems is the physiological disturbance underlying the clinical phenomena of aphasia as ordinarily understood. Thus, in a general but none the less definite sense, all aphasia, physiologically speaking, is transcortical. When an aphasic patient hears and sees, but does not understand the word-sounds he hears or the word-signs he sees, his is a transcortical physiological defect ; when he understands both of these, but cannot speak correctly, again there is a transcortical impairment somewhere between projection-centres of the sensory and motor order respectively.

Stress, however, is to be laid on the fact that, anatomically, association paths between cortical projection-fields must start from, and run into, these fields ; hence ordinary lesions of the latter must inevitably involve origins or terminations of such inter-central paths. For this and other reasons complete 'isolation' by disease of the physiological systems concerned is scarcely to be expected ; that is to say, the most typical aphasic syndromes

must be the result of lesions implicating association fibre-systems away from the points where these anastomose with projection-centre nerve-cells, otherwise we should expect to have symptoms referable to involvement of projection mechanisms as well, and these would complicate the true aphasic picture.

It is important to bear in mind, further, that the same motor cells of the cortex, say those innervating mouth, lips, tongue, palate, are used at one time in the function of speech, at others in totally different physiological combinations, and, since the function of motor speech may be grossly disturbed while the cells can be utilised for other functions, we cannot suppose the physiological defect to be localised in the cell-groups concerned. It must be outside them. Theoretically, therefore, apart from clinical and pathological considerations, disorders of speech must be disorders, physiologically, of transcortical mechanisms (Hughlings Jackson's 'highest level') which play on those of the cortical projection class (Jackson's 'middle level.')

Though it be a platitude that speech

itself is something beyond mere function of physiological systems, the reader has been given this sketch of the elements because of the importance of recognizing that on the physiological side the mechanisms relating to speech are in every way similar to those connected with the function of the non-speech parts of the brain.

A great deal of discussion has raged round the question of how speech on its receptive and expressive sides is facilitated by previous experience. All stimuli leave behind them traces or remainders, existing as an unconscious possession of the cortex and enabling it to reawaken memories in consciousness of former sensations or movements, and to perform repeated movements more easily and completely. In the clear diction of Sir William Hamilton: "Memory strictly so denominated is the power of retaining knowledge in the mind, but out of consciousness. . . . To bring the *retentum* out of memory into consciousness is the function of a totally different faculty, recollection. . . . It is not enough that we possess the

faculty of acquiring knowledge and of retaining it in the mind, but out of consciousness; we must further be endowed with a power of recalling it out of unconsciousness into consciousness—in short, a reproductive power (recollection)." Bastian[1], who cites this statement from Hamilton, supposes that there is on fitting occasions revival of something like the original molecular activity in the nervous elements concerned with the primary perceptional or intellectual process of this or that kind. Evidently, then, loss of memory and loss of recollection are far from synonymous; in cases of aphasia we constantly meet with illustrations of the fact that the patient may be unable to recollect words spontaneously when he has not lost his memories of them, as is shown when he repeats them on hearing or seeing them. Now at this point the problem is, where in the cortex are the physiological mechanisms for recollection of speech elements (memories of sounds, sights, and movements)

[1] H. C. Bastian, *l.c.*

situated, or, rather, is there any area or areas apart from reception-fields in which we may postulate such speech memories to be stored? When the sight of an orange recalls gustatory, olfactory, tactile, and possibly still other memories of the fruit, are these facilitated by their having been a *retentum* (unconscious and probably purely physiological) in corresponding sensory fields? We believe it is so. The association between memories of the same order is effected in the cortical region concerned; that between memories of different orders is effected by long tracts of fibres passing from one region of the cortex to another, as when an odour or smell recalls a vivid visual memory. It is really immaterial for our present purpose *how* and *in what form* memory elements lie dormant, and it is a matter of indifference how they are to be designated, as 'memories,' 'images,' 'engrams,' and so forth. But it is of some importance from the point of view of aphasic syndromes and their physiological causation to know if the neural arrangements underlying retention of

previous sensory experiences are situated in the projection-fields of the corresponding sense avenues, or in their immediate neighbourhood, or independently of them. Without delaying unduly on this question, we say that the conclusion from much available pathological evidence is, that in the immediate vicinity of the respective sensory projection-fields are cortical areas whose neurones serve in the retention of the traces or remainders of which we have spoken above. In respect of movements, further, such as those of articulation and phonation, the statement is that they are similarly facilitated by recalling memories of previous movements, and these in all probability are of a kinaesthetic character, that is, they are memory traces of the muscular sensations accompanying movement. A pianist who plays a composition 'by heart' remembers the movements required to bring his fingers to the appropriate notes in sequence, and is utilising kinaesthetic 'images'; indeed, an expert can so play with his eyes shut, and some fine pianists have been blind. For preservation of such

kinaesthetic memories, neurones in the vicinity of the corresponding motor projection-centres of Hughlings Jackson's middle level are responsible.

Consideration of the data supplied by clinico-physiological research will scarcely allow any other conclusion than that the physiological basis of word-storage for the purposes of recall cannot be a property resident in projection-centres themselves. On the 'final common path,' in Sherringtonian terminology, of the motor projection-system from the rolandic cortex are converging a number of 'private' paths from different physiological centres, viz., those for the use of the motor cells in articulating and phonating, in swallowing, eating, chewing, in whistling, singing, and so on. Nothing is more certain clinically than that one function may be paralysed and the others not. The lesion singling out one function must of necessity implicate a 'private,' not the 'common,' path.

A further physiological question of importance concerns the way in which words are recalled to mind during ordinary intellectual activity. Accord-

ing to one view, words are revived purely as *motor* processes, that is, as faint excitations of the processes occurring in motor centres during the articulation of words. The idea is that in 'thinking' of words faint innervation of the motor units whose activity would produce the word audibly is taking place. This view is declared by Bastian, erroneously, as I think, to have been frequently promulgated by Hughlings Jackson, for the latter specifically states:[1] "To remember a word is to have a faint excitation of the *sensori-motor* process of that word" (italics mine). Again, the same author says:[2] "It is not meant that a word, which is a psychical thing, is an activity of any nervous arrangements for highly special and complex articulatory movements, but that such nervous arrangements (or, rather, *audito-articulatory* nervous arrangements) are the physical bases

[1] HUGHLINGS JACKSON, *West Riding Lunatic Asylum Medical Reports*, 1873, iii, 175.

[2] HUGHLINGS JACKSON, *Croonian Lectures*, *British Medical Journal*, April 5, 1884.

or anatomical substrata of words" (italics mine). These quotations serve to show that Jackson held that a sensory element is involved in the recall of words, and in the majority of persons this probably originates in the auditory centres, a view for which there is much to be said. Doubtless there are differences in different individuals; some apparently see rather than hear words in thought, and it is known, as Galton's researches have well demonstrated, that in respect of arithmetical figures some persons see them in their mind's eye in a regular and unvarying order and position. Distinctions between 'visuals' and 'auditives' obtain here. In any case the thought of a word naturally runs on to an incipient articulation; some people's lips move as they read silently. At a music-hall it is not uncommon to hear a faint audible whisper from some of the audience as they note the change of figure on the indicator of the programme items, and automatically articulate the number which they see, while at some cinema theatres a notice is actually thrown on the

screen requesting the audience not to read aloud the wording of the sub-titles between the picture sections of the film !

Physiologically considered, the action of the ' inner speech ' mechanism must be somewhat as follows :—

In speaking, the processes underlying words are aroused in the auditory area, whence stimuli reach the kinaesthetic area for speech situated in the vicinity of the cortical projection-centre for tongue, lips, vocal cords, etc.

In writing, the memories of the words (that is, whatever underlies physiologically the memories of words) are aroused, one supposes practically simultaneously, in auditory and in visual centres, whence the kinaesthetic region for writing movements, close to the cortical (rolandic) arm area, is awakened, and so the fingers are innervated.

On the receptive, as opposed to the executive, side, the stimulus for the understanding of spoken language proceeds from the ear to the auditory projection-centre and thence to that

allied auditory region which has to do with the processes underlying auditory memories. For the understanding of written language an analogous route to the visual memory region is traversed.

In speaking thus of physiological routes I do not wish the reader to have the impression that these are isolated in the sense that the sight of a word arouses only visual connexions, and so on. Such is the rapidity and facility with which processes at the basis of speech are carried on, and so wide are the interrelated physiological systems, that in simple acts of perception, at the sound or the sight of a word, cerebral activity probably ensues in varied cortical areas and in both hemispheres of the brain.

Finally, we know, of course, that the physiological centres of speech can be stimulated into activity either from without or from within ; either because we are reading or hearing words, or because we begin intentionally to speak or to write. There is no good reason to believe the processes are in the two

cases essentially different; in our ignorance of the physiological nature of volition we can only imagine that a stimulus capable of exciting the neurones of the auditory centre is initiated from elsewhere in the cortex, *i.e.*, transcortically, in the latter case, and by the afferent projection route in the former.

Chapter III

Psychological Considerations.

The subject increases enormously in complexity and intricacy when we turn to read, heard, written or spoken words, not as sounds, movements, etc., but as symbols or expressions of ideas. Speech is symbolic thinking, as Head has again recently emphasized; to adopt an illustration of Lloyd Morgan's,[1] used by him in another connection, the anatomo-physiological basis of speech is as different from symbolic thinking as is the movement of the needle on the gramophone record from the tune it can be made to play.

Letters and words are the signs for sounds, as already mentioned, but words are, in addition, symbols of another order altogether; they have a 'con-

[1] Lloyd Morgan, *Journal of Neurology and Psychopathology*, 1920-1, i, 211.

tent,' a 'meaning'; as Bastian said they are the counters by means of which part of our thought is carried on. It has always, therefore, appeared to me otiose to attempt to distinguish speech disorders from intellectual disorders; the controversy, in any case, is bound to prove somewhat barren.

To specify where speech defect ends and general intellectual defect begins is obviously impracticable; in view of what has just been said, every impairment of speech, psychologically considered, is *pro tanto* an impairment of a psychical function. On the other hand, I am in accord with those who hold that a speech imperfection may co-exist with perfect preservation of the general intellectual level; and in a previous communication I have criticised the view, advanced notably by Moutier,[1] that in every case of aphasia (that is, in Moutier's sense, of Wernicke's or so-called sensory aphasia) *both* a general intellectual deficit *and* a special intellectual deficit of language are to be found. Still

[1] FRANÇOIS MOUTIER, *l.c.*

more open to criticism is his corollary, that the intensity in degree of the psychical disorders produced by the lesions in Wernicke's zone is "very notably proportional to the extent of the lesions in the zone." I have under my care at the present time two cases of motor aphasia, that is, of almost complete mutism, following organic lesions of the left hemisphere, and in each case unaccompanied by defect of writing; one of the patients, a lady, is a polyglot, who can read (not, of course, aloud) and write in four languages (English, French, Flemish, German) in spite of her lesion; and in each instance, as well as in some mild 'sensory' cases I have seen, investigation has convinced me of the complete general integrity of the intellect. Osnato[2] has stated that intelligence in its widest sense, speech, and eupraxia, are one and the same process, and that they develop together and disintegrate together; but this is a generalisation of so broad a character

[2] MICHAEL OSNATO, *Aphasia and Associated Speech Problems*, New York, 1920.

that it becomes shallow at the same time, though it contains the truism that speech is a mental process. Psychologically, speech disorders are disorders of the Gesamtpsyche; local lesions of structure, limited disorders of physiological mechanism, and partial disturbances of psychical process, cannot conceivably be superimposed one on the other within identical boundaries or be made to correspond precisely; the units of the standards whereby they are measured are not of the same order.

Intellectual and Emotional Speech

To the acute intellect of Hughlings Jackson we owe appreciation of the difference between the 'interjectional' and the 'propositionising' use of words as symbols of thought. The former is an element in emotional and the latter in intellectual speech and one may be grossly affected without proportionate impairment of the other. In ordinary aphasia there is commonly relative conservation of emotional speech; a patient with a limited vocabulary, the

result of disease, will nevertheless frequently contrive to convey the most varied meaning to his words by alteration of inflection, tone, accent, stress, and emphasis, and this can be done even when he has but one word to say, but one string to play on. Often, too, abundance of gesture makes up what is wanting in intellectual speech. The aphasic may say 'Yes' when he means 'No' and may have to depend on emotional speech, or on one or other of the above makeshifts, to express his real meaning. Or, again, he can often ejaculate words emotionally which he cannot use at all in the form of a proposition—*i.e.*, in correct, logical speech. One of my patients who suffered from motor aphasia in the ordinary sense saw a Zeppelin descend in flames one night on the outskirts of London and ejaculated 'Hallelujah!' under the stress of his emotion, which word he has never since been able to repeat 'propositionally.'

A case is recorded by Gowers[1] in which a patient with aphasia was unable

[1] W. R. GOWERS, *Diseases of the Nervous System*, London, 1888, vol. ii, p. 109.

to say 'no' to order, and plaintively replied, after many repeated requests and under the stress of emotion, "I can't say 'no.'" Years ago I showed[2] at the old Neurological Society of Great Britain a patient (a military officer's servant) who, as the result of a fall from a cart, sustained an injury to the left parietal region, with serious aphasic disturbance as a sequel. He presented the syndrome of a variety of so-called sensory aphasia, his utterance being one long torrent of speech fragments, an unintelligible jargon. Nevertheless his gesture language, so to say, was intact. Handed a pencil in the form of a little champagne bottle, he immediately went through all the performance of drawing the cork, pouring out the wine into a glass, holding it up and drinking to the health of the King, with military salute, in an amazingly graphic fashion.

An aphasic patient may swear with fluency when no amount of urging will get the same words out in the form of a 'proposition.' In one case of

[2] *Brain*, 1906, xxix, 416.

paraphasia from a temporal lesion the patient was so irritated by his frequent mistakes—at breakfast he said "pass the ink" instead of "pass the salt"—that an only too correct "damn!" escaped his lips, whereupon his wife rated him soundly for swearing before the children.

Loss of emotional with conservation of intellectual speech has not been the subject of any serious investigation, as far as I am aware, though it deserves study. Long ago Brissaud spoke of patients who had lost "le chanson du langage," and who were incapable of adding emotional meaning to their words by the light and shade of accent and emphasis. Their speech was one long monotone. In a paper on apraxia I referred to the case of a right-handed patient with severe left hemiplegia following embolism of the right sylvian artery. This lady exhibited a curious general immobility and absence of spontaneity, even as regards movements of the right (unparalysed) limbs. Her face was more or less fixed in an expression suggesting anxiety; her eye-

brows were symmetrically elevated and her eyes looked straight in front of her. This facial expression never appeared to vary. She was quite incapable of making any grimace, of smiling, of laughing ; when asked merely to mimic such appearances, her reply always was, "I can't." On examination, however, apart from slight left facial weakness, no paresis or paralysis of the musculature or emotional expression was present. Now the interesting feature of the case from the present viewpoint is, that her voice had become, since the stroke, peculiarly monotonous and just a little shrill ; and she was completely incapable of modulating it, or of imparting any inflection, accent, or emphasis to her words, which, for that matter, were in the intellectual sense correct and appropriate. The late Sir Felix Semon examined her larynx and found its innervations normal in all respects. Here, then, is a seemingly unimpeachable case of loss of emotional speech with preservation of intellectual, and it is not without interest that the lesion was in the right hemisphere.

The explanation of the use of words by mute aphasics under the influence of emotion, as in circumstances illustrations of the effect of which are given above, is possibly that the functional effectiveness of the centres concerned is so lowered by disease that only powerful stimuli will arouse impaired mechanisms and temporarily force them into action. When voluntary recall of words is for the aphasic impossible, the summation of stimuli from without may be sufficient to arouse dormant activity; hence we shall be careful to avoid the assumption that speech mechanisms and the memory traces of speech processes are permanently destroyed because volition by itself cannot effect their excitation.

At the same time we must not fail to observe how in ejaculatory and interjectional speech the content of the words emitted has often degenerated from its former intellectual significance and become almost meaningless. Such words scarcely come any longer within the category of speech in the intellectual sense, and are largely outside its rules.

Speech Automatisms

Pursuing this question of the awakening of speech under the influence of emotion as a proof (in some instances) not of destruction but of functional isolation of mechanisms, or of their impaired vitality, we should bear in mind the possibility of speech acquisitions having become so automatic as to be but faintly voluntary; their reproduction being thoroughly organised and systematised, they become as it were independent elements in the domain of speech and are not readily put out of action, or, being out of action, they can be recalled, though 'propositionising' speech is materially impaired. Very many cases of aphasia exhibit this peculiarity. Henschen has recently collected evidence which suggests that such acquired automatisms are located physiologically in the right hemisphere, and it is of interest to remember that Hughlings Jackson always held that the left hemisphere was 'creative' and the right 'automatic.' Thus a patient was unable to 'say' any of the numerals to order,

or to use any of them correctly in a sentence or 'proposition,' but when shown the figure 1 in print he suddenly began to count and counted rapidly to over 20, until he had to be told to stop. Some aphasics can sing what they cannot 'say'; the patient already alluded to above as exhibiting jargon-aphasia in a severe form was altogether incapable of 'propositionising' speech, but when asked to sing anything suddenly began to sing the following:

"I am longing for my dear old home again,
That cottage in the little winding lane;
I can see the roses climbing,
I can hear the sweet bells chiming,
Oh, I'm longing for my dear old home again."

Both words and air were perfectly accomplished. I might mention, too, that he was able to whistle tunes with accuracy. In another instance, the Lord's Prayer was repeated, although volitional speech to order was almost entirely lost. I have also seen one curious instance of difficulty in speaking

spontaneously or on request being associated with the power of reading aloud, provided the words were simultaneously read aloud by the examiner. This peculiarity is well known to characterise many cases of the stammering neurosis, but it must be extremely rare in aphasia.

Thus again clinical data lead us to the conclusion that the problem in many cases of aphasia is not one of destruction of ' engrams ' or ' images,' but of inability to arouse them. The neurologist knows well that in this respect the phenomena of aphasia fall in line with those of motor apraxia. To see an apraxic making futile efforts to protrude the tongue on request, when the next moment he puts it out to lick a crumb off his lips ; to watch his failure to bring the fingers of the apraxic limb to the nose when a moment later he automatically scratches his cheek with them—this is by analogy to gain an insight into many of the supposedly incongruous symptoms of aphasia and to realise their essentially transcortical character. Since speech acquisitions that

have become automatisms can still be 'tapped,' as it were, while voluntary employment of the same words in deliberate speech is impossible, we must be dealing with physiological constellations of living neurones, whose function, being automatised, is perhaps less vulnerable, or whose cortical *local* is not identical with that of volitional processes. Whatever the explanation, the fact remains; they escape because they are in an annex to the main hall of speech.

AGRAMMATISM

Not enough attention has been paid, perhaps, to the grammatical and syntactical aspect of the subject, which is of fundamental importance. As words are made up of letters and syllables, so sentences are made up of words, and they are intellectually meaningless unless they are properly constructed. The path from thought to intelligent expression lies through the sentence; hence in aphasia difficulties with the sentence are prone to occur, and the aphasic often reverts by a process of involution to the standard of the child

learner, who has trouble with those parts of speech that are concerned with qualifying and correlating. In the words of Mills,[1] the grammar of language no longer exists for the aphasic. Agrammatism as defined by Arnold Pick[2] is " that form of pathologically altered speech in which the essential elements for the grammatical and syntactical structure of speech are either lost or imperfectly employed."

From a series of letters, written by a business man who suffered from a severe degree of aphasia, and who improved materially under treatment, a selection is given below, in chronological order, the condition of agrammatism being very well shown.

(1) July, 1917.

" Dear Sir,

Yours of date. Sorry war news, tearful things.

Yours truly,

A—— S——."

[1] C. K. MILLS, *Journal of the American Medical Association*, Dec. 24, 1904, p. 1940.

[2] ARNOLD PICK, *Die agrammatischen Sprachstörungen*, Berlin, 1913.

(2) March, 1918.

" Dear Sir,

Speech, difficult of speaking. Arm, slow improvement, no crip. Dragging of leg, no irovement. Loss memory, confusion, change, etc. Rheumatics, pain and lumb.

Yours truly,

A—— S——."

(3) October, 1918.

" Dear Sir,

We have a difficult slow job before us; however, time on her side. Apparently still drop the leg. Brain fag is recovering, although slowly. Rheumatics; sorry to say that has seized me. Can you nothing for me?

Yours truly,

A—— S——."

The improvement from the marked agrammatism of the first two letters to the freer composition of sentences in the third is noteworthy, and the reader will also note that though

the elements of syntactical construction are wanting, for instance, in No. 2, the sense is clear enough. Illustrative of the same basic impairment is the tendency of many subjects of aphasia to speak or write in the 'telegram style,' omitting parts of speech which may not be strictly necessary for comprehension but are, nevertheless, essential for correct speaking. Prepositions and articles, more particularly, are omitted, and nothing shows the agrammatism of the patient's language so much as their omission.

Allied to this defect is the failure of many aphasics to end sentences which they begin properly enough ; they put two or three words together quite correctly and grammatically, but that appears to be the limit of what they can do. A patient with pronounced aphasia of a mixed type carried on the following 'conversation' with me :—

"How are you to-day ? "

"Well, a matter of . . . I can't . . . I never . . . I put the . . . There's the . . ."

"How's your leg ? "

"I can't . . . It's a little . . . It's really . . ."

"Where's your boy to-day?"

"Down in . . . He's right down in . . . He's . . . The thing is to . . ."

As the child at school is taught by letters and by syllables to acquire his knowledge of the morphology of words, so in a reversed way the disintegration in the morphology of speech caused by a cerebral lesion may result in the aphasic's having great difficulty with letters and syllables, not as regards their enunciation, but as regards their correct employment. He may persistently use wrong letters or syllables and often a trial-and-error process is necessary to get the correct result. Thus a patient said 'shapple,' 'shaplin,' 'shapnel' in succession before he got 'shrapnel'; another, shown a tuning-fork and asked for its name, said 'tuning-thorn,' 'tuning-thork' before he gave the correct answer. This same patient, given a tape-measure and asked to show how to use it, replied, "that's a lamp, a wate, a tate (fancy that!), it's a tape."

These defects are to be distinguished

from what has been improperly called 'pseudo-agrammatism,' viz., difficulty in recalling the names of objects, a condition referred to below. Undoubtedly disorders of the nature of agrammatism run like a thread through some of the clinical types, and yet we must take care not to extend its range unduly. Pick[1] considers that higher grades of paraphasia and of jargon-aphasia, for example, ought not to be classed with agrammatism, since in them the impairment lies at another level; he furnishes evidence, nevertheless, that agrammatism and paraphasia are apt to occur in combination, as, in fact, is illustrated in minor degree above. The following excerpt from the examination of a case of left temporal tumour, conducted on the day after operation, exhibits clearly the association of agrammatism and paraphasia. The patient was a young woman of 24.

Shown a paper knife.—"That's a knife.. a knife.. what you cut the sign . . . the iron . . . it's for the iron . . . walker . . ."

[1] ARNOLD PICK, *l.c.*

A safety pin.—"That's a piece of water . . . water . . . what you drink with water . . . what you went the nen with . . . what you send your dress to do late on . . ."

A piece of soap.—"Piece of inch tape . . . inch . . . inchness . . . what you wet yourself . . . inch . . . what you wash yourself . . . iron . . ."

A pencil.—"That's a bensha . . . what you press . . . what you say you fire in the nent . . . scratch in the lead work . . . what you wish is your neck . . . nick a nick . . ."

Here are well exemplified both the constructional defects of the sentences, their ungrammatical character and very frequent incompleteness, and the wrong use of words characteristic of paraphasia. According to my own experience, agrammatism is most prone to occur when the lesions producing the aphasic disorder are situated in the left temporal lobe, in this respect corroborating the views of Pick, who says the physiological mechanisms

underlying the grammatical structure of speech are localised mainly in that lobe. Henschen's studies lead to a similar conclusion. But the subject needs further investigation. Maas[1] has recorded two excellent examples of the symptom, both occurring in temporal tumours; he argues, however, that agrammatism is the result of the imperfectly equipped right hemisphere essaying the function of speech when the speech areas of the left are destroyed by large lesions. On the other hand, E. Forster[2] records a case of the same condition following a gunshot wound of the posterior part of the left frontal lobe, but on the clinical side it is open to criticism.

[1] O. MAAS, *Neurologisches Centralblatt*, 1920, xxxix, 465.

[2] E. FORSTER, *Monatsschrift für Psychiatrie und Neurologie*, 1919, xlvi, 1.

Chapter IV

Classification of Aphasic Disorders

Much of the dissatisfaction expressed from time to time in reference to the classification of aphasic disorders is due to confusion of thought as to the basis of that classification. From what has already been said, it will be clear that aphasia can be classified in an anatomical, physiological, or psychological scheme, but we must avoid, as has also been remarked, any transference of the terms used in the description of defects of one of these systems to the defects of either of the others.

(1) Aphasia being, in essence, a symptom or symptom-complex, we might ascertain the general site of the lesions calculated to produce it, and endeavour to correlate clinical varieties with variation in the position of the latter. We might, conceivably, speak of frontal,

parietal, temporal aphasia, and so forth, and if corresponding clinical distinctions could be correlated therewith, such a scheme would be undeniably useful, as it certainly would be legitimate. There exists no possible room for doubt that when the lesion is temporal, for instance, the type of aphasic syndrome is commonly very different from that associated with frontal lesions, and each of these from what is found if the lesion is in the distribution of the posterior cerebral artery ; frontal aphasia, so to call it, is definitely distinguishable from parietal aphasia, and so on. In view of the ascertained facts of cerebral anatomy and physiology, this is no more than is justifiably to be expected. 'Island aphasia' (*Inselaphasie*) is a term which has never come into vogue in England, but it represents a form of aphasic defect that appears to be the expression of local lesions situated in the island of Reil and involving speech-fibres uniting the temporal and the frontal lobes, and in this sense is an anatomically descriptive term for an aphasic syndrome.

The question, as can be readily under-

stood, is whether, if such lesions correspond to clinical types capable of differentiation on the one hand, and of occasional occurrence in a pure state on the other, an anatomical classification is desirable. This is probably not the case, not because firm localisation is impossible, but because the pure cases are always in a minority compared with the mixed types, and because the cerebral lesions of aphasia are frequently rather extensive. Nevertheless, given sufficiently small and localised lesions, it need occasion no surprise to find in association therewith strictly circumscribed aphasic disturbances. As a single instance, I shall refer to a particular variety of aphasia seen in temporal cases. Years ago attention was directed by Mills[1] to the occurrence of difficulty in naming objects and in recalling substantives, as the main if not almost the sole impairment in some temporal cases, and the definiteness of the association led him to suggest the localisation of a 'naming

[1] C. K. Mills, *Journal of Nervous and Mental Disease*, 1895, xxii, 1.

centre' in that part of the brain. More recently, Head[1] has coined the term 'nominal aphasia' for a type which includes this particular defect. Now while general amnesia, inclusive of words, may of course be encountered in senile and fatigue states, in nervousness, in intoxication, and so on, amnesia verbalis is a specific amnesia, and clinical evidence definitely points to its frequent development in cases of temporal disease; further, amnesia for substantives is even more special, and Henschen's analyses lead to the conclusion that these amnestic aphasias are prone to make their appearance when the site of the pathological process is in the second and third temporal gyri. Otitic abscesses extending through the tegmen tympani commonly invade the lower part of the temporal lobe, and it is precisely in such abscess cases that the variety we are considering is liable to occur, and as one of the earliest symptoms. If the reader will look back to the temporal tumour case cited above, he will not fail to

[1] HENRY HEAD, *l.c.*

note how the patient constantly has recourse to periphrases to cover word forgetfulness as regards substantives, and how many of her phrases begin with "what you do" something or other with.

The evidence for the specific association of amnesia verbalis with lesions of the left temporal lobe in its lower part is too impressive to be ignored, and it furnishes no support to the views of Head, alluded to at the outset, according to which amnesia verbalis (*inter alia*) is a hypothetical condition which cannot be associated with limited destruction of any part of the brain.

Some day, perhaps, knowledge of clinico-anatomical cerebral localisation will be sufficiently advanced to justify its use as a basis for aphasic classification, but at present the data are still incomplete.

(2) Taking, next, a physiological classification, we all are familiar with the time-honoured types of motor, sensory, and mixed aphasia, according as the main features of the disorder concern motor or sensory mechanisms respectively, or both. This nomen-

clature is in the first place thoroughly legitimate, and in the second clinically useful. When a patient cannot express himself in speech, though by other methods he indicates that he understands, it is natural to describe his defect as a motor defect; it is a form of paralysis. Unfortunately, however, we have no specific term to indicate that the paralysis is not of projection but of association-systems, and since he can utilise the same muscles for other physiological functions we are again led to the idea of the apraxic nature of this motor disorder, a condition for which the word 'paralysis' is inapposite. Further, since motor aphasia is a transcortical disturbance, physiologically considered, it is obviously illogical to use the expression 'transcortical motor aphasia' for another clinical group, with special features of its own. Yet this expression is in current use.

Again, when a patient does not understand what he sees written or what he hears spoken, it is surely evident, physiologically speaking, that there is an afferent or sensory impair-

ment of function, and though it is not on the primary perception level it is afferent none the less. Thus the term 'sensory aphasia' is perfectly comprehensible. But when a clinical syndrome, with distinctive features, is called 'transcortical sensory aphasia' as though to differentiate it from the other, confusion at once arises, since ordinary sensory aphasia is physiologically a disorder of transcortical mechanisms.

When the clinical picture is one of partial deafness or blindness for words, coupled with partial difficulty on the motor or expressional side, we speak of a mixed aphasia.

In spite of its serviceableness a purely physiological classification is at present unsatisfactory. Apart from the fact of its ignoring what must always be the fundamentally significant side of speech, viz., its psychical aspect, such a division has the disadvantage of introducing an artificial simplicity into a particularly complex subject. Besides, physiological terminology has not reached a sufficiently advanced stage for the purposes of our subject-matter.

A patient may be unable to distinguish spoken words as words, *i.e.*, as word-sounds, or he may know the sounds to be words, but may not know their meaning. In the first instance he is word-sound-deaf, in the second he is word-meaning-deaf. The latter condition belongs to a higher physiological level than the other, though both are 'sensory,' in the physiological nomenclature of aphasia. Clinical types can be justifiably differentiated in some instances, for which the physiological counterpart has not been reduced to a systematisation.

(3) A third classification takes into consideration solely the psychological side and analyses speech disorders as disorders of the Gesamtpsyche, anatomo-physiological correlates not entering into the scheme. The most recent and noteworthy endeavour to classify aphasia psychologically is that made by Dr. Henry Head, for the details of which the student may be referred to his recent papers on the subject. It may be questioned whether in the present state of knowledge a pure psychological grouping must not remain more of scientific than of

clinical interest, but for that matter no one of the three schemes is by itself satisfactory. There are certain important psychological characteristics of aphasic disorder in a general sense, largely common to all varieties ; on the other hand, there is a definite association between certain clinical varieties and the interruption of certain mechanisms of a physiological order. It appears to me that until further advance is made a psychological arrangement has the serious disadvantage of losing touch with cerebral function, and this is not compensated for by the greater scientific legitimacy which is claimed for it. In no subject of neurological study is the personal point of view of the investigator more often exhibited than in his approach to the question of aphasia. To read the communications of some psychologists, one would suppose they had never heard of the science of neuropathology ; on the other hand, when examining the localising deductions of some neuropathologists, one fears the idea that speech is an intellectual function is foreign to their conceptions.

The conclusion is, that a scientific classification of aphasia, as of any cerebral symptom-complex, ought to be pathological, that is, physio-pathological; this desirable end not having as yet been attained, the clinician must perforce be content with empirical clinical divisions, the usefulness of which, I repeat, outweighs their patent disadvantages. A purely psychological scheme ignores the anatomo-pathological side of the subject altogether, whereas the ideal to be advocated is the correlation of the anatomical, the physiological, and the psychological. However this may be, we shall make no advance in the study of aphasia either as a cerebral symptom or a psychical disturbance if we do not take our stand on the clinical certainty that varieties of speech impairment occur, are recognisable, can be differentiated, and are of practical and localising significance. For the clinician the problems of aphasia have to be faced in exactly the same way as those of disorders of function of the non-speech parts of the brain.

Chapter V.

Some Clinical Types of Aphasia

(1) Expressive Aphasia.

For the familiar ' motor aphasia ' of the text-books I prefer to substitute the term ' expressive aphasia.' The patient cannot express himself in words, and this without paralysis of articulatory mechanisms, for what words he has he articulates correctly enough. He will show by his actions and his response to stimuli that his intelligence is largely unimpaired and that his sole handicap is inability to express what is obviously in his mind, except by such makeshifts as have already been mentioned. It is no uncommon experience, as already remarked, to find the subject of expressive aphasia indicating by varied stress and intonation of his single word or few words how much meaning he is trying to put into them.

Thus an aphasic read aloud, "This paper must not be taken away" as "oh - ah - oh - ah - oh - ah - oh - ah - oh," with every endeavour by modulation and accent to show that he knew what he was reading.

It is impossible to say where expressive aphasia begins or ends and nothing is to be gained by hard-and-fast delimitations, which are, indeed, contrary to what one has reason to expect. In my use of the term I simply wish to emphasise the clinical fact that in the general class of aphasic disorders is a type characterised, above all, by this defect in audible expression of words.

Much discussion has centred round cases showing a variety of expressive aphasia known as 'pure or subcortical motor aphasia,' a particularly misleading term, though of the clinical occurrence of such cases there is no doubt. In them there is impairment or loss only of spontaneous and repeated speech ; reading and writing are intact ; the state is one of pure word-dumbness.

Liepmann's[1] explanation is that the majority of persons spell words inwardly as they read or write, especially the less well educated ; others, who do not to the same extent use motor components in reading or writing, are not likely to have reading or writing difficulties if a lesion involves that part of the general speech area where physiological mechanisms are mainly concerned with motor speech. The case of the polyglot lady mentioned above is a typical instance of pure motor aphasia ; no other aphasic symptom is present than almost total word-dumbness. An intelligent woman, she discovered that after the stroke she " could not hear her own words in her head," that is, could not recall word-sounds in thought, and this corresponded to her period of absolute word-dumbness ; recently, she has been able to say a few words (' Ida,' ' better,' ' butter,' etc.), and this recovery, such as it is, has coincided with ability to " hear these words in her mind." She now " hears numerous words," which as yet she has not been

[1] H. LIEPMANN, *Neurologisches Centralblatt*, 1909, xxviii, 449.

able to articulate. Data of this kind are of considerable importance, as showing that words are recalled in the auditory centre in her case, and that the effect of the lesion has been, by action at a distance, to impair transiently the power of word-recollection, a physiological mechanism of temporal site ; this function has now materially improved, and is perhaps almost normal, whereas word-expression has not kept pace with recovery of auditory word-recall, and is still extremely limited.

Some allusion has been made above to the localisation of the lesions which reveal themselves by expressive aphasia in varying degrees to complete mutism. Henschen's conclusions are, that if the lesion is in the posterior end of the third left frontal gyrus and implicates also the operculum of the island of Reil, that is, that part of the lower precentral gyrus which overhangs the island, mutism results. If there is no dysarthria, that is, if the patient's few words are correctly enough articulated, the lesion of this aphemia is in the frontal gyrus alone. Word-dumbness has also apparently been the result of

lesions interrupting the transcortical association paths that converge on this area from the temporal and occasionally those from the parietal parts of the general speech region, but cases of this character are possibly a little dubious. The matter is one requiring careful re-investigation.[1]

Allied to the above is a further interesting clinical variety whose chief feature is that the patient is not able to say spontaneously what he can easily repeat to order; similarly he cannot write spontaneously, or only very poorly indeed, whereas he copies and writes to dictation with fluency. Such cases bear a close resemblance to those cases of apraxia in which the patient is unable to perform spontaneously

[1] In a recent monograph Niessl von Mayendorf (*Kritische Studien zur Methodik der Aphasielehre*, Berlin, 1925) criticises Henschen's interpretation of the cases relied on by the latter for the conclusions given above, and advances again an older view of his own, that there is no 'Broca's area' and that fibres pass directly from the auditory area in the temporal lobe to the rolandic centres for articulation. This view, however, is itself open to no little criticism.

what he can do by imitating movements made in front of him. It would appear that what cannot well be said voluntarily, can be said on receipt of adequate stimuli from certain sense avenues. To this variety the name ' transcortical motor aphasia ' has been given, but we have seen that all aphasia is, physiologically, transcortical, so that the term is confusing and should be abandoned.

(2) Receptive Aphasia.

For that large class of case in which the patient is unable to understand what he sees or hears the term ' receptive aphasia ' is preferable to sensory aphasia. As regards audition, an aphasic may suffer either from word-sound-deafness or word-meaning-deafness, and these two conditions are distinct. A foreigner ignorant of English is word-meaning-deaf to the English tongue when he hears it spoken, but he is not word-sound-deaf. The words are not mere noises to him ; he knows the sounds are word-sounds. Similarly, as regards vision, the patient may be word-sight-blind, or word-meaning-

blind. In the former case he sees written or printed letters, syllables, and words as mere objects, and is unaware that they are speech-symbols ; in the latter he is conscious of their being symbols for ideas, but these symbolic meanings are not aroused by the sight of the words.

Word-deafness in the sense of word-sound-deafness sometimes occurs almost in a pure state. The sounds of words are not distinguished by the patient from any other sounds—in a wider terminology, this is a variety of agnosia —but his store of auditory memories of words is utilisable for reading, writing, and speaking, though his disability prevents him from repeating words on request and from writing to dictation. Before the diagnosis is made one must be sure that his actual hearing is unimpaired. In such cases the conclusion is warranted that auditory perception of word-sounds is mediated by a mechanism distinct from that which is in activity when auditory word memories are recalled, or, rather, that in them the field can be reached and the excitation of auditory ' en-

grams' effected from within (transcortically), though not from without, by the usual afferent route. In Henschen's collection two cases will be found of complete speech-deafness from bilateral temporal lesions, in which, nevertheless, expressive speech was normal, as was reading aloud. I cannot here examine all the details of temporal aphasia, one of the most instructive studies for the neurologist, but will briefly allude to Henschen's conclusions. From his analysis of hundreds of recorded cases of temporal lobe lesions he argues that the sounds of words are perceived by syllables; that this is a function of the first or upper temporal convolution, lesions of the posterior part of that gyrus being responsible for agrammatism (see above). For the comprehension of word meanings a wider area of the temporal cortex is requisite, especially the middle and posterior sections of the third temporal. Amnestic aphasia, as has been pointed out already, is specially apt to be associated with lesions of the lower temporal lobe, but it can certainly be found where

speech-deafness is not present. Minor lesions of the second and third temporal gyri are revealed by paraphasia, a condition found with great frequency in temporal abscess; as we have also seen, abscess is often associated with amnesia verbalis. The combination of paraphasia and of agrammatism, also referred to above, suggests definitely a fairly wide temporal lesion. The views of Bastian and of Wernicke, according to which alexia and agraphia are prone to follow on word-deafness, are refuted by Henschen.

Word-deafness in the sense of word-meaning-deafness is a condition of considerable interest. The patient hears words and knows that they are words, but these word-sounds, reaching his auditory centres, do not from thence awaken the meaning of the word, the concept for which it stands. Such patients often repeat correctly, parrot-like, what they hear, not knowing what it means; they write correctly to dictation, or copy, or read aloud, without knowing the meaning of what they read or write. To this variety the name 'transcortical sensory

aphasia ' has been given, a term which for reasons stated at some length above is obviously unsatisfactory and confusing. Of the reality of the clinical type, however, there can be no question, objectionable though the nomenclature is. ' Echolalia ' is the descriptive epithet applied to this variety of meaningless repetition of words. Naturally, perhaps, the patient is often taken to be demented ; the most striking example I have seen occurred in an inmate of a lunatic asylum. In severe atrophic lesions of the left temporal lobe the syndrome is prone to develop ; according to some authorities, echolalia is a function of the right temporal lobe in these cases, intelligent repetition of words being prevented by the left lobe defect. For further information the student is referred to the excellent monograph by Kurt Goldstein[1].

Turning, next, to word-blindness as a form of receptive aphasia, we find the same general principles applicable

[1] KURT GOLDSTEIN, *Ergebnisse der Neurologie und Psychiatrie*, 1917, ii, 349.

as in the case of word-deafness. Individuals differ as to the way in which they have been trained to read; thus the visual centres concerned may, or may not, be in close physiological association with other centres of the speech group. Henschen says well that "the scholars who learn a new language, such as hieroglyphics or the Assyrian cuneiform script, can read these with understanding before the pronunciation has been determined." It is appropriate to recall in this connexion the peculiarities of such a language as the Chinese; the Chinese read by ideographs, the symbols for ideas expressed in their reading being completely independent of their spoken tongue. As regards the English language, considerable diversity of word-blindness is to be expected. Sometimes letters can be read correctly, but not words, sometimes precisely the reverse. Sometimes numerals are read when letters and words are impossible; sometimes, further, pictures are 'read' in the sense that their meaning is fully grasped when the meaning of word-'pictures' cannot be aroused. From

these and other data that might be given, only one conclusion is possible, that the visual centres for speech form a sort of physiological mosaic in which distinct groups can be segregated, one or more of which may be lost through disease while others are still in activity.

Again, the varieties of word-sight-blindness and word-meaning-blindness are clearly distinguishable clinically in some instances. In the case of the latter, the patient can read aloud in a mechanical way and be at the same time ignorant or oblivious of the meaning of what he reads; exactly as a person of sufficient education can pronounce and read a foreign language from the text while every particle of meaning is hid from him.

Only passing reference can here be made to the condition known as congenital word-blindness, which presents certain curious features of its own that are of special interest to the neurologist. A recent communication by Orton[1] is likely to prove of much value in this connexion, suggesting as it does that

[1] S. T. Orton, *Archives of Neurology and Psychiatry*, 1925, xiv, 581.

the underlying defect is a congenital inability to distinguish between reading from left to right and from right to left. These patients cannot distinguish p's from q's, b's from d's, and so on, and cannot separate 'dog' from 'god,' etc.

A common corollary of any degree of word-blindness is inability to write, writing being largely dependent on knowledge and use of visual symbols. On the other hand, many alexics can speak perfectly well and exhibit no trace of word-deafness. Under my care at present at one of my hospitals is a patient from whose left parieto-occipital region a large endothelioma was successfully removed no less than twenty years ago. His present clinical condition is, that he hears and speaks normally, whereas his reading is minimal, being confined to recognition of letters and short words (capitals being read more easily than script), and his writing is an illegible scrawl, in which individual letters are with difficulty distinguished. There is no paralysis of his right arm, it need scarcely be said. The closeness of this association

of alexia and agraphia has been explained by some on the hypothesis of an additional writing 'centre' in the occipital lobe, since a few apparently trustworthy cases are on record of agraphia without any alexia following lesions strictly occipital in site, but this speculation is probably unnecessary.

Failure to recognise the meaning of words can be regarded as a particular variety of visual agnosia; for discussion in greater detail of the relationship of so-called 'optic aphasia' to agnosia as a whole reference may be made to the article on apraxia already mentioned.

Speaking generally, lesions in the vicinity of the angular gyrus are especially likely to reveal themselves by some form of alexia, the gyrus being regarded as having a parieto-occipital *local*; subcortical lesions in the distribution of the posterior cerebral artery, underlying this region, are also often causative of alexia, by transcortical separation of word-perception from word-recognition and word-association processes. In this case right homon-

ymous hemianopia is a frequent concomitant (for anatomical reasons).

(3) AGRAPHIA.

Repeated allusion has been made to the occurrence of agraphia in various aphasic syndromes; a word on its general aspect is perhaps called for.

Extreme care must be taken in the use of the term, for cases are not infrequently seen of inability to write with the right hand, in which, none the less, the left hand can be employed for the purpose with some success. Unless the attempt has thus been made and proved impossible, a condition of agraphia cannot be justifiably diagnosed. For that matter, an individual can learn to write with his toes, or can hold a pen in his teeth and trace correct letters by movements of his head. Writing, in short, is a very complex process, with visual and kinaesthetic components, probably also with an auditory component in some persons, since in a fair percentage of island of Reil cases the lesion, interrupting association fibres between the temporal and the frontal cortex which pass by the external capsule, has

resulted in agraphia. In view of the wide anatomical range (occipital, parietal, temporal, frontal) of the sites of association systems contributing their quota to the function of writing we can understand how lesions in different cortical areas can have agraphia for a sequel. These systems have elements converging on the eupraxic centre for writing in front of the middle zone of the rolandic region (arm centre). Some impressive instances of pure dysgraphia from destruction of this area, in the posterior end of the second left frontal gyrus, will be found in the literature; one of the best is that recorded by Macfie Campbell.[1]

(4) A brief comment on the interesting symptom of defect in spelling, aloud or in writing, must finish this part of our subject.

The process of spelling is clearly initiated, with most people, in the visual centre. Spelling is a matter of the order of arrangement of letters in a word, and this procedure must

[1] MACFIE CAMPBELL, *Review of Neurology and Psychiatry*, 1911, ix, 289.

depend largely if not entirely on visual processes, for we spell, in writing, by letter-signs and not by letter-sounds (although, of course, the signs represent or stand for sounds). If, then, dyslexia of any degree exists it is likely to be accompanied by inability to spell correctly, and this combination has often been found by the clinician. An alexic patient (case of gunshot wound of the occipito-parietal region) spelled 'good morning' as 'corr monnugnts.' It is more particularly in severe occipito-parietal lesions that those remarkable varieties of combined errors in spelling and use of totally wrong signs for letters are found, of which the following is an excellent example; it is the address on an envelope handed me by such a patient after being requested to write an ordinary letter.

Mr P1$\frac{1}{2}$lb$\frac{11}{22}$
6 $\frac{11111111}{22224222}$ $\frac{111}{222}$
1 $\frac{1111111111111111}{2444222222222222}$.

The letter closes with the following 'signature':

Yout c/2 $\frac{1}{2}$
$\frac{1111111}{2224222}$.

In respect of spelling by voluntary

recall of letter signs, this may be impossible while the patient is still able to copy capitals and script without any difficulty ; in such cases the meaning of the words is unknown, and they cannot be read aloud. That is to say, the visual symbols are copied as any drawing might be, or as the letters of the Greek or Hebrew alphabet might be copied by someone who is entirely unaware of their significance. Thus the visual centre can be stimulated from without when correct spelling and writing by volitional recall from within are rendered impossible by reason of disease.

In this sketch of some of the clinical varieties of aphasia no attempt has been made at a complete survey of the whole field ; it is hoped, however, that the 'lie of the land' has been sufficiently indicated for the clinician to find his way with some prospect of arriving at the desired end. For this purpose I have avoided complicating my subject by reference to certain aphasic phenomena of scientific and clinical interest which require separate study. For example, the symptom of palilalia, or repetition

(twice or oftener) of words, phrases and sentences; recurring utterance, verbigeration, and 'word-intoxication'; the condition of amusia and its numerous sub-varieties, and of acalculia (inability to reckon)—these all deserve annotation. To keep this little volume within the limits assigned, however, they must be omitted.

Another aspect of the subject that needs careful discrimination is the relation of aphasic symptoms in the strict sense to those of general intellectual deterioration, with which, in the nature of things, they are often found in combination. I may refer, for instance, to the ideational inertia and perseveration of the arteriopath or the senile dement, which will affect the processes of speech and give rise to symptoms in speaking or writing that are not specific, but can be found analogously in other fields of mental activity. I have before me the diary of an educated gentleman who eventually suffered from severe cerebral arteriosclerosis of a general kind; taking his average entries as comprising the following:

" Took out the dog this morning ;
Took him out again this afternoon ;
I did not go to church ;
Had a good night's rest " :

I find them replaced at a later date by the following :

" Took out the dog this mirughing;
Took out the dog this mirughng ;
Took out the dog agagalling ;
Took out the dog agallaagnn ;
Took out the dg alallaagn ;
Hoat a all good nighest ling."

Another arteriopath, essaying a letter, wrote as follows :

" Dear Mr Eynard,
I am sorry to sorry to sorry to sorry to sorry to sorry " . . .

And a general paralytic, similarly :

" Dear Brother,
I now find that I am quite out of the way of exprentishing myself all the same. I am right off exprensing myself. This is a case having lost the old reason. Wants and reason reasoned. However, my health is greating better and I am I much health " . . .

No more than passing allusion can be made to this speech deterioration as an aspect of general mental disintegration of no little interest and significance from the viewpoint both of the neurologist and the psychiatrist. At the same time it must be emphasised that the aphasic syndromes of such demented states are the result of invasion of speech mechanisms by the general disease and *pro tanto* are of localising significance.

Relation of Aphasia to Apraxia and Agnosia

At several points in this discussion attention has been directed to certain analogies that obtain between aphasic disorders and other transcortical disturbances of function conveniently termed agnosia and apraxia. The latter may be briefly defined as inability to perform certain subjectively purposive movements or combinations of movements, with conservation of motility, of sensitivity, and of co-ordination. Agnosia may be defined as inability to recognise objects, with conservation

of primary sense perception. Clinical varieties of each are known ; there are as many forms of agnosia as there are senses, and recognition of an object by one sense avenue may be at fault when it is possible through other channels. Another variety occurs, further, when a concept of an object cannot be formed, although the sense components are intact, and this has been described as ideational agnosia. As for apraxia, several varieties exist ; for details the reader may consult the article mentioned in my preface. Now evidence has accumulated which goes to show that the lesions of motor apraxia are transcortical, and that the cortical area associated with the function of eupraxia is in front of the central gyri, in the posterior region of the frontal lobe. When this cortical area is separated by disease from the Betz-cell region of the precentral gyrus, and (or) from the parietal and posterior parts of the brain generally, the patient will probably exhibit apraxic phenomena, that is, will be unable to utilise the limbs for various kinds of voluntary action, although they are not paralysed.

Since the articulatory muscles are not paralysed in motor aphasia, that symptom may legitimately be called an apraxia of the speech musculature. The motor aphasic can move the muscles of his lips, palate, tongue, throat, etc., though he is unable to reproduce the combinations of their movements requisite for the act of speaking. As the arousing of latent visual and kinaesthetic 'engrams' is essential for the execution of limb movements, so the motor or executive part of the speech function depends on ability to awaken auditory and kinaesthetic 'engrams.' If a patient cannot revive kinaesthetic 'memories' of complex acts he cannot make them on request; if he cannot arouse auditory word 'memories' he cannot speak spontaneously, though there is no paralysis of the muscles concerned. In respect of agraphia, again, we are dealing with a transcortical impairment of function consisting in inability to make certain consecutive series of fine finger movements, with conservation of the ordinary use of the limb for other purposes; thus in one sense the

symptom belongs to the group of motor apraxia.

On the afferent side, it is at once apparent that word-blindness and word-deafness are sub-varieties of agnosia as defined, and in the article already referred to will be found a more detailed account than can here be given of the relation of these forms of agnosia to the wider agnosia badly described as 'mind-blindness,' and known also as 'asymboly' and 'imperception.' The usefulness of this manner of looking at aphasic symptoms consists more in the analogies thereby drawn between speech disorders and disorders of other cerebral functions than in the strict similarity of the phenomena ; speech as a function is more complex than simple motor eupraxia, although on its receptive side it is no more than eugnosia.

Chapter VI

The Treatment of Aphasia

When the practitioner is faced with a case of aphasia it is often difficult to say what the outlook really is. Some severe cases clear up unexpectedly, others, slight from the beginning, remain uninfluenced by treatment. In the endeavour to give an accurate prognosis the following considerations are worthy of attention.

(1) It is true, as a general rule, that the more advanced the age of the patient at the onset of aphasia, the less likely is improvement to occur. Nevertheless, there are well marked exceptions to this rule. Some children, after an encephalitis causing aphasia, never make that improvement which we expect; and some old folk make a rapid recovery. In this connection a further consideration must be mentioned; the longer the interval between the stroke and the reawakening of speech, the less likely is the improvement to be material. To this

generalization I attach much practical importance.

(2) Still more depends on the severity of the original attack. In a large class of case the aphasia may be regarded as strictly transient, and in these the prognosis naturally is good. Reference is here directed to certain attacks of Jacksonian epilepsy, migraine, cerebral arteriospasm, to incomplete thrombosis or endarteritis obliterans, certain toxic and toxi-infective conditions, congestive attacks in general paralysis, etc. In such cases the aphasic disturbance may clear up entirely in a very short period. It is well, on the other hand, not to forget that transient aphasia from cerebral vascular disease may become permanent if an ictus occurs.

In an elderly patient with aphasia definitely established, the following points may be put before the physician as worthy of remark.

(*a*) If there is little or no indication of generalized cardiac, arterial, or renal disease, the outlook is better than if such systems show signs of degeneration.

(*b*) Should the aphasia be unaccompanied by any paralysis, facial, brachial,

hemiplegic, etc., the chances of improvement are better than otherwise. Palsies are simply an indication of the degree of the lesion, and the larger the lesion, the less likely is improvement to take place.

There is evidence to show that some cases of aphasia improve because of functional restitution in parts of the brain damaged but not destroyed by the lesion; in other instances, the evidence suggests that auxiliary speech mechanisms develop or come to fuller development in undamaged areas of the cerebrum, a condition of functional compensation. Over this matter much controversy has raged and it would occupy too much space in this small volume to attempt to resume it. As was shown in an earlier section, both hemispheres are largely concerned in the acquisition of speech on the receptive side, and the right hemisphere has speech potentialities to which insufficient attention has been paid. Doubtless Hughlings Jackson was right in his 'guess' that that hemisphere was more concerned with automatic than with creative action, and it may

well be that some at least of the imperfect speech of the aphasic is the outcome of the incompletely equipped right hemisphere trying to make up for what is awanting in the left.

In all cases of aphasia treatment should be directed along two lines, that of the causative morbid process and that of the symptomatic condition once it is established.

Of the former (embolism, thrombosis, haemorrhage, inflammation, tumour, etc.) nothing need be said in this place.

In regard to the latter, a number of cases are on record in which the organs of speech have been re-educated *de novo* with encouraging results. As I have said in another place, "the evidence favourable to the re-education method is ample enough to render its comparative neglect by the profession rather surprising." It consists essentially in imitating the procedures by which a child first learns the elements of speech. Dana[1] has given an excellent technique for the details of which

[1] CHARLES DANA, *Studies from the Department of Neurology, Cornell University*, 1904.

his papers may be consulted. Another method is more strictly phonetic, consisting in teaching the patient the letter-sounds of each vowel and consonant. A combination of this procedure with lip-reading is often of value in motor aphasia.

It has been held that sensory aphasia is more difficult to treat successfully. In a case of word-deafness the aim of the teacher, physiologically expressed, is to develop the auxiliary right temporal centres and to bring them into functional relation with the frontal part of the speech area proper. With cases of word-blindness similar methods are adopted. The tactics pursued are somewhat as follows : the patient first of all learns the alphabet, attention being concentrated on each letter in turn ; the letter is pronounced by the teacher and lip-reading is resorted to. Thus afferent impressions from more than one sense avenue are being utilized together. Then monosyllabic words are tackled, the patient spelling aloud letter by letter, while the teacher pronounces each letter and the former also reads by the lips.