Before accurately defining aphasia and describing its different forms, it is not without interest to take a brief retrospective glance at the principal stages of evolution through which our knowledge on this subject has passed, from the time when it was regarded as a mere morbid symptom noticeable to everyone though unexplained, down to the present time when its chief clinical and pathological varieties have been differentiated.

Before the end of the 18th century very little light was thrown on the subject. Thenkous observed that some patients could not speak owing to loss of memory of words, the tongue being unparalyzed.

Van Swieten, Foranui, Cullen, and others certainly distinguished theoretically between the symptoms of motor and sensory aphasia - or aphonia; but not referring to their cases the differentiation can hardly be made out. Later, Crichton published cases of prevented memory, which certainly may be classed under the head of aphasia as we now understand it. In 1820 Professor Lordat of Paris ascribed Alalia (as the symptom was very generally called), to a loss of co-ordination of the muscles subserving speech, not to paralysis of the tongue - with Gall, however, begins a new epoch in the study of this symptom for he conjectured that the center for articulate speech was in the anterior lobes of the brain. Bouilland, in 1825, followed Gall in localizing this center in the same cerebral region, but he further carefully distinguished

between one series of symptoms, arising from loss of memory of words, and another due to loss of co-ordinating power over the muscles sub-serving speech. This is the first attempt at differentiation between cortical and sub-cortical motor aphasia. During 30 years he held this view, and in 1848 further aided pathology by teaching that in many cases of aphasia he had found a lesion in the posterior part of the frontal lobes. Notwithstanding that his antagonists, amongst whom were Cruveilhier, Velpeau, and Andral, repeatedly published cases, which exhibited, either loss of speech without any lesion in the frontal lobes, or, marked destruction of these lobes with no attendant disturbance of speech. Dr. max. Dax read a paper at Montpellier in 1836 in which he elucidates the question by noting that according to his experience in cases of aphasia with hemiplegia, the latter always affected the right side of the body and concluded that the lesion causing aphasia was situated in the right hemisphere. This paper was not published until April 1865 (Gazette Hebdomadaire). In 1861 Broca happened to have the opportunity of examining two very typical cases of motor aphasia at Bicêtre Hospital, before and after death; from these he concluded that the centre for articulate speech was situated in part of the 2nd and 3rd frontal convolution on the left side close to the fissure of Sylvius and overlapping the island of Reil. He arrived at this conclusion independently of Dr. Dax whose paper he had not seen. Troussseau admits the fact that a lesion in the left frontal lobes accompanied certain forms of aphasia, but protests against localizing the lesion to a particular spot. Since then hundreds of post-mortem examinations have proved that this motor

Aphasia was correctly described by Broca, confirming evidence having been furnished by Ferrier's experiments, showing that this region is the centre of articulation in monkeys, and insisting, however, that this centre is represented on both sides of the brain, a point of great importance as we will see further on.

Intimately related to, and often complicating Ktor's aphasia, cases had been from time to time noticed, which could not be explained, except by supposing that the loss of speech was due to a disturbance of sensory centres or tracts in the brain, thus interfering with the transmission of impressions from outside to other parts of the organ. Ogilby, Popham, Broadbent and others reported such cases, but it was not until Virchow and Bastian in 1874 gave a full clinical and pathological description of his cases under the heading of Sensory Aphasia, to distinguish them from the better known variety, described by Broca. It is maintained that in addition to Broca's motor centre, speech in the widest sense of the word, was dependent on the integrity of two additional centres, namely the Word-reading and the Word-writing centres, the former he localized in the Superior Temporal Parietal Lobes, the latter in the Occipito-frontal region of the left hemisphere; these three areas with their intimal nervous communication from the zone of Speech. This discovery together with Bastian's notable work on the subject made a second epoch in the evolution of our knowledge on the subject. Since then the careful analysis of facts and record of cases furnished by Broadbent, Ferrier, Bastian and more especially Charcot have given us a much more definite conception of a subject than we could have hoped for.

1) Medical Times 1874 Vol. 4
4) Phonationsymptomencomplex
5) Die Sprach Störungen.
at one time, considering the complexity of psycho-physiological elements with which it is connected. Even now, as we shall see, there are several points on which authorities do not agree, which stimulate the careful examination and publishing of individual cases.

In speaking of aphasia, one does not refer to defects or absence of speech which are dependant on altered functions of the peripheral organs of expression, neither are cases included in which the symptoms are obviously due to a general want of intelligence as in mental idiocy, mental imbecility, and such-like mental affections, which have generally existed in the individual before he has acquired speech; I lastly, we exclude congenital dumbness, whether congenital or acquired within the first years of life.

Broca apparently meant to limit the term aphasia to a loss of speech from a cranial lesion, subsequent to the period when speech had been fully acquired by the individual. If we look upon speech as a generic term for "forms of expression" (the three principal forms being articulate language, writing, and gestures), we find that this was practically the definition which Faubert gave; he said, "Aphasia means a defect in one or more of the modes of expression of thought, such as speech, writing, gestures, drawing, music, poetry, without, however, involving the general intelligence or the peripheral organs of phonation." This is perhaps the best definition that has been given, as it lays particular stress on the unimpaired condition of the intelligence and of the muscles used in articulation, but it only applies to that form which Broca described and in which the patient has lost to a more or less marked degree the conventional forms of expressing by which he can communicate with his fellow creatures. It is
clinically the commoner & is termed motor Aphasia. In it we find to a varying degree
1. Loss of Speech.
2. Inability to write (Agraphia).
3. Loss of power of expression by gesture.

The deficiency in the last form of expression being rare.

Lately however our knowledge in this branch of nervous pathology has widened & we can no longer accept their as a general definition. For Aphasia includes a second group of cases in which the patient cannot exchange ideas with another person, owing not to any defect in articulate speech or power of writing but owing to the loss of that power by which we interpret the conventional symbols used in expressing our thoughts, i.e. by speech or by writing. In other words

Motor Aphasia is an Aphasia of Expressioni. Sensory
Aphasia, one of impression.

In order to include both varieties we must define Aphasia, as a defect in one or more of the memories used by us in communicating with our fellow creatures due to a destructive cerebral lesion. The defect will vary according to the centre or centres involved & it will be explained further on how symptoms observed depend on our memories or recollections.

Varities of Aphasia

Two main groups with their subdivisions will be found to embrace the different pure varieties, i.e. those due to a lesion in one of the centres used in communicating with one another.

The reasons for excluding a writing centre, as advocated by Charcot & his followers, are given further on.

For will it just place emmunicate these varieties & then proceed to give a clinical description of each from cases seen.
Examined.

I. Motor Aphasia

A. Cortical - involving the centre for motor images of words. This lesion being in the part of the 3rd frontal convolution (area 44) in the left hemisphere. (Broca’s)

B. Subcortical variety - in this the foregognic centre is uninjured, the lesion implicates however the different fibres passing from this centre to the internal capsule. It can be looked upon as a center for coordination of the muscles subserving speech, but is absolutely dependent on the first name cortical centre.

II. Sensory Aphasia in which the lesion implicates the supramarginal and angular convolutions in the left hemisphere.

A. Pure Word deafness - in which Wernicke’s word hearing centre is alone involved i.e. the supramarginal convolution on the left side.

B. Pure Word blindness - In the word seeing centre in the supramarginal and angular convolutions are alone implicated.

Type of motor aphasia.

E. J. aged 60, a shoemaker by trade, found suddenly one evening that he was unable to utter a single word & noticed at the same time a certain degree of paresis on the right side of his body. The patient was perfectly conscious throughout the examination. The day after he presented the following symptoms:

He is a strongly built, intelligent looking man & is suffering
from slight hemiparesis, affecting the face, arm, and leg, sensation is perfect on both sides of the body. The paralysis is so slight that he can walk and move his arm fairly well. In answer to any question put to him, he can only repeat the syllable to, to, to, toit is his invariable answer and is quite unable to say anything else. He can protrude his tongue and move it about in all directions, the outline of the soft palate is unaltered. The mouth is in the middle line. He swallows with ease, he can whistle, the facial paresis is too slight to occasion any obvious unilateral deviation. All these points show that the loss of speech is not due to a deficient action of the peripheral muscles. His intelligence is apparently unimpaired; we tested it roughly by placing a number of articles within his reach and telling him to pick up the one we named, thus he did with ease, he never made a mistake. He can read perfectly to himself and understands what he reads. He makes some effort to answer our questions and shows signs of annoyance or anger, initially taking recourse to gestures (being the most primitive form of expression, it is generally retained and only greatly developed in some cases). He nodded the right hand. I asked his age, he opened and shut his hands six times. When it was suggested that he was 63, he shook his head, on the right number being mentioned he nodded appropriately. His writing is slightly less affected than his speech. The paresis of the right arm was so slight that we had to look elsewhere for the cause. If I could write his name easily, his address is not so easy to make out, as he leaves out letters here and there. When told to write down an account of the present attack, we could only make out something for not only were words misspelt, but inappropriate words were used so that it was quite senseless. We were further struck by the effort that this simple test cost the patient.
the slow rate at which he often travelled, his concentrated attention, the evident sigh of relief with which he handed us the specimen reminds us forcibly of a child who was learning to write.

In this patient, therefore, the striking symptoms are:
1. Inability to express his ideas by means of articulate words on writing. As in the majority of these cases, he could tell us his story.
2. Gestures are the only communicating agency retained by him. His intellect is clear, the peripheral muscles of articulation are perfectly under control and the parast of the right arm could not account for his deficiency in writing, for he could copy printed matter quite well, but failed at putting dictated sentences intelligibly on paper. On the other hand, this patient understood perfectly what was said to him, read and understood what he had read.

Subcortical motor aphasia. In this form, which is rare, the lesion affects the neurones leading from Broca's convolution to the internal capsule; these fibres are purely motor and can be looked upon as the coordinating centre for the movements of articulate speech. Kliethenius in 1885 described this form of aphasia under the name it now bears.

The symptoms are as follows:
1. The patient generally is hemiplegic on the right side.
2. Voluntary speech, the repetition of a word pronounced and reading aloud are abolished.
3. He can read to himself and understands what he reads.
4. Can write spontaneously, when dictated to, he can copy.
5. Lastly and most important as regards diagnosis, the motor images of words are unaffected, so that the
patient can correctly communicate by signs the number of syllables that a given word contains although he can not pronounce it.

These symptoms are not due to any paralysis of the tongue or muscles of the throat or larynx as can be ascertained experimentally - Dejerine points out that in this form it frequently occurs paralysis of the right vocal cord, but this is simply a coincidence and is explained by the fact which V. Hondey has demonstrated in monkeys namely that the laryngeal nerves used in phonation have a cortical centre at the base of the ascending frontal convolution - allowing the analogy in man, the close proximity of these two sets of fibres would account for the relative frequency of the complication.

We find therefore, that in both the cortical and subcortical varieties of aphasia the principal subjective symptoms are identical - in both cases there is generally right hemiplegia, which is often transient, but both the patient cannot speak.

In the cortical variety the patient can neither write spontaneously nor when dictated to; this will only improve pari passu with his vocabulary. He is quite unable to express by signs how many syllables a certain word contains.

In the subcortical variety, as we have seen, the patient very soon acquires the use of the left hand (if hemiplegic) for writing purposes and will express his ideas by dictation. This means with ease. In his attempt at repeating a given word one will notice in the expiratory sounds that the number of syllables in the word are correct - the fundamental difference being that in the
Former case the motor images of words, normally stored up in Broca's centre, are destroyed; in the latter this image or conception is intact, only the coordinating parts being destroyed.

We will proceed by giving a case of word deafness, one of word blindness and lastly one combining these two symptoms and thus presenting a type of total sensor of aphasia.

**Word deafness.** B.M. aged 50 in whom the following symptoms came on suddenly: if the patient is asked a question, he looks at one with a puzzled look. Speaking louder does not improve matters. We tell him to pick up something by his side, but he takes no notice; he is moreover, by no means deaf; for he turns his head at the least noise or when he hears his name pronounced. He apparently does not understand spoken words, for they do not evoke any ideas, and even sounds without meaning. He is in the position of a person, who is addressed in a language unknown to him. In order to communicate with the patient, we have to take recourse to writing, thus he reads quite readily and answers any questions either verbally or in writing. Articulate speech therefore is writing are quite under his control. Dictated matter he cannot put to paper, as he does not understand what is said. He can copy perfectly.

In this case the word forming mechanism is not affected—

In him it is the word hearing centre which is damaged for spoken words are meaningless to him; but by writing down main questions, thus communicating with him through
his wordfinding centre, he can express the ideas expressed either by speech or by writing. He must however be classed as an aphasic, for though words are no longer vocal symbols of ideas.

**Wordblindness.**

C.B., aged 45, whose intelligence is apparently unaffected, discovered one day that he was unable to read. He can see the letters but does not appreciate their meaning. There is no alexia. He recognizes his name and can point it out from amongst other words which somewhat resemble it. If we place put down the letters R. F. on the blackboard, he was unable to name either but as soon as we drew the outlines of a shield or coat of arms round them he pointed at them exclaiming, "Republique Francaise", showing that he preserves the initials with the shield as well Roman symbol, as the same way as he retains his name as a whole, the separate letters being unintelligible to him. This defect in the power of reading is not due to defective sight, for he can pick out any object named, we tried however by the perimenter that he has right homonymous hemianopia as is generally the case when the lesion is situated in the occipito-angular area.

As regards writing he can only put down his name, if he is equally unable to write from dictation, and if told to copy points he does so mechanically, as if it were a drawing, that this is a great effort is evidenced by his taking 1½ hours to copy the word "EDITION", his method being to trace the outlines of the letters and then to fill in the dark parts with ink. We paid justice that we cannot make him grasp the meaning.

of written language, by allowing him to trace the outlines of the letters with his fingers, his sense of muscular movement is useless for the destruction of the word seeing centre implying loss of the visual images of words.

His speech is somewhat affected, as is very general in these cases; for although the patient's intelligence is not altered enough to account for any of the symptoms named, there is no doubt that in Aphasics generally, especially in cases of injury to the occipital angular region, there follows a certain amount of mental deterioration.

Such are the prominent symptoms in a case of wordblindness implicating the word seeing centre itself.

There is a modified type which occurs in cases of interruption of the communicating fibres between the word seeing and word hearing centre. As these cases are also wordblind, the symptoms are less severe; we will enumerate the latter as such cases are often quoted as pure cases, though only indirectly, affecting the word seeing centre itself:

1. The speech is slightly impaired
2. Can read very imperfectly, often only his name.
3. Right homonymous hemianopsia generally present.
4. His writing is good; but as regards spontaneous writing, writing from dictation or copying, the hemianopsia will modify this somewhat.
5. By resorting to tracing the outlines of letters with his finger either on the paper or in the air, he can slowly spell words which are otherwise meaningless to him. The sense of muscular movement calls forth the visual images of words in the word seeing centre, the letter seeing in this case unaffected.
Total Sensory Aphasia. T.S. aged 78, has been a tailor by trade. Patient was admitted to the Bicêtre Hospital, as his age prevented him from following his occupation. His personal history was good. In April 1891 he was seized with profound aphasia, from coma and paralyse, which was more marked on the right side; in a few days he began to recover, but suffered intermittently during two months from less or severe attacks. Eventually these passed off, and the hemiplegia got better, but the following symptoms persisted:
1. He understood nothing that was said to him, answering however to his name.
2. Could not read a single letter, but pointed out his name when the latter was written down amongst other words.
3. Was quite unable to write spontaneously or when dictated; copied written or printed matter as if it were drawing—very slowly and with great effort.
4. Right homonymous hemiplegia.
5. Could speak a little volubly, not only occasionally, but there was a word with any meaning, the rest was a mere jargon (Paraphasia).
6. He recognized objects apparently knew the use of any familiar object, indicating that the symptom which Munk called psychical blindness was not present.

Such were the symptoms after recovery from the repeated acute attacks. At the present time, 2 years after the lesion occurred, the patient can understand with difficulty simple questions addressed to him. He can read a little as evidenced by reading aloud from a newspaper, as long as the words are short and familiar to him. But he stumbles as soon as these conditions are not fulfilled. His speech is very bad, apparently ranging considerably. However, for on some days
he seemed to have less difficulty in expressing himself than on others. His writing had not improved since his attack, for his name is the only legible word that he can put on paper.

It has often been noticed that, aphasic, in whom writing is practically lost, can put down numbers and make simple arithmetical sums.

In this last case we find a combination of the symptoms described under word deafness and word blindness with an additional mental deterioration, which is but natural considering the age of the patient and the length of time during which he has been unable to communicate with anyone except by signs."

Word deafness and word blindness are the only accepted subdivisions of sensory aphasia at present, and it is not likely that more than these can be differentiated, considering that we communicate with one another either by means of speech or writing (gestures are obviously quite inadequate), appealing in the one case to the sense of hearing, in the other to sight or vision. We can however imagine a third form of sensory aphasia in a person whose education a third sense to an unusual degree.

In a blind person, who can only read by an extraordinary development of the tactile and motor sense, we can imagine that a cerebral lesion might render the patient unable to judge of the relative value of letters - he would rank as a case of sensory aphasia. Although this is conceivable, I do not know of any such observation being recorded.

(1) Dr. West's case reported in Semanics Medicae 1833 p.221.)
Hanig described the clinical features presented in the collected cases, we will proceed to point out the pathological brains associated with them.

The introduction has shown that the symptom under consideration was the first to stimulate enquiry with regard to cerebral localization, and to Broca is the honour due of having established by post mortem examination the centre for the memory of spoken words. Thousands of cases have corroborated this discovery, and it is universally accepted that a lesion in the posterior part of the 3rd frontal convolution of the left hemisphere, close to the fissure of Sylvius, where it overlaps the island of Reil, causes the symptoms of cortical motor aphasia. Tanis has found this area to correspond with the centre of articulation in monkeys. The lesion is almost invariably found on the left side, although the centre is represented in the right hemisphere as well. Sequin collected 266 cases of motor aphasia with hemiplegia, of these the great majority were left-handed people, who have, in nearly all cases, educed the various centres in the right hemisphere more highly than those in the left, and in whom consequently, the position of the lesion is reversed as compared to right-handed persons. Considering two near this centre is to the Rolandic centres for movements, it appears that motor aphasia without paresis or paralysis is rare. Bastian cites 3 cases, the first due to occlusion of the external and internal branch of the middle meningeal artery, which supplies Broca's convolution exclusively, the second was due to a localized abscess, and the third to

(1) Analysis Cerebri, Bulbar + Spinal, p. 290.
a spicule of bone embedded in this region — we have already referred to the lesion in Subcortical Motor Aphasia being due to an interruption in the fibres passing from Broca’s centre to the internal capsule. These fibres may be identified with a centre of coordinating Swervig Speech.

The pathology of Sensory Aphasia is more difficult, partly because the cases are rarer and its study is of comparatively recent origin and partly because psychology plays a leading part in establishing the causal relations between lesions & symptoms. — The following facts are however established.

Wernicke maintained that the area of Speech comprised the motor centre of Broca, the word hearing & the word saying centres all these being intimately connected with each other by commissural fibres and with the corresponding parts in the right hemisphere through the Corpus Callosum. He also was the first to publish a the account of a post mortem examination held on a case of pure word deafness; he found that the lesion involved the centre of the left Superior Temporal Pheroideal lobe. Since then Broadbent recorded another & Seppilli collected 17 cases in which this lesion was found (in 12 of these cases the Second Temporal Pheroideal Convolution was also involved). These clinical observations were further indirectly confirmed by Ferri’s experiments, in which he localized the Auditory centre in the posterior

1) Wernicke: Der aphastische Symptomen Complex. Bruxelles 1874
3) Recita Speriment. di Ferreriat Vol 5. 1884.
division of the 3rd external convolution in the dog and its homologue the superior temporal sphenoidal in the monkey. We have, therefore, in each hemisphere a cortical common auditory centre, part of that on the left side being further differentiated so as to store up the auditory images of words. In an extensive bilateral lesion in this region, the result would be complete deafness, whereas a destructive lesion in the left side alone would involve the memory of spoken words and produce typical wordblindness with the symptoms already described, without interfering with the common sense of hearing, which appears to be carried on perfectly by commissural fibres from the right hemisphere.

Wordblindness is in the same way associated with certain anatomical lesions, which have repeatedly been found post mortem although pure types are decidedly rare. The convolutions around the posterior extremity of the horizontal limb of the Sylvian fissure are always implicated. Further, it has been shown that the cortical area subserving vision is the occipitoangular region in both hemispheres and that the angular gyrus is the most important part. We meet with cases of wordblindness accompanied by right homonymous hemiopia (and it is the more frequent) and also pure wordblindness without alteration of the field of vision; this points to one part of the common visual centre (probably part of the inferior parietal lobule) being educated to receive and retain visual images of words and that if the lesion implicates the angular gyrus homonymous hemiopia of the opposite side is the result. It is also the left hemisphere is generally the one more highly diff-
entrusted, as will be pointed out further on, however, the elements of these sensory centres on the right side are hardly susceptible to education, in marked contrast to the motor centre.

As regards the explanation of opposite homonymous hemianopsia in relation to cortical destructive lesions, the various theories advanced to account for clinical facts would seem too far. We must however dwell on the symptom which Brunet termed Psychical blindness, although its pathology is not fully established. The literature on the subject is extensive and I propose to quote a case which Brunet published (2), in which the symptoms are well marked.

The patient was a left-handed man, who had learned as a child to write with his right hand. If different articles were placed near him and he were told to pick one up by name, he did so readily; he could specify its use. When, however, he was shown an article he was told to name it and indicate its use, he was almost always wrong. This could not be explained by loss of memory of spoken words, for he was by no means word deaf. Complex blindness (according to Charcot) might account for it if the word-sensory centres were much developed, but in this case the patient was a peasant who could not even read fluently. There was true psychical blindness for certain objects, which the patient quite well before his illness. This applied not only to articles shown to him, but also to images evoked by memory, for he could not recollect

(1) For details see F. Forel, 'The Functions of the Brain,' Chap. 17.
(2) Brunet de Midecines, 1885, p. 636.
the relative situation of certain streets, houses & monuments in his native town, which he otherwise would have known perfectly. In some cases, he would, after much hesitation indicate the use of an article shown him, more rarely would give its name correctly, but he never found the latter before the former was clear to him.

In following the path of impressions, we find that we see an object by means of our common visual centres. Further, the object evokes the memory of its significance, and we recognize what is held before us. The word-learning centre is then called into play, and we remember it, its name.

This explains why the patient invariably recognized the use of the article before giving it a name.

Brunn himself does not admit that the symptom is due to a lesion in the communicating fibres between the word-seeing and word-learning centres. But in this case, on stimulating the latter, the former reacted perfectly, and one would have to allow the existence of a double set of fibres, which he does not think plausible (Broadbent, however, inclines to this view, judging from his comment on D. W.'s case[1]). He maintains that this symptom is probably very frequently overlooked in cases of word-blindness, as it is only perceived if a patient be asked to give the name or illustrate the use of an article shown him. His explanation is as follows: He cannot admit that there are separate centres for the memory of objects and of words, for the child learns the significance of a letter of a certain shape, just as he learns the meaning of a word upon its being articles having a certain shape. Each item, the significance
of which is acquired by the brain, and the memory of which is stored up and can be called up at will, is localized in one of the millions of cellular units which form the cortical layer. The sum total of the impressions thus acquired by vision has been identified with the occipital-angular convolutions. According to the extent of the injury in this region we may therefore have loss of visual memory for certain words, letters, images or objects and in total destruction, all visual memory will disappear. In such cases psychological blindness will be completely, or incomplete, varying infinitely in its details.

It seems therefore probable, that in cases where the patient cannot recall the names of familiar objects, but knows their use, the communicating fibres between the two principal sensory centres are at fault, where, however, the patient has lost the recollection of both attributes, as in the quoted case (in which left homonymous hemianopia was present) the angular gyrus is the site of the injury. In either case word-blindness would be well marked.

In order to accurately interpret the various clinical symptoms presented in such cases and to recognize their pathological significance, it is necessary to briefly study the parts played by these centres in the growth and development of speech in the normal subject.

Articulate speech and civilization have advanced hand in hand, the higher the standard of intellectual culture, the
race, the monkeys' language developed - articulate and written speech depend simply on conventional vocal or written symbols, which, through the nation, have a uniform value. These are the two forms of mutual communication for we set aside gestures as totally inadequate to express our ideas. An idea is any association of impressions received from without & those entirely acquired; in the newborn child he has no innate ideas, he only possesses a brain which, in virtue of natural selection & heredity, contains elements that may or may not be developed into intellectual culture, according to his surroundings & education. For attaining mental superiority, therefore, we rely mainly on our organs of sense and of these the most important are the eye & the ear. By these two we acquire a general notion of an object, in order to further differentiation from similar objects, other senses such as touch, smell, taste, etc. are brought into requisition. These well-defined impressions, which are called 'memories' collectively, are stored up for future use in the brain; it is evident, therefore, that in order to express an idea on a given subject, both this memory & the outward conducting path must be in a normal state.

It is important to remember that the expression 'memory' implies a number of separate memories, one for each sense and we can speak of an auditory, visual, tactile, gustatory or olfactory memory. To illustrate this, let us suppose that we wish to develop in a child the idea of a common object, such as an orange. By placing an orange before his eyes - an impression of its outline and colour is conveyed to the retina,
This is transmitted by the optic nerve to the corpora quadrigemina, where it gives rise to a sensation, which it passes to the common visual centre (occipital-angular region) and becomes a distinct perception. By repeating this process a certain number of times, we succeed in forming the visual memory of an orange in the child’s brain, so that, if it be shown another subsequently, he will recognize it. A functional differentiation has taken place (probably in the neighborhood of the inferioparietal lobule) in certain cells, by virtue of which they are not called into activity in future, except by the stimulus which has produced this primary change in them through the retina at this stage. The child is not able to differentiate between a yellow ball and an orange, so we must supplement his conception by making him repeatedly taste, touch, smell and feel the fruit so as to develop gustatory, tactile and olfactory images in his brain. Then, then only, will he have a perfect idea of an orange as a definite object. At this stage, the sight of an orange will evoke the various sensory images connected with it, and he will recollect its taste, smell, etc. The same will happen if one appeals to any one of the other good senses, the other images will be called up to furnish the full conception required, these centres being all intimately connected with each other. This method, with obvious modifications, applies to all surrounding objects.

At this stage, the child has acquired a certain number of sensory images of objects, and the next step consists in teaching him to express them by speech and by writing. As the former
necessarily precedes the letter we must educate his motor centres for articulation. The preparation to this step, is begun by the imitative faculty of the child, by virtue of which, he repeats often heard words; these he repeats as mere meaningless sounds at first, but by that means educates the coordinative centre for the movements of articulation. The difficult process however is to establish a lasting memory of these movements in Broca's convolution - A familiar object is shown to the child and its name repeatedly pronounced thus producing a fresh sensory image of the object for he will in future associate this auditory symbol with his previously conceived sensory images and gradually develop a memory for words and with it a word hearing centre. He next gradually acquires the power of imitating that sound by virtue of communicating fibres between the word hearing and word forming (motor) centres and thus acquires a motor image of words and the image of objects. Paralleling with the development of these motor and sensory images, a higher centre of ideation is acquired by which he can spontaneously interpret his own ideas and think out the meaning of complex sensory impressions. It is important to remember that the motor centre is in its development entirely dependant on the word hearing centre.

Once the child has learnt to speak, he is taught to read i.e. to associate certain letters and combinations of letters with familiar objects. This is accomplished by pronouncing the word and forming the written equivalent i.e. the sound renews the auditory image and with practice he will associate written words
with the sensory images of objects which he has acquired, these written symbols become impressed in part of the visual centre. It constitutes the word-learning centre, its function being the memory for written words. The child only understands the meaning of written words after having pronounced them. This translation of written languages into revived sounds before they are understood persists in many people, who are not much accustomed to reading, and who whisper the words to themselves as they proceed.

Finally, the child is taught the more complex action of writing. His hand is trained to execute the fine movements necessary to copy letters, and when certain muscles are under his control, he proceeds to produce on paper the words which have become familiar to him in the process of reading, and thus express his ideas by transferring their symbols from his word-learning centre to paper. More copying, especially when printed matter is not even rendered in his own handwriting, takes place mechanically, and depends on the common visual centre for muscular movements. In writing from dictation, his own words, writing the word-learning and word-seeing centres are called into play as well.

Charcot maintains that there is a separate centre for writing (centre graphique), which he localizes in the posterior part of the left frontal convolution in the left hemisphere. To this theory, which he advanced in 1883, we will refer more fully further on.

From this sketch it is clear that the word-learning centre is the one which promotes the development of the word-seeing and motor centres from the beginning.
In order to render more clear the reason for the various defects met with in the different forms of aphasia, I append a rough diagram of the centres and connecting fibres essential to a normal condition, and will briefly enumerate these defects with reference to the diagram, at the same time examining each case in the following order: As to the patient's capacity for:

1. Spontaneous speech
2. Repeating words
3. Understanding what is said to him
4. Reading aloud
5. Understanding what he reads
6. Spontaneous writing
7. Writing from dictation
8. Copying written or printed matter

In critical motor aphasia A is destroyed & with it the memory for the motor images of words. The symptoms are:
1. Spontaneous speech abolished or impaired
2. Cannot repeat words spoken to him
3. Understands perfectly what is said - for the paths from the ear to the word hearing centre & to the centre of ideation are
not affected.
4. Cannot read aloud for obvious reasons.
5. Understands what he reads unless he is accustomed through want of practice to pronounce the words.
6. Cannot write spontaneously, for words are reproduced in the motor centre before being heard mentally and then committed to writing. Commonly employed words, such as the patient's name, are so firmly impressed as complete images, that they can generally be expressed automatically.
7. Cannot write from dictation for the same reason.
8. Can copy intelligently i.e. with own handwriting.

I

In the Subcortical variety of Motor Aphasia: E is alone destroyed (E. standing for the motor fibres which interpret the motor word images by means of voluntary muscular movements). The motor images of words are intact, so that the consequences will only affect articulate speech. The patient will be incapable
1. To speak spontaneously
2. To repeat words
3. To read aloud
All other processes will be unaffected.

II

Subcortical Aphasia.
A. In Pure Word deafness - B (i.e. apr.) is destroyed & hence
1. Spontaneous speech is uninpaired.
2. Obviously he cannot repeat words for to him they convey no meaning.
3. Does not understand what is said to him
4. Understands what he reads.
5. Spontaneous writing is uninpaired.
7. Obviously he cannot write from dictation.
8. Can copy perfectly.

B. In pure wordblindness C (v. diagram) is destroyed i.e. just:
1. Spontaneous speech somewhat impairs.
2. Repeats words spoken in his presence perfectly.
3. Understands what is said.
4. Cannot read aloud, the visual images of words being destroyed.
5. Cannot understand written language.
6. As a rule he cannot write spontaneously, but as we pointed out, in certain cases the patient can do so, but is quite unable to read what he has put on paper.
7. Cannot write from dictation.
8. Can only copy mechanically. The patient never renders printed matter in his handwriting, but copies it like a drawing.

C. In total sensory aphasia, both B + C (v. diagram) are affected. The patient
1. Speaks volubly but unconnectedly.
2. Cannot repeat words.
3. Does not understand what is said to him.
4. Cannot read aloud, and therefore
5. Does not understand what he attempts to read.
6. Cannot write spontaneously.
7. Cannot write from dictation.
8. Copies words like he would a drawing or picture.

The intimate relation of these centres with each other will explain two processes which are originally acquired with difficulty, become with practice almost automatic; thus we have seen that at first written...
language is translated into articulate speech before it is understood, by practice however this is no longer
needed & writing we interpret the visual images of
the words directly, without the intervention of the auditory
image - in writing practice again enables us to
dispensize with mental articulation and the consequent
auditory image, for we transcribe our ideas directly
by means of the words writing centre and certain
manual movements - Education of these centres
enable us to go a step further, for one may read
aloud without giving a thought to the meaning,
for the words perceived are so familiar that the
visual images are transformed into motor images
without automatically, the intelligence not being
requisitioned at all - In the same way we can
copy written matter without in the least knowing the
subject on which we are writing, for here again, the
words are so familiar that we transcribe them
with our own handwriting, using only our words writing
centre and the thenceon depending movements
of the hand without the intermediary use of
other centres.
These short cuts are established within certain
limits according to the education of the individual
& from the explanation for the variation in severity
of symptoms found in patients suffering from
similar anatomical lesions. In two Cases of
motor aphasia involving Broca's convolution, one
patient may be able to read moderately well &
express himself in writing, another may only be
able to read & write his name.
According to this interpretation of symptoms, it is possible to lose or retain a series of partial memories according to the brain affecting the path by which ideas are impressed or expressed; hence anamnesia cannot be admitted as a definite subdivision of aphasia. A person, for instance, who is suffering from word deafness has lost the memory for spoken words that is not amnesic, as he retains the memory for written language—again word-blindness by no means implies amnesia. The memory for spoken words may be perfect—amnesia, therefore, meaning the loss of memory of words, has been replaced, more scientifically, by a loss of memory for spoken words and a loss of memory for written words.

According to Charles, the different sensory paths by which we receive impressions, one assumes a predominance; in some persons it is the auditory centre, in others the visual centre, in others again it may be the tactile centre etc., so that in the first case a person relies principally on his word hearing centre in remembering a word & hears it reproduced, in the second case he will invoke the visual image; in each case, shows that centre be damaged on which they almost entirely depend. Amnesia would remain, but the pathological lesion would be different.

He, on this principle, divides persons according to the centre, which they use most in invoking the idea of words or objects into:

1. Those within the word hearing centre predominately word seeing
3. Those in whom the writing centre predominated
4. Motor articulating centre
5. Those in whom there is no predominant centre but therefore use them indifferently.

In this way he ingeniously accounts for the marked variation in the varieties of the symptoms met in patients suffering from the similar lesions, to which we have alluded above. For, if the predominant centre be injured, the other centres will not have been exercised enough to take its place; having lost this initiative force, in which they were incessantly brought into action, they remain inactive and useless, although the lesion itself has not involved them. If however a centre be injured which has played a subordinate part, although the lesion be comparatively speaking extensive, the patient can manage fairly well.

This theory, especially as regards the inclusion of a writing centre, has found opponents. These maintain that there are no grounds for believing that such a separate centre exists. Charcot showed in his lectures (December 1884) a patient who had lost the power of spontaneous writing, writing from dictation; but could read, copy, speak, understand what was said, as a case of pure Agraphia, but it was found that the case had started by Motor Aphasia with Hemiplegia, all the symptoms having disappeared except those mentioned. Pares' case (6) which was also supposed to be a case of pure & uncomplicated Agraphia resolved itself into a lesion situated between the word-arranging centre & the Motor centre for the right arm.

(6) Revue Medicale. 1884.
For he could write easily with his left hand—Marchi in 1856 held that there was a separate centre for writing, but in all the twelve cases collected by him there was aphasia present as well as agraphia. Eichhorn also advocates this special centre (1) Charron localizes it in the posterior half of the middle frontal convolution on the left side; its proximity to the motor centre for the upper limb does not increase the probability of its existence, considering how easy it is to educate almost any part of the body to write.

I may mention one more case bearing on the point: The patient was a lawyer’s clerk, who had been accustomed to write constantly with the left hand. According to Charcot’s theory, the writing centre would have acquired a certain predominance. Two years ago, he was seized with motor aphasia with hemiplegia; the latter has improved; the former is, however, complete—his left hand and left hand are unable to use the pen with his left hand. Cézere in his own handwriting accurately well, but spontaneous writing is reduced to signing his name. In other respects also Charcot appears to have gone too far in assigning equal rank to the various sensory centres.

In the development of ideas in a child and in adults, study the principal channels of impression. It strikes us that from the first the eye and the ear are the organs by which the child primarily is enabled to acquire ideas, and, as regards speech, the former is the more important. It may be urged that

(1) Mémoires de la Société de Biologie de Paris 1856, p. 98.
reading and writing are equally dependant on the eye, and under normal circumstances, no doubt, they are, but the fact remains, that the prime mover in the sequence is the ear. Herbert Spencer and C. Bastian hold that in the adult ideas are on the main revived in the process of thinking as auditory feelings. There is no doubt that the more concrete the idea, the more are we apt to dissociate it from its name and it is revived through the sensory path to which it appeals most strongly. In thinking of a landscape that we have seen, we see it as a picture before us, on the other hand, in thinking of a favourite dish the taste is revived, and maybe in remembering a flower, its odour is recalled. In abstract ideas, however, the name is essential to its revival in the brain, and we find clinically that in many cases of aphasia this is borne out, and that abstract qualities and relations of objects existing in our minds solely by reason of words are retained, whereas more concrete objects are lost.

Wundt, Bain, Hughlings Jackson and lately Stricker advocate that words are revived in silent thought, as faint articulatory processes taking place in "motor centres," i.e. by the agency of the memory for articulation of speech (process innovation). In the evolution of these various centres in childhood, we have, however, shown that the motor centre of articulation is entirely dependant on the word hearing centre. Indeed, unless this were true there is no explanation of the fact that if a child up
its fifth or sixth year, in full possession of speech, become totally deaf, dumbness is a necessary sequel until it be taught lip reading, in other words, unless the word seeing centres be made to take the place of the word hearing centres. The fact that many persons in the process of thinking mentally articulate the words, whisper them or even pronounce them aloud, does hardly point to the revival of ideas by articulating means, but rather that the thoughts are articulated to be more firmly impressed on the word hearing centres.

In most cases of aphasia of whatever variety, we find that the patient retains the memory of certain words with which he is intimately associated, in which he has frequently used in conversation. Even in the most pronounced cases, his name or part of it generally survives. Thus we have seen that a tuberculous aphasic, though totally unable to express his ideas in words, will repeat his name over and over again. Again in a word-deaf patient, we usually see him turning round if his name is pronounced, though he cannot understand anything else. In word-blindness the same happens in reading and writing. In all these cases those words which are retained vary in number according to the strain of the lesion and the education of the patient.

In the foregoing description I have refrained from attempting a description of the symptoms which occur in lesions involving the connecting fibres between the various centres. The modifications are endless, no two cases being exactly alike, whereas by setting forth in detail the growth development
+ function of the centres themselves. Their implication or exclusion can be diagnosed with certainty in any given clinical case.

Causes of Aphasia.

Considering that the middle meningeal arteries supply by far the greater part of the cerebral cortex and that third of the branches on the left side viz: The artery to Broca's convolution, the parieto-sphenoidal, and the sphenoidal supply more particularly the areas under discussion, it is not unnatural to find that vascular changes, followed by softening of the brain substance form the main causes. Of these again the most frequent is

1. Embolism. Hughlings Jackson was the first to point out the frequent occurrence of hemiplegia and aphasia in vascular endocarditis. The latter may be either simple or ulcerative. It has further been noticed to follow initial stenosis. It is recognized that the Sylvian artery is much more likely to suffer under such conditions than the other branches of the Carotid, partly owing to its size and partly owing to its direction.

2. Thrombosis due to disease arterial walls, as in atheroma, valvular aorta or Brights disease. In syphilitic endarteritis and aneurysm thrombosis may occur within the first three years of the requirement of the disease. A merely acting heart will naturally encourage this result.

3. Carotid Haemorrhages which in the great majority of cases is due to primary disease of the artery, such as aneurism, but may also be due to softening of the arterial wall round an embolus, or to inflammatory causes. This form is not frequent as regards the critical branches.
of the Sylvian sulci, but meningeal haemorrhage, if situated over the zone of speech produces the same effects.

In either case cerebral softening is the result of the injury being considerable.

4. Tumours of the brain may affect the speech centres, causing functional derangements through pressure or producing softening of the brain tissue in their neighborhood. The chief forms are gliomata, in which there is further danger from haemorrhages, which is apt to occur in their substance—gummata, which are the most amenable to treatment, and tuberculous nodules.

5. Traumatism followed by destruction of tissue.

6. Tubercular meningitis, which according to Fagge, is frequently found to affect the pia mater as it dips into the Sylvian fissure, producing Hemiplegia or maybe aphasia.

Such are the chief causes of true aphasia due to a destructive cerebral lesion. Many cases of hemiplegic or Transitory aphasia are met with, in which there is a temporary functional aberrance of the centres under consideration—although not falling under our definition, I propose to name these conditions under which they occur partly as an aid to differential diagnosis, but more especially

(1) Prof. Brangier Stewart makes cortical haemorrhages rank sixth in order of frequency as compared to other parts of the brain; meningeal haemorrhage (including Traumatic causes) being the most frequent—v. Lect. 1889/90.

(2) V. Launay 1887 "aphasia", case of W. Turner.
as they may be forerunners of the more serious form. Transitory aphasia may be due to

1. Certain emotional conditions, such as fear, anger etc., in which case it is probably caused by a passing anemia or congestion of the brain.

2. Toxic conditions, as in the course of certain fevers especially Typhoid in children and in Smallpox. A serious case has been recorded lasting 24 hours due to Scurvy. Troussseau mentions some cases which he attributed to Diabetes and Albuminuria, the latter is also considered as a possible cause by Hughlings Jackson.

3. Although rare cases have been recorded in the course of certain nervous diseases, e.g. Epilepsy, General Paralysis and Migraine. It is more important in connection with Hysteria, for this form of mutism may last a considerable time and may be misleading; the diagnosis must rest on the presence of other hysterical symptoms and on the careful examination of the patient's cultural functions such as reading and writing, in which case inconsistencies will be probably detected.

4. Injury is a common cause, either resulting in concussion or a slight haemorrhage.

5. The most important cases of this variety are those which are frequently preceded an attack of true aphasia. These occur chiefly in the young or syphilitic patients, and also in the rheumatic temperament.

(1) v. LaFRANCE MEDICAL 1884 Vol II p 1485 also Troussseau A. Lett 1875 p 263
(2) v. Lancer October 11th 1884
(4) "Mrs Turner Case v. Lancer 1887 upon aphasia", also Lancer April 26 1893
In such patients changes are going on silently tending to diminish the calibers of the cerebral vessels, and such a patient may have repeated attacks of transient hemiparesis and aphasia owing to bad circulation, persisting long enough to produce a functional disturbance, until finally the artery becomes too small to admit of the necessary blood supply (aided maybe by a thrombus) and he is seized with permanent symptoms due to softening and destruction of the brain tissue. Such cases are comparatively frequent.

Prognosis.

In a case of true aphasia, one in which the symptoms have persisted long enough to exclude temporary disturbances and in which, therefore destruction of tissue has occurred; the chances of recovering lost faculties will depend on the site and extent of the lesion and also on the capacity of other special cells for discharging these functions. In order to do so these cells must be induced; hence the point that the age of the patient is an important point. That a destructive lesion in any of the principal speech centres is ominous, as regards recovery of function, is undeniable. Transcerebral even pronounced motor aphasia, when accompanied by Hemiplegia is generally incurable, and consists of the frequent fatal termination from Apoplexy. But exceptions occur especially in the cortical motor form, the speech gradually improving. In these cases, the 3rd frontal convolution on the right side becomes the
Substitute for the originally trained centre. That the cells in this region are on either side susceptible to development is well seen in the case of left-handed persons, in whom the symptoms in question, with or without Hemiplegia, follow on a lesion in the right frontal lobe in that area. Wernicke reports a case with right hemiplegia and motor aphasia in which the condition improved. The patient however suffered from a second attack, in which he again lost his speech; in the post mortem examination the third frontal convolution was destroyed on both sides. A further indication that the reacquisition of speech comes about in this way is furnished by an old soldier, aged 45, whom I had the opportunity of examining. Three years ago he was seized with motor aphasia and right hemiplegia; at the present time his right leg is quite useless and his arm contracted, he has however gradually learnt to speak and does so fairly fluently. To this besides scanning slightly he speaks with a marked accent (resembling a kerman speaking French). He is however a Parisian before his illness & speaks like a native. This fact points to the radical change in the elements subserving speech and that, as far as the lesion in Broca's convolution is concerned, there has been a cure by substitution. In support of this view we may add that to our knowledge, there is no case on record of a child, let us say between 5 & 7 years of age, hemiplegic hemiplegic with Aphasia, the adaptability being so marked at that early age that the right side immediately takes the place of the left hemisphere.

Age is therefore a great factor in prognosis, the younger the patient (under 40), the better is his chance. This
practically, in only guide (apart from general health), if the symptoms have existed long enough to warrant the assumption of a destructive lesion. In middle age, one can hardly give a definite opinion for we have seen cases of syphilitic origin, which have been in the same condition over twice their attack 8 or 10 years ago. The soldier, whose case is noted above, on the other hand showed no signs of improvement for nearly two years. In both cases anti-syphilitic treatment was carried out. Another case that of a man aged 53 who was seized with motor aphasia right hemiplegia, he has been in this condition for two years. The hemiplegia is hardly changed. The arm however being more useless than the leg, as generally happens in these cases, he has, however, gradually improved in other respects, for he can express himself very well through his speech is slow and scanning. He has taught himself to write with his left hand, he has improved in this respect pari passu with his speech.

In all the cases we have seen, where the left hand was trained to write, the writing was not reversed, but this does occasionally occur.

In cases of sensory aphasia the prognosis is even less favourable; the patient may recover at all or improve, but as a rule the symptoms persist to a greater or less extent. From this we must conclude that the corresponding elements on the opposite hemisphere are provided with very little adaptability in the adult. Cases have been recorded in very young children in which total sensory aphasia is followed by the right hemisphere undertaking the lost functions.
Treatment: From the nature of the lesion it is obvious that drugs can do nothing locally, for in a well established case recovery is only by expectation in regeneration of tissue or increased physiological activity of certain cells. Therapeutics are however very useful in averting the catastrophe when we recognize the danger in time. It is for this reason that I have alluded to transient aphasia in this paper, as such a symptom is a true danger signal in certain cases, especially in syphilitic + guilty patients, in these, prompt constitutional treatment will often save the patient from the more permanent lesion when the symptoms have fully declared themselves in a persistent form. We can only indirectly improve the condition of the patient by rest, good nursing, and general treatment. If hemiplegia be present massage should be employed + after a few weeks the gentle application of Electricity will restore any diminished sensibility and will check wasting. In cases which can be traced to Syphilis or Venereal, the appropriate treatment should be persevered in, were it only to avert further mischief. The patient should also be made to aid physiological regeneration by mental exercise.

Lastly, operative interference is indicated especially where pressure is the primary cause, as in cases of tumours, blood clot, pus or a depressed fracture of the skull.

J. Egerton Grant.