THE VARIETIES OF APHASIA,

With special reference to the localisation of the centres concerned in the function of Speech.

Being a Thesis for the Degree of M.D.,
University of Edinburgh.

by

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M.B.,Ch.B.
1903.

April, 1907.

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INTRODUCTION.

In this paper I do not propose to take up a full consideration of this important and complicated subject. What I shall endeavour to do is to make a study of parts of it under the following headings.

(1) Historical Introduction.

(2) Short notes on the physiology of the mental processes concerned in the reception and production of Speech.

(3) A consideration of the methods at our disposal in attempting to determine the more exact localisation of the various centres for Speech.

(4) The localisation of the centre for the reception of spoken speech.

(5) The localisation of the centre for the reception of written or printed speech.

(6) The localisation of the centre for the production of spoken speech.

(7) The views of Professor Pierre Marie regarding Aphasia.

(8) A criticism of Marie's opinion.

(9) The localisation of the centre of the production of written speech.

(10) Short notes regarding the localisation of the centres for the musical faculty and for gesture language.

(1) HISTORICAL INTRODUCTION.

The subject of Aphasia is one to which a large amount of study has been devoted for many years. Aphasia is the term introduced by Trousseau to denote the inability to express thought by means of speech which is produced by certain diseases of the brain.

In the year 1841 Professor Lordat used the term Alalia to denote this affection; and in 1861 Broca, whose name is so closely connected with the subject changed this name for that of Aphemia.

Since Trousseau's work on the subject, however, the word Aphasia derived from a, privative and φασις speech, has been universally accepted as the general term to denote this affection.

Previous to this, however, the subject had attracted the attention of thinking men.

Schenkins[1] who lived at the end of the 16th century, noticed that, in some cerebral affections, although the tongue was not in the least paralysed, the patients could not speak, because they had lost their memory.

Up to the year 1820 no attempt was made to specify the part of the brain injury to which might cause loss of speech. Shortly after that time, however, Gall suggested that the faculty of Articulate Language was located in the anterior lobes of

the brain.

In 1825 Bouillaud[2] who had studied Gall's theories stated that "the anterior lobes of the brain are the organs for the formation and recollection of words, or the principal signs which represent our ideas" and he also further affirms that "the anterior portion of the brain is the organ of articulate language".

Up to this time, therefore, very little work had been done towards endeavouring to localise the sites of the lesion or lesions responsible for the disturbance of articulate language.

In the year 1836 Dr Marc Dax noted the fact that when there was loss of the memory of words, the lesion was always situated on the left side of the brain; and that he had never met with this affection in cases of cerebral disease exclusively limited to the right hemisphere.

G. Dax, the son of the latter also studied the subject and held that the centre for language was situated at the junction of the left middle and frontal lobes. The greatest advance in the subject was however made in 1861 when Broca[3] published his classical observations upon two cases of Motor Aphasia which he had under his care in the Bicetre Hospital. In the first of these two cases the lesion was situated in the left frontal lobe, and

in the second one[4] the lesion was limited to the posterior third of the second and third left frontal convolutions. Broca in consequence claimed that the posterior portion of the third left frontal convolution was the centre for the faculty of speech.

Thus we have first Bouillaud[2] who considered that the organ of the manifestation of thought by speech was situated in the two anterior lobes of the brain, then Marc Dax who located it in the left hemisphere exclusively, G. Dax who placed it at the point of union of the middle with the frontal lobe of the left hemisphere, and finally Broca, who stated that the seat of the faculty of speech was situated in the posterior portion of the 3rd left frontal convolution.

Subsequent to this, Charcot showed numerous cases in support of Broca's theory of Aphasia in all of which there was destruction of the third left frontal convolution. Later, however, he showed a case of Aphasia in which the third left frontal convolution was intact.

Broca assisted Charcot at the post-mortem examination of this case and recognised that it did not conform to the results of his observations. From this time onwards numerous cases of disturbance of speech were published. Dr James Russell[5] of

Birmingham in a series of papers on Aphasia pointed out the distinction between a loss of speech due to a mechanical difficulty in articulation and a loss of speech due to loss of memory of words, although as Professor Wyllie points out this point had already been made clear by Lordat.

In 1867 Professor Gairdner wrote a paper distinguishing between the Ideation and Innervation of Language.

Ogle, who was the first to use the term Agraphia for the loss of the power of writing, Bastian, Hughlings-Jackson, Broadbent and other observers published many cases with the results of post-mortem examinations.

It was reserved for Wernicke[6], however, in the year 1874, to show clearly that cases of Aphasia could be divided into two distinct classes.

(1) Motor Aphasia:

Characterised by inability to speak, although the patient understood what was said to him.

(2) Sensory Aphasia:

In which the patient was able to speak but did not understand. He pointed out that whilst the former was produced by a lesion of the posterior part of the third left frontal convolution the latter was due to a lesion in the left tempero-

sphenoidal lobe. He also described the variety of Aphasia that results from lesion of the white conducting fibres connecting the sensory with the motor speech centre. A great advance in the subject of Cerebral Physiology was made in 1870 by the work of Fritz and Hitzig and also in 1873 by Ferrier.(7)

Since that time much work has been done on this subject by Horsley, Semon, Franck and others.

In 1877 Kussmaul wrote his comprehensive treatise on the "Disturbances of Speech". He elaborated the views of Wernicke and described the varieties of Aphasia known as word-blindness and word-deafness.

In 1885 Lichtheim's(8) paper on the subject was published and in later years much work has been done on the subject by Dejerine(9), Serieux and Henschen.

In 1894 Professor Wyllie published his exhaustive work on "The Disorders of Speech" in which he gives the fullest and most scientific description that has so far been published of the different varieties of Aphasia. The views expressed by Professor Wyllie have been generally accepted as correct. Within the last few months, however, Professor Pierre Marie of the Bicetre Hospital, Paris has published several papers(10) on Aphasia. In these articles he expresses opinions which are directly opposed in many respects to the generally

accepted views on the subject. Coming from such a careful observer his statements are worthy of careful consideration and therefore in a later part of this paper I shall take up a consideration and criticism of his views regarding the subject.

Notes on the Physiology of the Mental processes concerned in the Reception and Production of Speech.

The interchange of thought consists of an exceedingly complicated series of processes. In the first place it involves the expression of mental processes by means of certain conventional symbols, and in the second place it involves the interpretation of these symbols. There are three chief methods by means of which thought can be communicated, namely by:-

(1) Speech. (2) Writing. (3) Gestures.

In addition to these there are certain other channels through which the brain may receive and interpret outside impressions. Examples of these are to be found in the use of the Muscular sense by means of which the blind are enabled to read; and in the case of the deaf by the interpretation of

the movements required for the articulation of words.

This interchange of thought from one person to another may be interrupted in the two following ways.

First, there may be a derangement of the channels by means of which symbols are interpreted as thought: or

Second, there may be an interference with the channels through which thought is translated into symbols.

In other words Aphasia may be divided into two great varieties:-

First, Sensory Aphasia.

Second, Motor Aphasia,

and further these types may affect one or more of the three chief classes of symbols previously mentioned.

I do not intend in this paper to take up in detail the consideration of the development of Intelligent Speech by the child; but I may refer to the fact that before this faculty is acquired by the child it has other ways of receiving outside impressions and of expressing its feelings and emotions. This is accomplished by means of the language of Gesture, by variations in the tone of voice used in speaking to the child and by facial expression.

The first thing a child probably understands is the meaning of one or other of these ways of communicating thought. Thus it is able to interpret the meaning of the tone in which words are spoken long before it is able to understand the meaning of the words. The production of one or other of those methods for expressing thought is the next step in the child's progress. Such steps are seen in the stretching out of the hands, grasping with the finger or the shaking of the head as an indication of negation.

With regard to speech proper it will therefore readily be understood that the reception and interpretation of Spoken Speech is the first step in the education of the child. It is natural that words should be understood before they are able to be intelligently produced, and further it is also natural that throughout the whole period of the process of learning to speak the understanding of what is spoken is always in advance of the power of producing spoken words. The ears are, therefore, the organs by means of which the child gets its first lesson in speech.

As mentioned above, however, it is able to understand the tone and manner in which words are spoken before it is able to understand the meaning of the words spoken.

The child learns from frequent repetition of a certain word along with its associations the meaning of that word. It is enabled to do this by means of the fact that the sound images of spoken words are stored in certain cells of the cerebral cortex so that they are capable of being called up for future use either by a stimulus coming from without ot by the revival of such sound image from within.

The next step in the progress of the child is for him to accomplish the power of producing words himself. This has been gradually led up to by the use of baby language in the form of Babbling, Crowing and Mimic Reading.(11) Soon, however, the child by imitation of sounds heard, assisted by imitation of movements which it sees produces sounds - thus making use of the visual route in addition to the auditory - is enabled to repeat these mechanically without at first attempting to understand them.

It is following on this that the next advance is made. As Professor Wyllie says: "The child has already learned to understand the meaning of a few words and perhaps has reproduced some of these by Echolalia; but now for the first time he begins to produce these words, not as mere echoes of sounds, but as words with definite signification;

on being asked to pronounce a certain word, he attempts to do so by a distinct effort of his will. These are the real beginnings of speech production". "A new agent, namely, the higher Intellectual Consciousness is thus beginning to interfere with the operations of the speech centres. At this early stage, however, it has not yet acquired complete control; and it can get the speech-producing centres to produce the simplest words only after many futile attempts and much persevering practice."

We now have to look briefly at the way in which Written and printed speech is received and reproduced by the child.

This accomplishment of course takes place at a considerably later stage in the child's life and requires an elaborate process of further education. We have seen that spoken speech is acquired primarily by means of hearing. Written and printed speech is acquired chiefly by means of sight.

The child learns that a certain symbol represents a certain sound by having the symbol interpreted to him by means of spoken speech. He next learns to unite these individual signs so as to produce words, and just as in the case of spoken speech the sensory impressions of these signs are gradually received and stored up in special cortical

cells devoted to this purpose. As in spoken speech the next step is for the child to acquire the faculty of producing written speech. This is accomplished by a similar process. The difference being that the child learns to produce the symbol which represents a particular meaning to him instead of producing the sound for that particular meaning. This resolves itself of course into a question of copying the form of letters. At first this is accomplished laboriously but with practice the child can with increasing facility call up the memory of the muscular movements required until the writing can be done easily and proficiently.

It follows, therefore, from a consideration of the mechanism of speech that there are four chief centres employed in forming one's idea of words.

As we have seen two of these are sensory, namely, the Auditory word centre by means of which a word can be recognised from its sound; and the Visual word centre, by which a word can be comprehended from its appearance in writing or printing.

To make use of these two sensory memories there are two motor centres, namely, a centre for the production of the associated movements requisite for articulation, and a centre for the co-ordination of the muscles used in writing.

A consideration of the methods at our disposal in attempting to determine the more exact localisation of the various cortical centres concerned in the reception and production of Speech.

In the study of this subject there are three chief lines of investigation which we can make use of.

These are respectively -

(1) Experimental Physiology.

(2) Clinico-pathological observation.

(3) Histological and Embryological research.

It is only, I think, by a careful consideration of the results obtained in these different departments of research that we can hope to finally accomplish the difficult task of definitely settling the functional sub-division of the cerebral cortex.

Experimental Physiology:

This method of research has done a great deal in advancing our knowledge of the sub-division of the motor area of the cerebral cortex.

Mention must be made of the work on this subject by Ferrier, Schafer, Horsley, Beevor, Sherrington, Mott and Ballance in this country and that of Hitzig, Goltz, Munk and Bechterew on the continent.

The animals used in their experiments have for the greater part been monkeys. These animals though not endowed with the power of speech, possess, however, a language of their own by means of which they can to a certain extent communicate thought to each other, and in doing so they make use of the same mechanism as man employs for the purpose of articulate speech, namely, the vocal and oral articulative mechanisms.

Clinico-pathological observation:

With regard to this method I need only refer to the enormous amount of work that has been done on the subject since the time of Broca's publication of his original cases.

By the comparison of clinical facts with the results of post-mortem examinations our knowledge of the sites of the various lesions responsible for the clinical varieties of Aphasia has reached a degree of accuracy that is in many respects highly satisfactory.

Lastly we have the aid of -

Histological and Embryological Research in the elucidation of this difficult subject.

This method offers a large field for further research. Valuable work has been done on this subject from the Embryological point of view by

Flechsig. In 1872 Meynert published his classical research on the nerve cell. Since that time Bevan Lewis, Henry Clarke and other observers have gradually added to our knowledge of the subject. In 1905 Campbell[12] published the results of his researches on the histology of the cerebral cortex. His conclusions are exceedingly interesting and furnish an additional aid to the efforts being made at exact scientific functional sub-division of the cerebral cortex.

In his work on the Localisation of Cerebral Function, Campbell notes three main directions in which the histological study of the brain can be carried out. These are

(1) The study of the Brain during development.
(2) The study of the Brain in conditions of disease.
(3) The study of the Brain in the normal state.

As an illustration of the value of histological work in the study of the Brain during development it may be noted that the calcarine cortex, which is the centre for sight, is found to contain myelinised fibres at a relatively early date.

These are present before similar fibres show themselves in the surrounding field.

By this means its surface distribution is readily determined, and as the fibres with connect this

area with inter-mediate visual stations become myelinated at a corresponding time, their course can in consequence be also readily followed.

He suggests that further work should be done with regard to the time that the nerve cells in the cortex make their appearance from the assumption that these follow the same sequence as the nerve fibres. For example, he says that "There is full likelihood that in the case of those animals which at the time of birth are incapable of locomotion, man is of course one, sensory cells develop in advance of motor cells, and in this way light might be thrown on at all events one very vexed question." As yet, however, owing to our defective methods no work has been done on this subject.

In the histological study of the Brain in conditions of disease, Campbell makes use of two important methods by which cerebral localisation may be forwarded.

First; By studying the course and destination of the secondary degenerations, produced either experimentally or in the course of nature. By this means information can be obtained concerning the motor areas, sub-cortical association tracts, and also the various sensory areas.

His second method is based on the principle that division of a nerve is followed not only by central, but by peripheral changes. These changes not being limited to the divided segment but affecting all the links and stations in the neuronic chain of which it forms a part. This principle, as Campbell says, embodies Wallerian degeneration and Guddens atrophy. He points out that Bolton has made use of old-standing cases of blindness of peripheral origin in defining the exact limits of the visual area, and he himself in his work on the subject gives a most detailed account of the changes produced in the motor area of the cortex as a result of the amputation of one or other of the extremities; and of the histological changes produced in the sensory area of the cortex in cases of Tabes Dorsalis.

The results obtained by these methods are highly interesting reviewed in the light of the results of clinical and pathological observation.

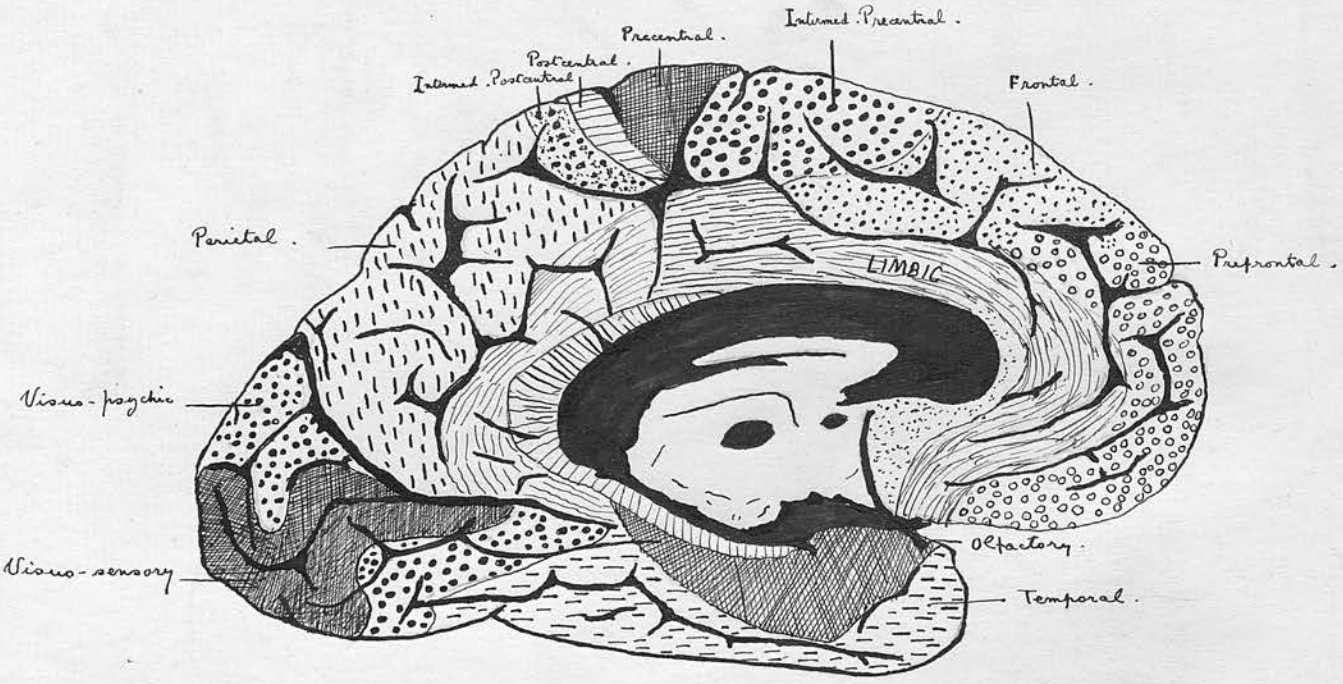
More work, however, is needed on this subject. Thus the study of the brain in old-standing cases of deafness, or in cases of loss of the senses of Smell, and Taste due to peripheral lesions - such as in the latter case is produced by excision of the tongue - would be of great interest.

Lastly, Campbell has devoted an enormous amount of work to the study of the Cerebral Cortex in the normal condition.

He has published the results of his work regarding the variations in size, arrangement and number of the nerve fibres in the different regions of the normal cortex and also though to a lesser extent of the arrangement and characteristics of the nerve cells.

His investigations have been carried out"with the end expressly and constantly in view of endeavouring to ascertain whether or not fields having established connections with different physiological functions are characterised by the possession of some specific fibre arrangement which will render their correct delineation possible."

He has made a collateral comparison of cell lamination and fibre arrangement in section after section and millimetre by millimetre over the entire surface of the human cerebrum. The result is that by these means he has been enabled to construct a sub-divisional map of the human cerebral cortex showing the boundaries of areas which possess a certain definite arrangement of their nerve fibres and nerve cells, as distinct from other areas which show a different arrangement of these structures. The position and limitations of these areas as defined



Drawing showing distribution of the various types of cortex as mapped out by histological research (after Campbell).

by Campbell, in so far as they affect the question of the localisation of the speech centres will be considered in the following parts of this paper and compared with the current views regarding the localisation of the speech centres derived from clinical and pathological data.

I will now proceed to take up the question of the Localisation of the Centres concerned in the reception and production of Spoken and Written or Printed Speech in the light of these three methods of investigation.

1st. The localisation of the centre for the reception of spoken speech.

In studying this subject we have in the first place to consider the localisation of the centre for the reception of Ordinary Auditory Stimuli.

(1) The Experimental evidence:

Munk in his experimental work showed that bilateral extirpation of the temporal lobes in dogs was followed by "complete cortical deafness".

Ferrier demonstrated the fact that in the case of the monkey the centre for auditory perception was situated in the 1st temporo-sphenoidal convolution.

Electrical stimulation of this area caused the animal to behave as if it had heard a sound in the opposite ear. He further showed that destruction of this convolution on both sides caused total deafness.

Schafer, however, repeated Ferrier's experiments on six monkeys more or less completely destroying the superior temporal gyrus on both sides and yet in not a single instance was hearing permanently affected.

This is contradictory evidence. (A possible explanation for this will be given later, however).

Munk in further experiments found that the removal of a small piece of cortex from the centre of the auditory area in the dog produced the condition known as "mind-blindness".

Larionow found that slight lesions of the auditory cortex gave rise to a loss of appreciation of single tones without any disturbance of common hearing. Reviewed as a whole the experimental evidence tends to prove that removal of the superior temporal convolutions on both sides causes total deafness.

The Clinico-pathological evidence.

There is a well known case reported by Friedlander and Wernicke in which the posterior part of the 1st left temporal convolution and at a later

date the posterior part of the 1st right temporal convolution, the whole of the supra-marginal and the adjacent parts of the angular gyrus were destroyed by a gummatous lesion.

The result of the lesion on the left side was that the patient in addition to being rendered hemiplegic also showed the symptoms of word-deafness, Aphasia and Paraphasia. When the other side became affected the result was that the woman was rendered completely deaf.

Serieux and Mignot[13] report a case in which the presence of hydatid cysts in both temporal lobes gave rise to complete deafness.

Mills[14] records a case of total deafness in which the posterior two-thirds of the first temporal convolution on the left side were reduced to a thin strip, and the posterior fourth of the second temporal convolution partly destroyed by an old embolic softening; on the right side the first and second temporal convolutions, the insula and the lower end of the central convolutions, along with the lenticular nucleus and external capsule had been obliterated by an old haemorrhage.

In considering these cases one cannot fail to see that the extensive nature of the lesion in all the cases renders them less useful for our present purpose than would less extensive lesions.

Nevertheless, they supply strong additional evidence in support of the conclusion that the centres for the reception of ordinary auditory stimuli are situated in the superior, temporal convolutions on each side, more especially in their posterior half or three-quarters.

Let us look here shortly at the interesting question as to the bilateral representation of the ears in the cerebral cortex.

From a consideration of the above we should expect that destruction of one first temporal convolution would give rise to either

(1) Unilateral deafness on the same side.

(2) Unilateral deafness on the opposite side.

(3) Bilateral deafness corresponding to hemianopsia due to a semi-decussation of the auditory fibres, such as we know takes place in the case of the optic fibres.

The evidence on this point is very conflicting. There are cases on record in which a lesion of one temporal lobe has given rise to sudden dulling of the sharpness of hearing.

This, of course, supports the view that each ear is connected with both cortical centres. In opposition to this, however, Kaufmann(15) and Fergusson(16) have recorded cases of unilateral deafness due to lesions of the first temporal

convolution of the opposite side. On the whole, however, the weight of the evidence tends to support the view that each cortical auditory centre is connected with both ears and that, therefore, a similar arrangement exists in connection with the auditory route as is well known exists in the case of the visual apparatus.

We have now to consider if we are able to determine accurately which part or parts of the centres for the reception of ordinary auditory stimuli are specially connected with the reception and interpretation of spoken speech.

On this point we have abundant clinical evidence to prove that though both auditory centres, as we have seen, have stored in them the images of common sounds, only one of them, in all right handed people the left, is utilised for storing the sound images of words. The question now arises, are we able from a study of clinical cases supplemented by post-mortem examination, to differentiate that part of the auditory cortex concerned in this storing of the sound images of words from the other parts of the hearing centre on the left side.

In a typical case of this description the patient would exhibit the symptom known as word-deafness

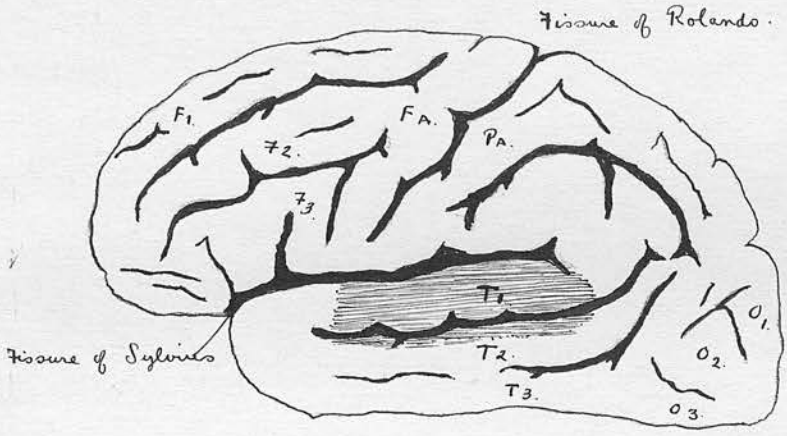
while the power of hearing and understanding other sounds would be intact.

The following case quoted by Professor Wyllie in his work on "The Disorders of Speech" is a good example of the value of clinico-pathological observation in the furtherance of our knowledge of this subject. The case was originally recorded by Giraudeau.(17)

A widow, aged 46, had suffered for three months from constant headache, and had become quite word-deaf. To all questions she invariably replied: "What is it you say?" "I don't understand you. Cure me!" Hearing and sight were intact. She read well and answered questions which were written and shown to her. She died comatose. "On autopsy, there was found a tumour, the size of a walnut, in the posterior part of the left temporal bone. Above, it reached the fissure of Sylvius; below, it involved three-quarters of the breadth of the second temporal convolution. It reached forward to within three centimetres of the anterior extremity of the temporal lobe, backward to within one centimetre of the posterior extremity of the fissure of Sylvius.

It encroached slightly on the white matter, and was easily enucleated."

It is comparatively seldom that one has the opportunity of obtaining a case in which the lesion



Drawing to show the localisation of the word-hearing centre. (after Wyllie.)

is so comparatively limited. A consideration of other cases of a similar nature, however, confirms the view that so far as we can discover from clinico-pathological investigation the centre for storing the sound images of words is situated in the posterior half or three-quarters of the first left temporal convolution and probably extends into the second temporal convolution. The accompanying diagram shows the area in question.

The histological evidence.

Lastly we come to consider what help we can get in endeavouring to localise the centre for hearing and the part of it concerned in the storing of auditory word images from a study of the histology of the temporal lobe.

The results obtained by histological research are on the whole strongly corroborative of the conclusions as to the localisation of these centres that we have come to from a consideration of the experimental and clinico-pathological evidence. In connection with this part of the subject, I shall have to refer largely to the work of Campbell. Campbell finds from an exhaustive study of the arrangement of the nerve cells and nerve fibres in the temporal lobe that three distinct types of cortex may be mapped out. Without going into the full details of the histological structure we have

to note that the first type is characterised by a specially dense arrangement of the nerve fibres. The position of the area covered by this type is as follows.

Generally speaking it lies on the superior temporal convolution within the Sylvian fissure and is therefore concealed from view.

Anteriorly it reaches to the line of junction between the most anterior of the transverse temporal gyri, or the Gyri of Heschl as they are termed and the deeper lying surface of the insula proper.

These transverse gyri lie on that portion of the superior temporal convolution which is concealed within the fissure of Sylvius.

They, therefore, look upwards and inwards towards the insula. Sometimes as many as five distinct gyri may be made out but the most constant and best developed is the anterior one.

This gyrus rises from the superior temporal convolution about the middle of the fissure of Sylvius, and the sulcus which divides it from the succeeding transverse gyrus behind is often very deep and may appear on the lateral surface, where it may even bisect the superior temporal convolution.

This arrangement is found to be more frequent in the left hemisphere than in the right and in males than females.

Laterally this type of cortex begins to appear on the surface at the point where the anterior gyrus of Heschl springs from the superior temporal gyrus and behind this the Sylvian lip of the posterior half of the first temporal gyrus may be regarded as a boundary. In some brains this dense fibre arrangement is completely concealed, while in others it is found extending over the lip of the fissure on to the free surface of the first temporal convolution.

Posteriorly the area is again hidden from view and this type of cortex is only found on the walls of the posterior forked extremity of the fissure of Sylvius.

Towards the mesial part of the brain the area comes to a point where the transverse temporal gyri converge, and it is interesting to observe that the arrangement does not show any indication of ascending the parietal operculum.

The second type of cortex in the temporal lobe as described by Campbell has the following characteristics and distribution.

This type shows an intermediate degree of nerve fibre wealth. It forms a broad margin to the previous mentioned area and is almost entirely confined to the first temporal convolution.

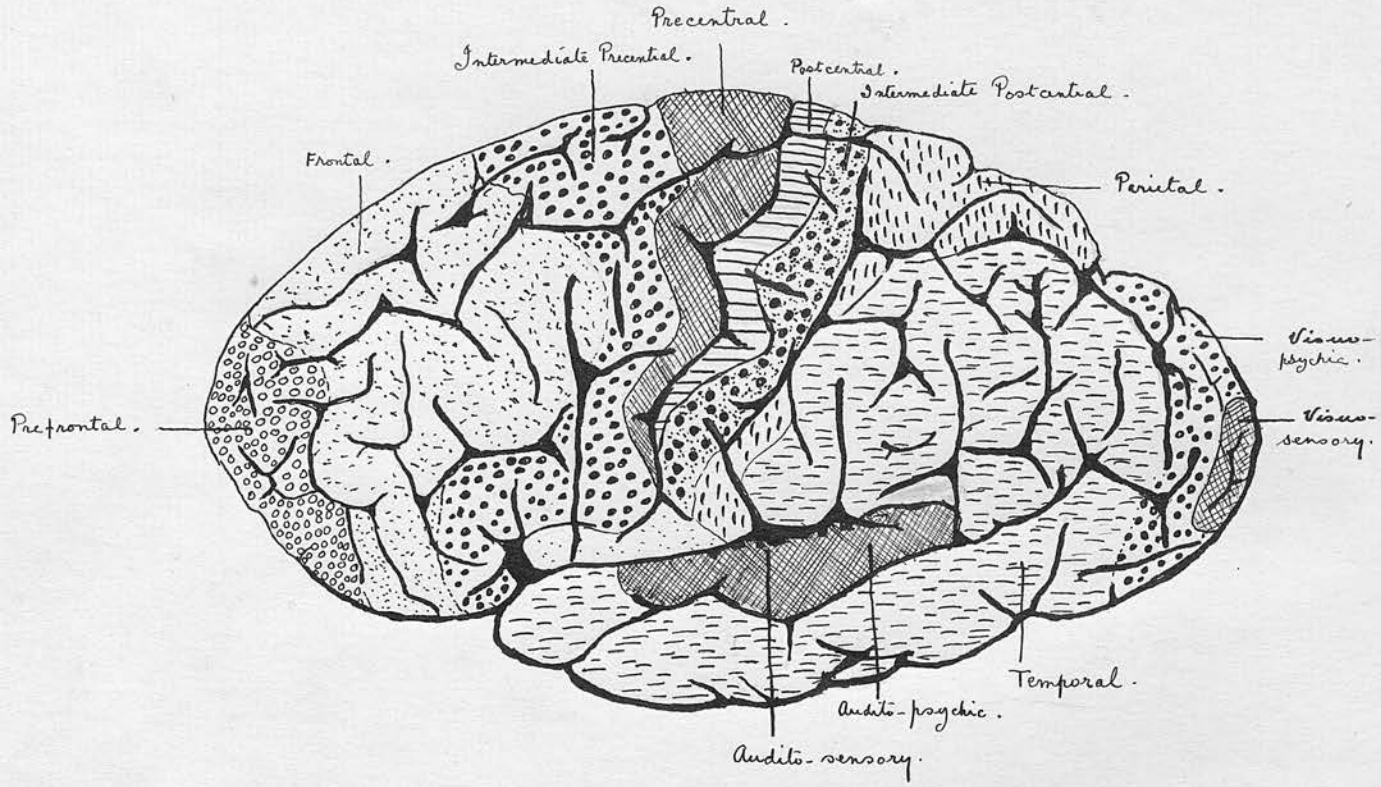
Anteriorly the limit of this area corresponds to the point where an imaginary line drawn from the inferior extremity of the fissure of Rolando would cross the temporal lobe.

Inferiorly the first temporal sulcus is the boundary. Transverse sections show that the intermediate arrangement does not extend more than about half way down the upper wall of the sulcus.

Posteriorly the first temporal sulcus is again the boundary. Campbell says that in this position the area is subject to slight variations, one of which is an extension on to the second temporal convolution. This is a point to which reference will have to be made later.

It may be noted here that as we shall see later the fibre arrangement in this area is homologous to that found on the outskirts of other regions such as the visuo-sensory area.

The third and last type of cortex which we have to take note of in the temporal lobe is mainly characterised by a marked diminution in the wealth of its nerve cells and nerve fibres. This type is found to cover almost the whole of the second and third temporal convolutions and much of the fourth temporal or fusiform gyrus.



Drawing to illustrate the distribution of the various histological areas of cortex. (After Campbell.)

On the inferior and mesial surface it does not cease until the collateral fissure is reached. The boundary here, however, is vague.

From a consideration of the above researches Campbell forms the opinion that the centre for the reception of simple auditory stimuli is the area of cortex possessing the first type of structural arrangement described above and located in the transverse temporal gyri.

His reasons for this conclusion are as follows: (1) "Microscopic examination proves that the area possesses a type of histological structure entirely different from that of any other part of the temporal lobe; not only so, the type of arrangement of medullated nerve fibres is peculiar inasmuch as the fibres of large calibre which are present in great abundance have the appearance which has been noted elsewhere as characteristic of corticipetal fibres; that is to say, instead of descending in the radiary fasciculi and striking the white substance more or less at a right angle, they seem to issue from the white substance at an acute angle and then cross obliquely for some distance in the radiary zone, as if seeking one of the large nerve cells which constitute another distinguishing feature of this area".

Flechsig's embryological work confirms this view. Let us look for a moment at the most recent idea concerning the auditory route from the organ of Corti to the cortex as brought out by the work of Flechsig, Monakow, Ferrier and Turner. From the organ of Corti the auditory impulses pass along the cochlear nerve to the ventral and dorsal cochlear nuclei. From this point some of the fibres pass by way of the striae acousticae or the striae medullares to the superior olivary bodies on both sides and to nuclei in the trapezoid body; other fibres proceed to the posterior corpora quadrigemina and the middle geniculate bodies.

Lastly, fibres leave these bodies to pass through the retro-lentiform portion of the internal capsule and make their way along the corona radiata to the temporal cortex. It is these latter fibres that we have to trace in the present instance. Flechsig states that they are divisible into two bundles, one of which ascends near the external capsule and gains the auditory cortex from the posterior and superior side, whilst the other passes for some distance in company with the occipito-tholamic radiations, and then passing behind and below the fossa Sylvii pierces the bases of the second and third temporal convolutions to gain the transverse temporal gyri.

Flechsig has further proved that the fibres from the middle geniculate body proceed to the anterior transverse temporal gyrus and as they become medullated at an earlier date than the fibres from the posterior quadrigeminal bodies, he believes that the same gyrus represents the cortical end - station of the cochlear nerve.

Flechsig states that the area to which these fibres traceable by embryological methods pass consists of the two transverse temporal gyri, particularly the anterior, and that part of the superior temporal convolution immediately adjacent.

We, therefore, come to the conclusion from a study of the histological and embryological evidence that the cortical centre for the reception of ordinary auditory stimuli is situated in the transverse temporal gyri. This it will be noted does not entirely agree with the conclusions we came to as a result of studying the experimental and clinical evidence. By this means we have limited this centre to the more restricted and definite area mentioned above instead of including roughly the whole of the superior temporal convolution.

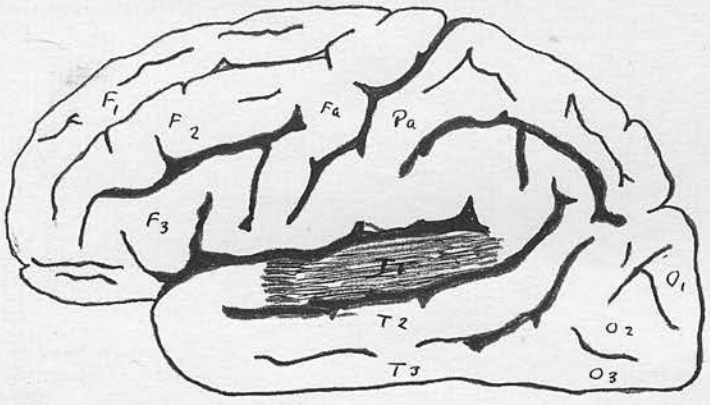
The localisation of the word-hearing Centre.

We are now in a position to proceed to the study of this important question. The histological evidence is of great interest in this connection.

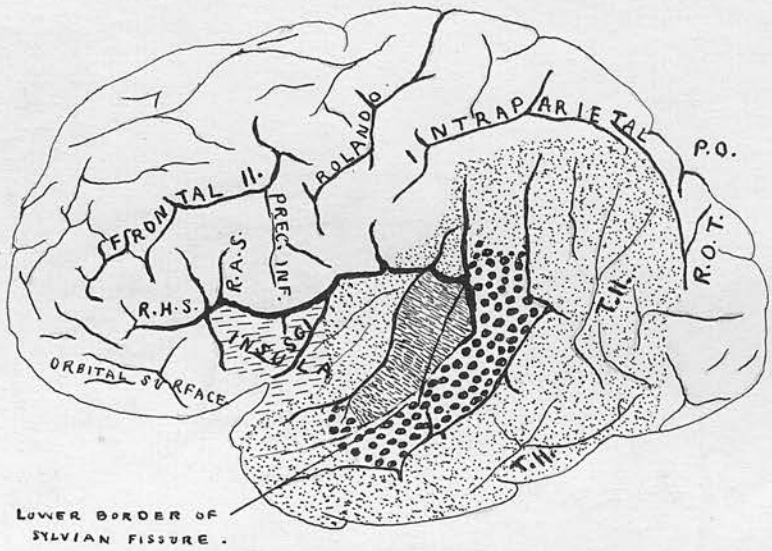
That such a centre exists is proved by the fact that there are many cases of word-deafness on record in which the faculty of hearing and interpreting ordinary sounds remained unimpaired.

Campbell suggests that the word-hearing centre is situated in that part of the temporal lobe which is covered by the type of cortex previously described as the second or intermediate type. We have seen that this specialised structural arrangement of the cortex formed a broad margin to the first type and that roughly speaking it was almost entirely confined to the first temporal convolution. The fact that this area possesses a type of cortex resembling but not identical with that found in the transverse temporal gyri, and one which is readily distinguishable from the other surrounding cortex, certainly suggests very strongly that it is concerned with a function closely allied to that of the transverse temporal gyri.

The question then is, does this area mapped out by histological observation correspond with the area which clinico-pathological observation has shown to be the part of the cortex lesion of which gives rise



The situation of the word hearing centre. as marked out by clinico - pathological examination.



Drawing of left cerebral hemisphere with Sylvian fissure opened out. Showing (1) the Audito-sensory area (shaded) confined to the two transverse temporal gyri and not extending on to the insula;

(2) the Audito-psychic area (large dots) on the free surface of the posterior three-fifths of the first temporal gyrus;

(3) the extent on the lateral surface of the hemisphere, of the common temporal cortex (small dots). (after Campbell.)

S.C.I. = sulcus centralis insulae;
R.H.S. = ramus horizontalis Sylvii;
R.A.S. = ramus ascendens Sylvii;
Prec. Inf. = sulcus precentralis inferior;
P.O. = parieto-occipital fissure;
R.O.T. = ramus occipitalis transversus;
T.II. = sulcus temporalis secundus:

to the symptom of word-deafness?

The answer is that the areas mapped out by these two methods do resemble each other very closely. It may be useful here to recall the boundaries of the lesion in the case of word-deafness previously referred to.

It will be remembered that in that case the patient though word-deaf, could, however, hear ordinary sounds perfectly well. Sight also was intact and she could read well and answer questions which were written and shown to her. The post-mortem showed the cause of the symptoms to have been a tumour which was situated as follows. Above it reached the fissure of Sylvius; below it involved three quarters of the breadth of the second temporal convolution. Anteriorly it reached to within three centimetres of the anterior extremity of the temporal lobe.

Posteriorly to within one centimetre of the posterior extremity of the fissure of Sylvius. It encroached slightly on the white matter.

From this it will be seen how closely the areas do correspond. I have made here two drawings illustrating the area which had been previously marked out from clinico-pathological observation as representing the word-hearing centre and the area as mapped out by the histological evidence of Campbell.

Lastly, I think it is very probable that this "audito-psychic" area is not merely a word hearing centre but the centre for the reception and elaboration of other specialised forms of auditory stimuli such as for example the various forms of musical sounds etc. A further sub-division of this centre may, therefore, eventually be brought about, and even the part of this area of cortex concerned in the reception and interpretation of spoken speech may itself be sub-divided into still more specialised sections.

S U M M A R Y .

The Experimental evidence shows that :-

(1) There is a centre for the reception of ordinary auditory stimuli in both hemispheres.

(2) That these centres are situated somewhere in the superior temporal convolutions.

(3) That if both of those convolutions are destroyed the animal is rendered totally deaf. (Schafer's negative results were probably due to the fact that he had not completely destroyed the transverse temporal gyri).

The Clinico-pathological evidence tends to prove that:-

(1) The centre for the reception of ordinary auditory stimuli is situated in the superior temporal convolutions on either side.

(2) The ears are bilaterally represented in the cortex.

(3) The centre for storing the sound - images of words is situated in the posterior half or three-quarters of the first temporal convolution on the left side.

The Histological evidence tends to prove that:-

(1) The centre for the reception of ordinary auditory stimuli is situated in the transverse temporal convolutions.

(2) The centre for storing the sound - images of words is situated in the area shown above which corresponds closely with that mapped out by the clinico-pathological evidence.

(3) It is also probable that this area of cortex in addition to having the function of storing the sound-images of words, is also concerned in the interpretation and elaboration of other varieties of auditory stimuli auch as those of musical sounds.

We now pass to consider the evidence regarding the localisation of the Centres for the Reception of Written and Printed Speech.

This is a part of the subject about which there has been a great deal of controversy, and there still is a divergence of opinion regarding the precise localisation of these centres. There is, however, a large amount of evidence to prove that in studying this subject there are, as in the case of the Auditory mechanism, two cortical centres to be reckoned with:-

(1) A centre for the reception of primary visual impressions.

(2) A centre, connected with the latter which has the function of interpreting and storing the visual images of words written and printed.

We shall consider first the localisation of the Centre for the Reception of primary visual impressions.

As before we shall look first at the results of:-

(1) Experimental Physiology:

Here we are faced with the difficulty that the experiments have necessarily to be carried out in the lower animals and that even in the most

intelligent of them it is practically impossible to be sure of the visual condition which exists after operation.

This shows that great care must be exercised in coming to a conclusion regarding this subject, as a result of experimental observation alone.

Munk, who was the first to attempt to determine this centre by experiments on dogs localised it in the outer convex part of the occipital lobe. Ferrier[18] on the other hand, disagreed with Munk's results and placed it in the angular gyrus. It is now believed, however, that in destroying the Angular gyrus Ferrier also interfered with important fibres, known as the occipito-thalamic fibres situated below this region and thus caused blindness. Schafer and Sanger Brown[19] removed both angular gyri without producing any effect on vision. With regard to the production of ocular movements from electrical stimulation of the cortex Sherrington and Grunbaum found that in experimenting on the higher apes electrical stimulation of the angular gyrus gave negative results and they further found that in the case of the occipital lobe stimulation of the cortex along the calcarine region and the extreme posterior apex was the only part which gave rise to ocular movements, and then only with difficulty.

Finally Munk and other experimenters found that destruction of both occipital lobes resulted in total blindness.

From a study of the experimental evidence, therefore, we can say that in the dog, monkey, and other animals there is an area of cortex in the occipital lobes removal of which is followed by blindness. An exact definition of the limits of this area is not possible by this means.

(2) The Clinico-pathological evidence:

It has been clearly shown by the observation of a great many cases that the primary visual centre is located in the occipital lobes. We must here note the fact, however, that the exact localisation of the centre for the reception of primary visual impressions, from a study of the results of lesions of the occipital lobe, is not calculated to give us very precise information for the following reason. Most of the cases studied in this connection have been cases of cerebral softening, and a consideration of the blood supply of the occipital lobe shows, that this part of the brain is supplied by branches derived from the occipital artery of Duret, which arises from the posterior cerebral artery. We find, however, that neither the distribution of the whole vessel nor any of its branches, corresponds exactly

with that part of the occipital cortex which, as we shall see, we have reason to believe is the cortical centre for vision. And further, when any of the branches of this artery are occluded damage is always done to important underlying tracts of fibres radiating to all parts of the occipital cortex. These we shall have to consider later. It will be recognised, therefore, that from the anatomical arrangements it is practically impossible to have a small definite lesion restricted to the cortex only.

Henschen of Upsala[20], however, from a study of forty cases of hemianopsia comes to the conclusion that a lesion of the cortex of the calcarine fissure, more especially of its anterior two-thirds, will suffice of itself to produce permanent hemianopsia, and that the remainder of the occipital cortex is of no consequence so far as the production of a visual defect is concerned.

Seguin[21] from an analysis of 40 cases locates the visual centre on the inner surface of the occipital lobe, but is is to be noted that in all his cases the lesion was in the neighbourhood of the calcarine fissure.

Vialet[22] from a consideration of eight cases gives the following as the boundaries of the visual area:- Anteriorly, the parieto-occipital fissure; above and behind, the margin of the hemisphere;

below, the lower border of the third occipital convolution.

Finally, other writers extend the area further forwards as far as the angular gyrus. For example, vonMonakow states that "the visual area occupies in addition to the entire cortex of the individual occipital gyri, at least the hinder part of the gyrus angularis".

From a consideration of the clinico-pathological evidence, therefore, we see that though opinions vary considerably, the bulk of the evidence shows that the centre for the reception of primary visual impressions is situated in the occipital lobe especially its posterior extremity, and its inner surface in the neighbourhood of the calcarine fissure.

There is, of course, a primary visual centre in each occipital lobe and each occipital lobe is connected with both eyes. Each primary visual centre, however, is not connected with the whole of the retina in each eye but only with the half. This is the explanation of the fact that destruction of one primary visual centre gives rise to blindness in half of the field of vision, the right half, or left half as the case may be or in technical language homonymous hemianopsia.

(3) Lastly we have to look at the histological and embryological evidence with regard to the localisation of the primary visual centre.

In the first place we have to note that in the cortex of the occipital lobe, in the region of the calcarine fissure, there exists a layer of nerve fibres capable of being seen by the naked eye when sections of this part of the cortex are made. This layer is known as the "line of Gennari". It is believed that the part of the cortex of the occipital lobe characterised by the possession of this lamina is the cortical centre for the reception of primary visual impressions. Campbell has shown that the cortex in this situation is of a distinct type, as shown by the specialised arrangement of its nerve fibres and cells. It would take up too much space here to give even a resume of the histological structure as detailed by Campbell, but the following is the distribution of this type of cortex called by Campbell "Visuo-sensory".

In the first place, however, as the distribution of this type of Cortex bears such an intimate relation to the calcarine fissure, it is well here to look for a moment at the anatomy of this important landmark. Cunningham divides the fissure into two portions, an anterior part or stem, which he would call the "fissura calcarina anterior", and a posterior

part of "fissura calcarina posterior". The fissura calcarine anterior, or stem, is that part of the fissure which lies anterior to the junction with the parieto-occipital fissure, and it differs from the posterior portion in being much deeper and in being a "complete" fissure. The stem is separated from the parieto-occipital fissure by the deep annectant gyrus.

A deep annectant gyrus is likewise interposed between the stem and the posterior portion of the calcarine fissure. This barrier may generally be found at a short distance behind the apex of the cuneus, and as it connects the cuneus and lingual lobule it has been named the anterior deep cuneo-lingual gyrus. The posterior calcarine fissure in addition to being shallower is also somewhat shorter than the anterior. It is also divided into two parts posteriorly by a deep annectant gyrus which traverses its floor and connects the cuneus with the posterior part of the gyrus lingualis. This is called the posterior deep cuneo-lingual gyrus.

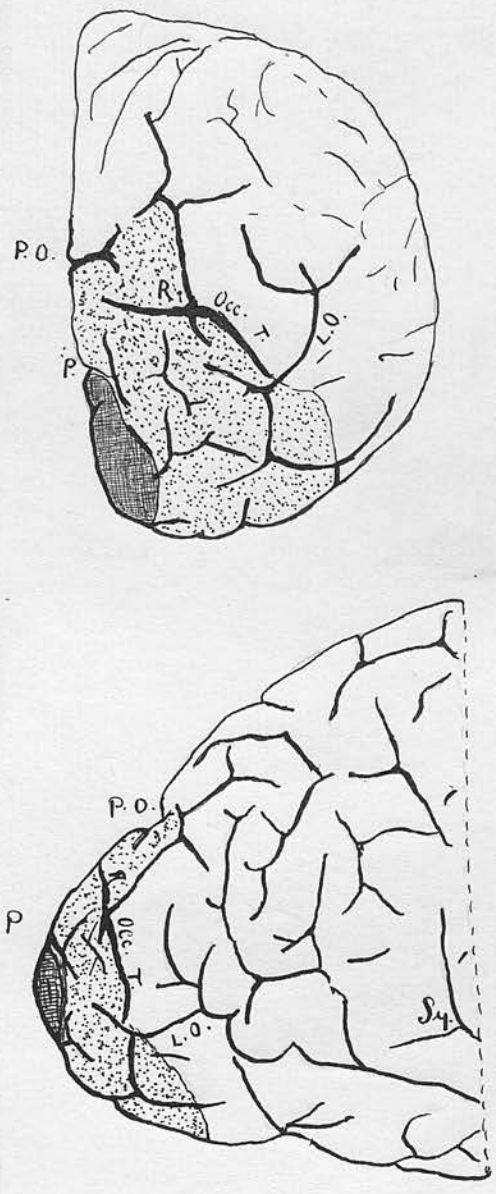
In following out what Campbell terms the Visuo-sebsory or Calcarine type of Cortex, we shall see how closely it follows the above described calcarine fissure.

By making sections at right angles to the fissure he finds that this type of cortex does not quite

reach the anterior extremity of the stem or anterior calcarine fissure but commences at a point from 5 to 10 m.m. further back. It first shows itself on the lower wall of the sulcus about half way down, and as we proceed backwards, it gradually spreads over the lip of the sulcus on to the lingual lobule, in the outward or surface direction, and down to the floor of the sulcus, in the downward or central direction.

Accordingly it is to be noted that the cortex covering the upper wall of the calcarine fissure does not show the peculiar lamination under consideration. And we have further to note that this type of cortex does not approach the parieto-occipital fissure.

As soon as the cuneus is reached, however, this type at once passes across the floor of the calcarine fissure and ascends the upper wall, and all along this hinder portion of the fissure the special lamination coats both its walls (including the anterior and posterior cuneo-lingual annectant gyri) it also spreads for a varying distance on to the free surface of the cuneus above, and of the lingual lobule below. On the cuneus the cuneal sulcus, a small fissure running parallel with the posterior calcarine fissure, commonly forms a boundary. As this is inconstant, however, it must not be taken as a fixed limit. On the lingual side the boundary



Distribution of the visuo-sensory (cross hatched) and visuo-psychic (dotted) areas.

P.O. = parieto-occipital fissure.
Sy. = Sylvian fissure.
L.O. = lateral occipital fissure.
P. = superior polar fissure of Bolton.
(after Campbell.)

is usually a small unnamed shallow sulcus which lies longitudinally on the lingual gyrus, mid-way between the calcarine and collateral fissures. This sulcus, however, is also inconstant and therefore cannot be regarded as an invariable limit. Towards the forked extremity of the posterior calcarine fissure the area widens out and becomes rather pear shaped. It is interesting to note that if one limb of the fork is short, the calcarine type of cortex is curtailed likewise. Thus there is a very close relationship between the limbs of the posterior calcarine fissure and this type of cortex. The boundary here is therefore variable.

Finally we come to the distribution of the area on the postero-lateral face of the hemisphere.

Here again it is found that the lateral limit runs in line with and bears a direct relation to the length and position of the fork of the posterior calcarine fissure. In a view of the lateral aspect of the hemisphere then, the field is just visible as a narrow crescent peeping round the tip of the occipital lobe and viewed in full from the occipital aspect it is not usually continued outwards for a distance of much more than 1 c.m.

The Embruological evidence is also of great interest and tends to bear out the results of the histological research. On looking up Flechsig's "Gehirn und Seele" figures 7 and 8, plate IV., it is seen that the closely-dotted calcarine area shown by him corresponds closely to that which we have just described under the term visuo-sensory.

In addition Flechsig describes the course of the sub-cortical band of fibres known as the radiations of Gratiolet. This band of white fibres has long been known to form part of the route by which sensory visual impressions pass between the retina and the occipital lobe.

Embryological investigation shows that at the time of birth only a portion of these white fibres, have acquired their myelinic investment, namely those related to the lateral geniculate bodies; all the other fibres of this radiation of Gratiolet remain unmedullated and further this early medullated band of fibres can be distinctly followed to that part of the occipital cortex which coats the calcarine fissure.

This it will be seen also furnishes very strong evidence in favour of the view that the centre for the reception of primary visual impressions is situated in the region of the calcarine fissure.

This brings us to consider the evidence regarding the localisation of the centre for the reception and interpretation of the visual images of Written and Printed Speech.

On this part of the subject Experimental Physiology can afford us no evidence for obvious reasons.

The Clinico-pathological evidence:

This is of great importance in this connection. A lesion destroying the centre for the storing of the visual images of words gives rise to the condition known as word-blindness, that is, it renders the patient unable to comprehend the meaning of written or printed language, although the letters may be clearly seen and vision generally unimpaired.

Monakow, Redlich, Verrey and other observers have described numerous cases of word-blindness in which careful examination has shown the lesion to be restricted to the region of the left angular gyrus and the second occipital convolution on the left side. The lesion also usually involved underlying tracts of white fibres such as the radiations of Gratiolet and a bundle of white fibres connecting the occipital and temporal lobes and known as the fasciculus longitudinalis inferior. This tract will be referred to when we consider the histological

and embryological evidence.

Dejerine describes a case, which is quoted by Professor Wyllie as follows. The patient was a man aged 68 who after a number of attacks of tingling in the right leg and arm suddenly perceived that he could not read a single word. He could write with perfect ease but was quite unable to read what he had written. He had also lost the power of reading musical notation but could sing well. He could write to dictation as well as spontaneously. The letters though well formed were written a little larger than before. His power of copying, however, was defective. He could only do this with each letter before him and then he merely traced it as he would a map or drawing. He copied printed letters as they were printed and could not transform them into written characters. He had preserved intact the power of reading figures and was able to calculate both mentally and on paper. He had right homonymous hemianopsia. He remained in this condition for four years. Ten days before his death he was suddenly affected with very marked paraphasia and with total agraphia. There was not, however, any paralysis of motion or loss of consciousness. His intelligence remained good. He had no word-deafness.

At the Autopsy there was found:-

(1) An old lesion in the occipital lobe consisting of a softening which had destroyed the base of the cuneus, and the lingual and fusiform lobules, and had extended deeply into the white matter, so as also to cut across the fibres passing from the right occipital lobe to the angular convolution. The grey substance of the convolutions which lie alongside of the posterior part of the internal temporo-occipital fissure was also destroyed.

(2) There was also present a recent lesion, viz. a softening of the angular and supra-marginal convolutions.

From the history of the case then we come to the conclusion that the old lesion accounted for the long-standing word-blindness without agraphia, and for the hemianopsia.

The recent lesion had evidently given rise to the appearance of agraphia and paraphasia.

The latter, no doubt, being produced by disturbance of the neighbouring auditory word centre.

Professor Wyllie[11] also quotes the following case of word-blindness described by Serieux[23]. A female aged 63 had lost the power of reading and writing. She made many efforts to write but these only resulted in confused collections of letters. The intelligence was normal and there were no

paralytic symptoms whatever. Vision was intact and it is expressly stated that there was no hemianopsia. It was as impossible for the patient to read as to write. She could, however, recognise a few of the individual letters. (This as Professor Wyllie points out being probably due to overflow of education into the other hemisphere). Two months after her admission to Hospital, she had an attack of apoplexy with haemorrhage into the ventricles and died. At the autopsy, besides the recent extravastion of blood, there was found in the brain an old lesion, namely, a circumscribed area of softening situated in the angular and supra-marginal convolutions.

OBJECT BLINDNESS.

In addition to these cases of word-blindness it will be well here to refer briefly to that class of case in which in addition to the symptom of word-blindness there is loss of the power of recognising objects. This condition is known as Psychic-blindness, Mind-blindness, or as Professor Wyllie prefers to call it Object-blindness. Cases of this kind vary in degree from a slight diminution of the perceptive faculty to complete inability to recognise objects seen. This condition as I have stated above is frequently associated with

word-blindness. It cannot be said that our knowledge regarding the part of the cerebral cortex concerned in the interpretation of the visual impressions of objects is very exact. The results of clinical and pathological observation show that although a lesion involving the cortex bounding the calcarine fissure is sufficient to produce simple blindness it is necessary for having in addition to this the psychic element that the lesion should be more widespread.

Von Monakow points out that the further the lesion extends into the white substance of the parieto-occipital lobe, the more are long tracts of association fibres interfered with and connections with other centres interrupted. There is evidence also which seems to suggest that a deep lesion in the left occipital lobe seems more likely to cause psychic disturbances than a similar lesion affecting the right lobe.

Professor Wyllie quotes an interesting case in this connection described by Serieux.(24)

The case was that of a female aged 62. She had a stroke in 1888 followed by temporary paralysis, and lasting troubles of vision. In June 1890 she had another attack with epileptic convulsions, hallucinations of hearing, delirium, etc. In the month of December of the same year she was found one

morning, to be suffering from word-blindness, and agraphia and also from word-deafness and paraphasia. Her intelligence was intact, except that there was marked object-blindness. This was exhibited in the inability of the patient to recognise her nearest relations when they called upon her. She said the people about her seemed to be wearing masks. She also made mistakes, for example, mistaking a comb for a pen-holder, and wiping it as such. After a period of some months, during which there was amelioration in all her symptoms, she died of a pneumonia. At the autopsy a lesion was found in the cortex of each hemisphere. On the left side there was a softening in the supra-marginal convolution, and also a limited patch of softening in the posterior extremity of the first temporal. On the right side, there was a large patch of softening involving the angular and supra-marginal convolutions, and also the posterior extremities of the first and second temporal convolutions.

Wernicke quotes a case of Object-blindness described by Gogal in which the patient bit into soap, micturated into the wash-hand basin and could not recognise many other objects. At an earlier stage he had been unable to speak and understand what was said to him. In this case a lesion was found in the posterior part of the first temporal convolution, and behind the fissure of Sylvius, and there was

also a lesion in the third left frontal convolution, the operculum shrivelled up and the island of Reil exposed. On the outer part of the right occipital lobe and exactly on the tip there was a yellow patch of indrawn cicatrix with superficial softening.

In conclusion it may be of interest to draw up a table showing briefly the symptoms in these previously described cases and the situation of the lesion in each case.

SYMPTOMS	SITES OF LESIONS.
Word-blindness, as described in cases by Monakow, Redlich and Verrey.	Angular Gyrus. 2nd Occipital convolution Radiation of Gratiolet. Fasciculus longitudinalis inferior.
Word-blindness without Agraphia (4 years duration) Homonymous hemianopsia.	Old lesion in left Cuneus, Lingual and fusiform lobules, involving white fibres from right occipital lobe to angular convolution.
Followed 10 days before death by	
The addition of Agraphia (as described by Dejerine).	Recent lesion in left angular and supra-marginal convolutions.
Word-blindness and Agraphia (Serieux).	Left angular and supra-marginal convolutions.

S Y M P T O M S	SITES OF LESIONS.
Word-blindness, Agraphia, word-deafness, Paraphasia and Object-blindness. (Serieux)	Left Supra-marginal, and posterior part of 1st temporal convolutions. Right angular and supra-marginal and posterior extremity of 1st and 2nd temporal convolutions.
Object-blindness (Gogal)	Posterior part of 1st left temporal convolution, and tip of outer part of right occipital lobe.

From a study of the clinico-pathological evidence, therefore, we arrive at the following conclusions:-

(1) That the centre for the reception and interprelation of the visual images of words is in the neighbourhood of the left supra-marginal and angular convolutions.

(2) That injury to the white fibres conducting impressions from both occipital lobes to the above region may cause word-blindness without the angular gyrus being itself involved.

(3) That when the angular gyrus is destroyed Agraphia as well as word-blindness results.

(4) That a bilateral lesion seems to be necessary for the production of object-blindness.

The Histological and Embryological evidence regarding the localisation of the centre for the reception and interpretation of written and printed speech.

The result of a study of this part of the evidence regarding the existence and localisation of a centre which may be concerned in the elaboration of the primary visual impressions of words written and printed is on the whole rather vague.

Campbell, however, has shown that investing the area of cortex previously described under the term Visuo-sensory, there is a type of cortex possessing again a distinctive histological structure. Its close relation to the calcarine area and other points to be referred to later suggest to him that this type of cortex is the site of the cortical centre for psychic visual representation. The peculiarities in the nerve-fibre arrangement and cell lamination I need not go into here.

We must, however, look carefully at the area under consideration as located by Campbell from his histological studies.

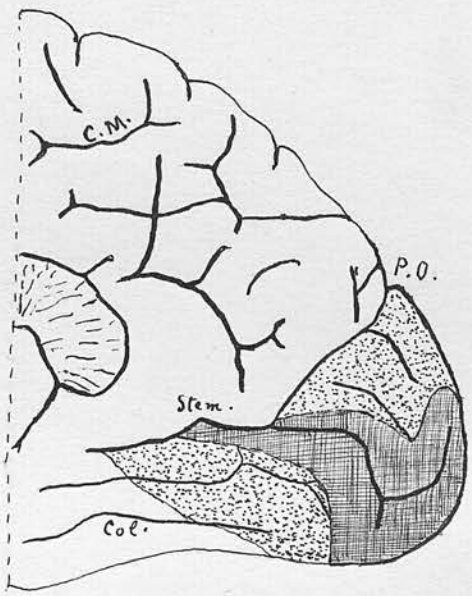
In the first place he says that though it is easy to note the distinctive characters of this type of cortex in the central parts of this area, the exact demarcation of its distribution is very difficult. This is accounted for by the fact that the change between the type of cortex under consideration and the surrounding types is gradual and not abrupt. This is especially the case where this field joins the parietal and temporal areas. (This is an admission which is important as we shall see later). The boundaries of this "Visuo-psychic" type of cortex are as follows:- "Starting with that part of the area situated on the mesial surface of the hemisphere, above the calcarine fissure it is to be observed that it does not extend on to any part of the gyrus fornicatus, or of the precuneus, lying above the anterior division of the fissure indicated; indeed it is not found even on the upper wall of this portion of the fissure.

Proceeding backwards, however, we find that it makes an appearance on the gyrus cunei (i.e. the deep cuneo-limbic annectant gyrus found at the point of junction of the parieto-occipital and calcarine fissures); and above this point the floor of the parieto-occipital fossa, not in its entire extent, but almost as far up as the margin

of the hemisphere, may be described as a boundary. From which it follows, that all that portion of the cuneus which remains uncovered by a visuo-sensory type of cortex is occupied by a visuo-psychic type. This distribution, however, does not obtain invariably, because in one of the brains examined a considerable extent of the surface of the cuneus, lying in the angle formed by the margin of the hemisphere and the parieto-occipital fossa, was left uncovered, in fact it seems to be the rule for this type of cortex to recede from the parieto-occipital fossa in this situation. And as a further consequence, when we inspect the margin of the hemisphere from the dorsal aspect, we find that the external prolongation of the parieto-occipital fossa lies in some cases anterior and in others posterior to the margin of the area, therefore it also cannot be regarded as a fixed boundary.

Continuing our examination of the distribution of the area on the postero-lateral surface of the hemisphere, we find that the outer edge assumes the form of a curve, running more or less parallel with and at a distance of 1.4 to 2 cms. from the margin of the calcarine or visuo-sensory area. To the visuo-sensory area, therefore, it bears a definite relation, but it cannot be said that its distribution in like manner is influenced or regulated with

any degree of constancy by the fissures which cut up this portion of the occipital surface; thus concerning its relation to the ramus occipitalis transversus of Ecker, anteriorly the area usually crosses the upper half of that sulcus and spreads for a varying extent over the convolution which is known by the name of the superior parieto-occipital annectant gyrus, while lower down the border does not reach so far forwards as the lower half of the same sulcus, accordingly the area does not touch the inferior parietal lobule. (This, it will be noted, is contradictory evidence compared with that of the clinico-pathological). And concerning its relations to the lateral occipital sulcus of Eberstaller, when this sulcus is present in typical form, the line of demarcation merely cuts across its posterior extremity. Coming lastly to the under syrface of the hemisphere, we find that the boundary of the area still closely follows the visuo-sensory outline. Posteriorly, it lies lateral to the hinder extremity of the collateral fissure, but it soon crosses this fissure and curves upwards to end in the stem of the calcarine fissure, at a point a few m.m. anterior to the commencement of the visuo-sensory area; and here it may be noted, that although the visuo-psychic and hippocampal fields converge at this point, there is not the



Drawing to illustrate the distribution of the visuo-sensory (cross hatched) and the visuo-psychic areas (dotted).

"Stem" is placed above the division distinguished by that name.

Col. = collateral fissure.

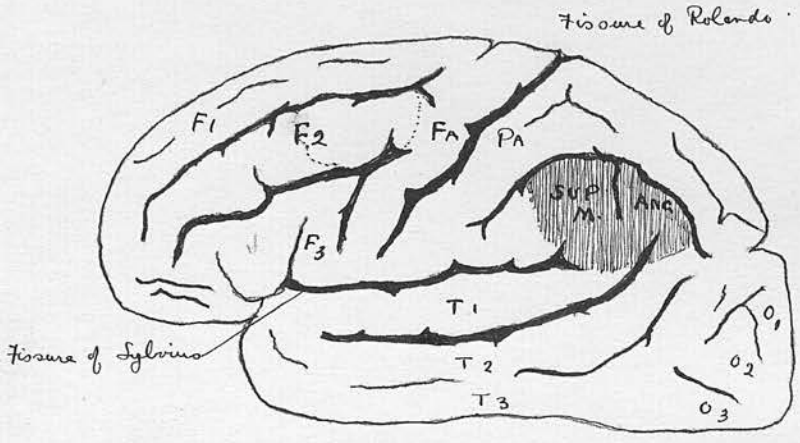
P.O. = parieto-occipital fissure.

C.M. = calloso-marginal fissure. (after Campbell.)

least difficulty in discriminating between the two types of cortex.

Summed up, therefore, the visuo-psychic area may be described as a zone of cortex from 1.3 to 2 cms. broad, investing the visuo-sensory area on all sides, that part above the stem of the calcarine fissure excepted."

The histological evidence then, is, in this instance, somewhat contradictory to that of the clinico-pathological. Particularly is this so with regard to the case of the angular gyrus. We have the strongest clinical and pathological evidence to show that the angular gyrus is most intimately connected with the storage of the visual word images. If reference be made to the cases previously quoted, it will be seen that when word-blindness has been present as a symptom, the post-mortem examination has invariably shown that either this part of the cortex itself was destroyed, or was cut off from its connections with both primary visual centres. It will also be noted that Campbell's evidence with reference to the exact demarcation of the type of cortex under consideration and its junction with the surrounding areas is, as he himself confesses, very vague. In addition to this, his own statements are to a certain extent



Drawing showing the localisation of the word-seeing centre in the supra-marginal and angular convolutions. (after Wyllie.)

contradictory of each other. Thus he says, in referring to the temporal type of cortex that this type covers the angular gyrus, but he further adds that the change from one type of cortex to another is in this area very gradual and "it is really impossible to lay down a sharp line of demarcation". Later also in referring to the distribution of the parietal type of cortex, he says that though it is exceedingly difficult to be sure of the exact point where"parietal"cortex ends and "temporal" cortex begins, yet he thinks it is correct to say, that the parietal type of cortex tends to cross the horizontal sulcal line and to trespass on the upper part of the angular gyrus. These contradictory statements show, I think, that in this case the histological evidence is decidedly lacking in definiteness and certainly cannot be held as refuting the conclusions we have come to from the clinico-pathological evidence that the centre for storing the images of written and printed speech is situated in the region of the left angular gyrus.

The clinico-pathological and histological evidence, however, seem to agree in leading one to the conclusion that the conditions of object-blindness, colour blindness and other interferences with the interpretation of visual stimuli, such as for

example the interpretation of arithmetical or algebraic signs, or the reading of musical notation, are due to lesions in that part of the occipital lobes which Campbell has marked out as the visuo-psychic area. This subject, however, owing to the extreme complexity of psychic visual processes is not yet capable of being put on a satisfactory basis.

It is rational, however, to expect, I think, that if this visuo-psychic area in the occipital lobes is, as we suppose, the centre where primary visual sensations, with the exception of the images of words, are elaborated that this centre would tend to be situated in close connection with the primary visual centre. On the other hand, a highly specialised centre such as that for the storage of the visual images of words would, one would naturally expect, be situated in the neighbourhood of that part of the cerebral cortex concerned in the function of speech.

Is it not also further possible, that the histological characters of such a specialised centre as this, might be, to a certain extent, different from that of the ordinary visuo-psychic area, and is not the fact that there is such an indefiniteness about the histology of the cortex in the region of the angular gyrus rather suggestive that further

investigation might show that in this situation there was situated, probably a small, but distinctive type of cortex ?

MOTOR APHASIA.

We now proceed to the consideration of the localisation of the Centres for the production of Spoken Speech.

In doing so we have to consider:-

(1) The localisation of the centres for the various Respiratory, Vocal and Oral articulative movements requisite for the production of spoken speech.

(2) The localisation of the centre for the storage of the psycho-motor memories of words.

That two such centres exist is proved by the fact that a patient may be quite unable to speak without there being any paralysis of the various sets of muscles used in the production of spoken speech.

Let us look then in the first place at the Respiratory, Vocal and Oral articulative centres.

The Respiratory Centre:

At the present time we have no definite knowledge regarding the localisation of this centre in the cerebral cortex. We know, however, that Respiration is carried on automatically by the primary respiratory centre in the bulb. If a higher controlling centre exists in the cortex, as is probable, then this centre, according to Broadbents law, will be bilaterally represented.

The cortical centres for the Vocal mechanism have, however, been definitely localised by means of experimental physiology.

Horsley and Semon[25] showed that the centre for the adduction of the vocal cords was situated in the foot of the ascending frontal convolution and that the centre was completely bilaterally represented in the hemispheres.

Accordingly, stimulation of one cortical centre gives rise to adduction of both vocal cords; and further, the destruction of one centre does not give rise to loss of the power of adduction on either side. With regard to the centre for the abduction of the vocal cords, Risien Russell[26] has shown that this centre is situated in the same region a little above and anterior to the centre for the movement of adduction.

The Oral Articulative Mechanism;

Ferrier in his work on the "Functions of the Brain" localised the centre for the movements necessary for articulation in (1) the foot of the third frontal convolution; (2) the foot of the ascending frontal convolution; (3) part of the foot of the ascending parietal convolution.

This, it will be seen, is the area known as "Broca's convolution". Horsley and Beevor[27], however, in their work on the same subject limited the representation of the movements for the lips and tongue to the foot of the ascending parietal and ascending frontal convolutions and did not include the foot of the third frontal convolution. As a result of these experiments which were confirmed by later observers Professor Wyllie pointed out, that while the foot of the third frontal convolution was not included in the motor area for the lips and tongue, yet a lesion involving the foot of this convolution gave rise to great derangement of the power of producing speech.

He accordingly suggested that the foot of the third frontal convolution was the storehouse of the guiding psycho-motor pictures or memories necessary for spoken speech and that the executive motor cells were situated in the foot of the ascending frontal and ascending parietal convolutions.

This brings us to consider the clinico-pathological evidence with regard to the localisation of the centre for storing the psycho-motor images of words.

In the year 1861, Broca published his cases of motor aphasia and drew attention to the close association that exists between the power of articulate speech and the posterior part of the third left frontal convolution.

It will be necessary here, for later reference, to make a note of the symptoms in both of those cases with the results of the post-mortem examination.

Case I:

Aphemia lasting for 21 years, produced by chronic and progressive softening of the second and third convolutions of the left frontal lobe.(28)

On the 11th of April, 1861 there was brought to the surgical department of the Bicetre Hospital a man of 51 years, named Leborgne, suffering from a diffuse gangrenous phlegmon of the whole of the right inferior extremity. He died from this affection five days after coming under Broca's care.

Leborgne had been an old inmate of the asylum having lived in it for a period of about 20 years.

He had been, since his youth, subject to attacks of epilepsy. At the age of thirty he lost his speech and this was the reason of his admission into the Bicetre Asylum. He had been unable to speak for two or three months before his admission. He was active and his intelligence was intact. He was known in the Hospital by the name of "Tan" because this was the only syllable he was able to pronounce. He expressed himself well by means of gesture language. When people did not understand what he meant, however, he became angry. On these occasions he added an oath to his vocabulary, namely "Saire nom de Dieu". He had been for 10 years in the Bicetre Hospital when a new symptom manifested itself. The muscles of his right arm became enfeebled and finally paralysed and the paralysis gradually extended to his right leg. His sight and intelligence became also affected. For the last six or seven years of his life he had been constantly confined to bed. In this condition he was seized with the phlegmonous erysipelas for which he was transferred to the surgical ward and placed under the care of Broca.

As already said, he died in about five days after his admission.

Post Mortem:

The external surface of the dura-mater was red and very vascular. Below this the pia-mater appeared very injected at certain parts and in some places thickened and opaque. In the left hemisphere a very extensive area of softening was found. It involved the greater part of the frontal lobe, as well as the corpus striatum, the island of Reil, and the convolutions lying along the margin of the Sylvian fissure. On careful examination Broca was able to satisfy himself that in this vast area of softening, the oldest portion was that at about the middle of the frontal lobe. It appeared evident that in this neighbourhood the softening had first begun, and that it was when the softening was limited to this neighbourhood that the patient had been merely aphasic and not paralysed. The subsequent hemiplegic paralysis had been caused by the backward extension of the softening. This was Broca's explanation of the case.

Case II:

The following is a resumé of Broca's second case. A full account of both these cases being given in the "Bulletins de la Societe Anatomique de Paris p.398."

The second cases is headed:-

"New observation on a case of Aphemia produced by a lesion of the posterior part of the second and third frontal convolutions."

The patient was a man named Lelong, aged 84 years. He was brought to the Bicetre Hospital on the 27th October 1861 owing to a fracture of the neck of his left femur. Previous to this, however, he had been admitted to hospital in April 1860 on account of an apoplectic stroke with temporary loss of consciousness. He had recovered in a few days without any hemiplegia, though his walk, without being lame, was a little uncertain. The attack, however, had rendered him aphasia so that his vocabulary was reduced to a few words. When asked a question he replied only in signs, accompanied by one or two syllables articulated energetically and with a certain effort. These syllables were "oui", "non", "tois" and "toujours".

When asked his name he said "Lelo" instead of Lelong, his real name.

The patient could express himself well in pantomime when words failed him. When asked what his former occupation had been, he imitated very well the action of using a shovel.(He had been a labourer).

This latter point will have to be referred to later with reference to a statement made by Marie in which he says that he has never found any patient

who could indicate by pantomime what his previous occupation had been.

Post Mortem:

On examining the brain there was seen a superficial lesion of the left frontal lobe immediately above the anterior extremity of the fissure of Sylvius. The lesion in this case was much more circumscribed than in the previous case. But in comparing the two cases one found that the centre of the lesion was identically the same in both.

In the case of Lelong, it consisted of a cyst-like cavity filled with serum. It involved the region of Broca and also extended to the posterior extremity of the 2nd frontal convolution. As we have noted above it was sharply defined in its margin and base, and the presence of haematin crystals in its wall showed that the cause of the local destruction of the cerebral tissue had been the apoplexy in 1860.

These two cases are of great interest historically. Clinically they present the characteristic features of Motor Aphasia. The site and extent of the lesion in both is very interesting and shall have to be referred to later.

The case described by Dr Wm. Ogle[29] and quoted by Professor Wyllie is of great interest also. The patient, a man of rheumatic constitution and the subject of aortic and mitral disease had fallen down suddenly three days before admission and found himself hemi-plegic and aphasia.

On examination his speech was found to be limited to the two words "yes" and "no".

At first he had difficulty in deglutition and in putting out his tongue, but these symptoms passed away in a few days.

He understood all that was said to him and expressed himself well by pantomime. "He could write with his left hand with sufficient distinctness words which he could not pronounce when asked to do so. In his writing there was often a tendency to reduplication of letters.

For instance, he wrote "Testatament" for "Testament". But I cannot say whether this was more than the result of defective education.

The Autopsy showed the brain to be healthy except at two spots, of which the chief was the posterior part of the third frontal convolution on the left side. Here was a softened and almost diffluent patch, about three-quarters of an inch in breadth, reaching from the highest part of the third convolution backwards and downwards to the Fissure

of Sylvius. The softened part was not actually the most posterior part of the convolution, for there was a narrow unsoftened strip between it and the ascending frontal convolution. On cutting into the brain a second small patch of softening was seen in the centre of the left hemisphere external to and rather above the corpus striatum, and extending towards the posterior extremity of the fissure of Sylvius. All the rest of the brain was apparently healthy."

The cause of the softening in Broca's convolution was found to be occlusion by embolism of a branch of the Sylvian artery.

It is specially to be noted in this case that (1) There was a double lesion - that is, a cortical and a substance lesion in the left hemisphere - and (2) That the patient in this case retained the power of writing.

The question now arises what would be the result of a lesion involving the oral articulative and vocal mechanisms, without the foot of the 3rd frontal or psycho-motor centre being affected?

This is a part of the subject of which, although our knowledge is in some points rather indefinite, yet from a study of many cases the following conclusions are held as being practically proved.

(1) That corresponding parts of the cortex in each hemisphere are connected with each other by means of commissural fibres passing through the corpus callosum.

(2) That. therefore, the oral articulative centres in the left side of the cerebrum are connected with the corresponding region on the right side.

(3) That the psycho-motor centre on the left side is connected with the corresponding region on the right side, and that impulses from the psycho-motor centre on the left side reach the oral articulative and vocal centres on the right side by means of either,

(a) The centre corresponding to the psycho-motor centre on the right side.

(b) Dircetly from the left psycho-motor centre to the oral articulative and vocal mechanism on the right side.

In addition to these connecting links high up in the cerebrum there are also important commissural fibres in the bulb connecting the oral articulative centres. There is thus a more or less complete bilateral representation of the centres for the movements of the lips and tongue necessary for the production of spoken speech.

It follows in consequence that destruction of either oral articulative centre will not give rise to loss of the power of speech, but will produce a certain amount of difficulty in articulation or Dysarthria, as it is termed. There are cases on record which prove that a lesion of the oral articulative centre on the right side gives rise to dysarthria in the same way that a lesion in the corresponding left centre would, with this difference, that a lesion on the left side produces more marked dysarthria than a similar lesion on the right side, and that a right sided lesion is more quickly recovered from. This also shows that since a lesion of the oral articulative centres on the right side gives rise to dysarthria, the commissural fibres in the pons are not quite sufficient for the innervation of both sides, and in consequence proves the existence of commissural fibres higher up in the cerebrum.

Lichtheim in discussing the subject of the existance of decussating fibres high up in the cerebrum, says "if the fibres from Broca's centre reached the basal organs down the left side only, the usual persistent aphasic symptoms would arise from lesion of the left peduncle or internal capsule as uniformly as they do from those of the centre itself." "But we know that this is only exception-

ally the case; hence there must be a partial decussation of the speech tract from left to right hemisphere within the brain itself, so that the left internal capsule does not contain the whole bundle of these fibres." It further seems probable that between Broca's convolution and the upper part of the left internal capsule the fibres passing directly downwards from the left psycho-motor centre and the commissural fibres passing to the speech centres on the right side are situated very close to each other and thus may be both cut across by the same lesion. This produces what is known as the infra-pictorial variety of motor aphasia.

The generally accepted views on this subject then, have been as follows:-

(1) That the foot of the 3rd left frontal convolution is the centre where the guiding psycho-motor images of words are stored.

(2) That the centres for the oral articulative and vocal mechanisms are situated in the foot of the ascending frontal and ascending parietal convolutions on either side.

(3) That lesion of the psycho-motor speech centre results in inability to speak or Motor Aphasia; whilst lesion of the oral articulative

centres on either side, or lesion of the fibres proceeding from one of those centres to the bulb, produces interference with articulation or dysarthria.

(4) That a lesion involving the fibres connecting the psycho-motor centre to the vocal and oral articulative centres on the same and opposite sides also produces loss of the power of speech. This is known as Infra-pictorial motor aphasia.

(5) That although the patient is unable to speak yet he understands what is said to him.

(6) That the intellectual capacity of the patient is retained.

The above views have been generally accepted by those who have made a special study of the subject as being correct. Within the last few months, however, Professor Pierre Marie of the Bicetre hospital has written several articles in "La Semaine Medicale" in which he expresses the opinion that the existing doctrines with regard to the subject of Aphasia require consideration. His views are very much at variance with those expressed above.

I propose, therefore, to consider in the following pages the results of Marie's investigations, and finally to compare his views with those which have been generally held, in the light of clinico-pathological observation and recent anatomical and histological research.

Marie's articles have been published in "La Semaine Medicale" the main part of his work being in the number dated 23rd May 1906. In that number his article is headed "Revision de la question de l'aphasie; la troisieme circonvolution frontale gauche ne joue aucun rôle special dans la function du langage".

This it will be seen is in direct variance with our previous ideas on the subject. We shall then have to proceed to consider in full what Marie's views are and on what grounds he has based his conclusions.

In introducing the subject Marie says that he has personally investigated all the cases of Aphasia that have come under his care in the Bicetre hospital in the last ten years. During that time the number of cases of Aphasia has been nearly 100 and he has had occasion to make more than 50 post-mortem examinations. He has, therefore, had large opportunities

of studying the subject and his conclusions have not been hurriedly arrived at. He says that in the first cases which he had the opportunity of examining, he was struck by the discordance of the facts presented as compared with the generally accepted theories on the subject.

He attacks in the first place the theories advanced by Wernicke. While giving Wernicke credit for his discovery of a variety of Aphasia distinct from that described by Broca, he regrets at the same time that he should have endeavoured to base it on the existence of a hypothetical auditory centre.

He says - "Nothing, absolutely nothing, from the point of view of careful clinical observation allows us to consider the first temporal convolution as the centre for hearing."

In the second place he goes on to say that one fact dominates the whole study of Aphasia, it is that - "in all cases of Aphasia there is present a more or less pronounced difficulty in the comprehension of spoken language."

The degree of this difficulty, is, however, he says, very variable. In certain very pronounced cases of Aphasia the patient is unable to understand almost every word that is said to him; in other slighter cases the patient understands simple questions quite well, and is able to execute simple

orders given to him, but if the questions or the orders become more complicated there is immediately evident the characteristic deficiences of the affection.

He, therefore, says that in order to test a patient with Broca's Aphasia as to his comprehension of spoken language it is not sufficient to ask him to perform such simple commands as to put out the tongue, or shut the eyes, which the patient may be well able to accomplish but he insists that it is necessary to ask him to perform more complicated acts such as - "Of the three pieces of paper of different size lying on this table you will give me the largest; the second largest you will crumple up and throw on the floor; and the smallest you will put in your pocket." Other similar orders may thus be given to test the patient's powers of comprehension, certain patients will be found to have great difficulty in performing even a single act, whilst in order to cause others difficulty it will be necessary to give two consecutive commands or even three or four. When a patient does not understand what is said to him Marie does not attribute this to word-deafness but to defective comprehension. In patients suffering from Aphasia he says there is a symptom more important and more serious than the mere loss of the meaning of words; <u>there is a very</u>

marked diminution of the intellectual capacity in general.

This is a striking statement when we consider that in practically all the previously described cases of Aphasia there was a special note to the effect that the general intelligence was good.

To put it shortly, Marie holds that the area of Wernicke is not simply a sensory, but an intellectual centre.

If he says, I had to give a definition of Aphasia, the facr I would lay especial stress on would be the diminution of the intelligence.

This diminution of the intellectual capacity may, however, not be apparent on casual examination and it may be necessary to spend some time with the patient and to make a careful and methodical examination. If one does so, it will then be seen that it is not merely the faculty of speech that is affected, but that there probably exists at the same time the loss of certain didactic acquirements.

Marie asks for example, if it is merely due to a simple disturbance of the faculty of speech that "musiciens aphasiques" find their musical faculties deranged, not only on attempting the composition or rendering of a piece, but also on endeavouring to play from memory pieces which were previously familiar to them; also if it is a simple disturbance of speech that renders the patient unable to recognise

the hour on a watch, or to place at a certain hour the hands of a watch, and is it due to the same cause that patients given a very simple addition or subtraction exercise fail entirely to do this, and often indeed begin at the left, thus showing that they have not the least idea of the most simple rules of arithmetic. Lastly it is still more certain that it is not a mere disturbance of the speech faculty that renders severe cases of Aphasia unable to imitate correctly a number of simple acts which another person performs before them.

He quotes the case of an aphasic cook who before his illness was expert in his work, but who when taken to the hospital kitchen and asked to make the well known dish "oef sur le plat" committed almost every blunder it was possible for a cook to make; mixing for example all the materials together in the saucepan before putting it on the fire, instead of melting the butter first and then breaking the egg into it, the result being that the dish was uneatable. This, says Marie,"was very evidently not due to a disorder of speech but to an intellectual deficiency.

Finally he says that the intellectual deficiency apart from the question of language is undeniably present but to recognise it a methodical examination

may be necessary. Superficially, the intelligence of these patients does not show any marked disturbance.

In their family they take part in the daily routine. In hospital they come and go like their fellow patients. They sleep and eat at the same hours and in the same conditions as their companions.

In a word, they conduct themselves like ordinary sensible people.

It is true, however, that their powers of ideation are considerably restricted. There is one misleading factor present in aphasic patients which tends to strengthen the impression of the integrity of their intellectual faculties. This is the preservation and sometimes even the exaggeration of their emotional faculties. These patients love and hate. They suffer and rejoice when others suffer or rejoice. In short they live a moral life perfectly similar to ours, and it is on account of this, that we have found ourselves led to consider their psychic faculties as normal. The observance of social distinctions is a point in their general conduct which is noteworthy. For example, one of the patients suffering from well marked aphasia, who previously had been a solicitor and was socially superior to his fellow patients, recognised his social standing, and did not condescend to any

familiarity with his fellow patients. Again those patients retain the power of becoming angry at mention of events in the past which have made them angry. Thus one of Marie's patients had been deserted by his wife, and on the slightest allusion to the name of "woman" he immediately got into a state of violent anger, his face became red, and the sweat ran off his forehead.

Lastly, he notes that aphasics show politeness in their general conduct, showing that in cases of Aphasia there is still retained "la sphere affective et morale".

These then, says Marie, are some of the reasons why the intelligence of these cases is often considered to be normal. There is still another. Their air of satisfaction, the brightness and exuberance of their powers of mimicry in certain directions, seem to be points in favour of the integrity of their mental faculties. But as a matter of fact this apparent richness of their powers of mimicry hides a real poverty. Marie holds that though they possess a wealth of "la mimique emotive" yet they have very slight command of "la mimique conventionelle". Thus if one tells such a patient to show that he wishes to lie down and sleep, or to make a sign of disgust, it is rarely that the patient

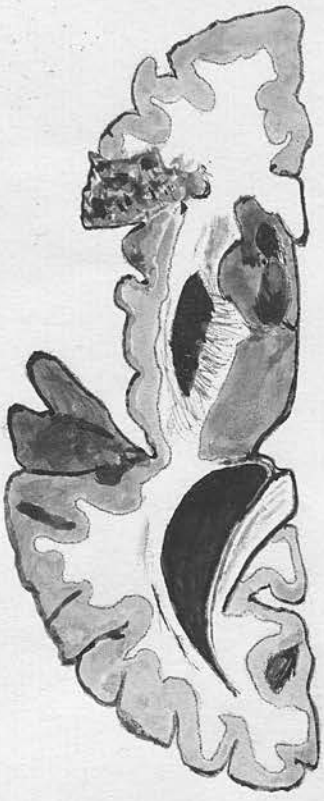
does so, or if he does, it is after some hesitation and uncertainty. Regarding "la mimique descriptive" Marie says that he has never seen an aphasic patient who could describe by means of gestures his previous occupation. This fact, he says, is another argument in favour of the theory that the intelligence of these patients is affected, as in this case the psychic disturbances are entirely independent of speech. From all this it follows, according to Marie, that the area of Wernicke is not merely a word-hearing but an intellectual centre.

We now come to consider Marie's views with regard to Broca's aphasia.

Marie holds that the third left frontal convolution is not possessed of the special function concerning speech that has been ascribed to it.

The arguments he brings forward against the localisation of the centre for the production of spoken speech in the third left frontal convolution are of two varieties.

(1) There have been cases of aphasia in right-handed people, in whom isolated destruction of the posterior part of the third left frontal convolution was not followed by Aphasia.



Reproduction of a drawing illustrating Maries paper in La Semaine Medicale: —

It represents a horizontal section of the left hemisphere. The third frontal convolution is almost entirely destroyed by a softening. This patient showed no sign of either Aphasia, or Anarthria.

He acknowledges, however, that the number of such cases is small; but explains it by the fact that aphasia is generally due to softening as a result of obliteration of the middle cerebral artery, at a point above the origin of the branch to this convolution, and that destruction of the 3rd left frontal convolution is therefore associated with that of parts which are the true seats of the function in question.

Marie has so far published scanty evidence in support of his theories but promises to publish full descriptive cases in the near future. In his paper, however, he gives a sketch, a reproduction of which is shown opposite, of a case of lesion of the foot of the third left frontal convolution in which the patient, though right-handed did not show any sign of aphasia.

It is disappointing to find that he does not state briefly what symptoms or physical signs were present, and that his description of the lesion is not more exact.

(2) He points out that there have been well marked cases of Broca's Aphasia in which there was no lesion whatsoever of the 3rd left frontal convolution.

Marie claims to possess records of several cases of this kind which he promises to publish at

a later date. From a consideration of such cases, he draws the conclusion that "la troisieme circonvolution frontale ne joue aucun role special dans la fonction du langage".

Finally, however, he admits that in about 50 per cent of all the cases, lesion of the third left frontal convolution has coincided with aphasia, but this, Marie says, is to be explained by the anatomical arrangement previously referred to.

When this lesion of the 3rd left frontal exists, it is, he holds, purely and simply a coincidence due to an extension of the obliterated vascular region and nothing more.

Let us now look at the reasons which Marie gives in support of his view that the 3rd left frontal convolution does not play a special rôle in the function of speech.

As he says, seeing that the two types of aphasia known as the aphasia of Broca, and the aphasia of Wernicke, are undoubted clinical realities, it is right that he should give his opinion regarding the nature and mode of production of these two varieties of aphasia.

His conclusions have been arrived at, he says, purely as a result of direct observation of clinical

and anatomico-pathological facts. In the first place he defines the terms:-

(1) Aphasie de Wernicke

(2) Aphasie de Broca

(3) Anarthrie (ou dysarthrie).

The Aphasia of Wernicke is characterised by the fact that the patients retain the power of speaking, sometimes even they speak too much, but they speak badly; they present the symptom known as paraphasia, and they do not understand to any extent what is said to them; this as we have seen being due not to word-deafness, but to an intellectual deficiency. For the same reason they are unable to read and write.

In the Aphasia of Broca the patients are also unable to read and write and do not understand well what is said to them, but there is the great distinction that in this variety they are unable to speak. In short the only essential difference between these two types of aphasia is that in the one the patient can speak, in the other he cannot. Otherwise they resemble each other in many respects; in both there is incapacity to understand what is said when the question is complicated.

Such facts have been noted by many authors and in particular the presence of word-deafness in

Broca's aphasia has been specially referred to in the works of Thomas and Roux. According to Marie, therefore, the aphasia of Broca is the aphasia of Wernicke minus speech.

Anarthria is the third term to be defined. Under this term is included not only absolute anarthria due to a lesion of the brain, but also cases of marked dysarthria. This condition is characterised by loss of comprehensible speech to such an extent that it may be possible to confound it with Broca's aphasia. The distinctions, however, are numerous and decisive. In anarthria the patient understands perfectly what is said to him, even when the phrases are complicated. He can read and write and is capable of indicating by signs the numbers of letters or syllables of which the words he is unable to articulate are composed. By these characteristics it is easy to recognise the clinical picture of sub-cortical, or as Professor Wyllie has termed it, Infra-pictorial motor aphasia. Marie thinks that this condition should be separated from the group of aphasias and placed with the pseudo-bulbar paralysis, since aphasia includes in the first place inability to understand language, dependent on intellectual decay, and in the second place loss of the power in reading and writing, neither of which

is found in anarthria.

We have now to consider in what regions of the brain Marie localises these different symptoms.

With regard to "Anarthria", there is no difficulty, he says. In this case the lesion is admitted to be situated in the region of the lenticular nucleus, in the nucleus itself, or in the anterior part of the "knee" of the internal capsule. "Anarthria" is not specially connected with the left hemisphere, but may occur when the lesion is situated on the right side, and when one side only is affected it shows a spontaneous tendency to recovery, or to considerable improvement, owing to the complimentary action of the unaffected hemisphere.

With regard to Aphasia, (Marie here uses the singular intentionally) we have seen that the only notable distinction between the aphasia of Wernicke and the aphasia of Broca is the fact that in the first the patients speak more or less badly, whilst in the second, they do not speak at all. In both forms, however, there is present the intellectual deficit which manifests itself in the difficulty of understanding phrases a little complicated and the difficulty or impossibility of reading and writing. In a word, Broca's aphasia is merely aphasia com-

plicated by anarthria, or anarthria complicated by aphasia.

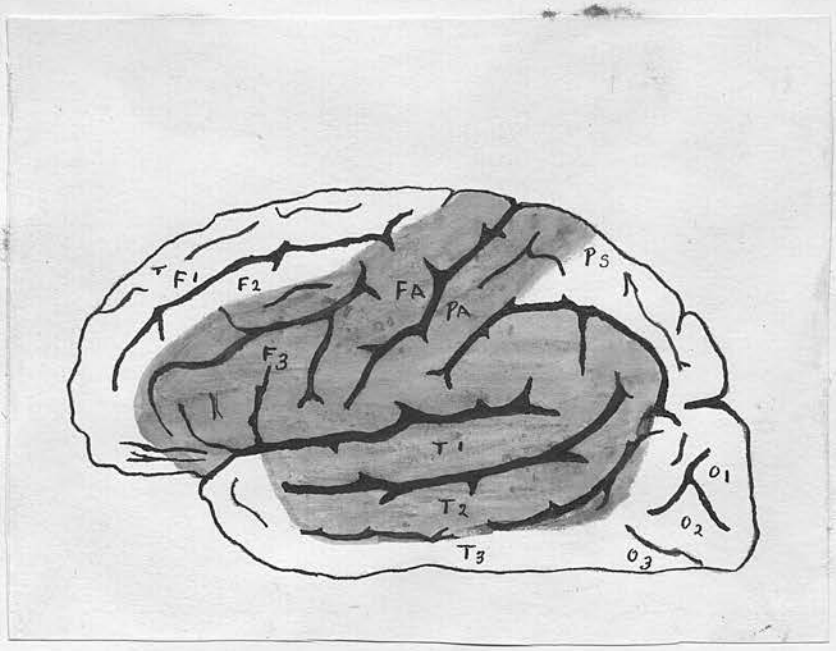
But it must follow that, since aphasia is one condition according to Marie, its localisation must likewise be one, and this, he says, is really so. The only region of the brain, lesion of which produces aphasia is the area of Wernicke. This includes the supra-marginal gyrus, the angular gyrus, and the posterior portions of the first two temporo-sphenoidal convolutions. Aphasia may be produced when there is present even a limited lesion in any part of this area. This is explained by the law that "complete one-sided cerebral symptoms may result from a lesion of merely a portion of the zone in which the functions are seated." Further if the lesion of this cortical area is very extensive, or if it involves the under-lying white substance so that tracts of fibres such as the inferior longitudinal bundle, are interfered with, the aphasia produced is very marked. On the other hand, if the lesion of the area of Wernicke and the sub-jacent white substance is less extensive, the aphasia will be less marked, sometimes even so slight, that it is difficult to notice it if one does not see the patient within the first few days subsequent to the attack.

Marie's views on the aphasia of Broca:

As we have seen in the previous page Broca's aphasia = Aphasia + Anarthria. This simple definition, says Marie, is sufficient to indicate to us what lesions will give rise to the aphasia of Broca. The aphasia is due to a lesion of the zone of Wernicke and the Anarthria to a lesion in the neighbourhood of the lenticular nucleus or in the nucleus itself. How then is produced from the clinico-anatomical point of view this combination of the lesion situated in the region of the lenticular nucleus producing anarthria, and that in the area of Wernicke giving rise to the aphasia. Marie explains it as follows:- It is generally a softening which gives rise to the condition. The softening produced by occlusion of the Sylvian artery which is responsible for the aphasia of Broca may be of two very different types depending on whether the lesion is cortical or situated in the white substance of the hemisphere.

(1) Forme a lesions corticales.

Obliteration of this artery (a) almost immediately after its origin in the circle of Willis gives rise to complete destruction of three-fourths or the middle two-thirds of the left cerebral hemisphere; the lesion includes the central nuclei and the white substance surrounding them, the 3rd frontal,



This drawing shows the cortical lesions produced by an enormous softening, due to obliteration of the Sylvian artery at its origin.
In addition to the central convolutions the lesion had destroyed the 2nd frontal, the angular and the two first temporal convolutions.
The patient showed the typical symptoms of Broca's Aphasia. Besides the lesion of the cortex in the area of Wernicke there was present a lesion of the central ganglions which was responsible for the production of Broca's aphasia. It is true that there is here a softening of the third frontal convolution, but this lesion is super-added and is not the cause of the Broca's aphasia. (After Marie.)

the Rolandic area, the supra-marginal and angular convolutions and part of the first two temporal convolutions.

(b) Between the anterior perforated space and the origin of the artery supplying the 3rd frontal convolution. In this case the convolutions affected are the same as in the proceeding case, with this difference, that the branches which pass through the anterior perforated space not being occluded, the central nuclei only show limited lesions localised especially in the neighbourhood of the external capsule and analogous to the form that we will next consider.

(2) Forme Profonde:

Another variety is the following which seems to occur with a relative frequency and gives rise to the aphasia of Broca as completely as the former type. The obliteration in this case is not in the trunk of the artery but in one of the branches of its bifurcation. As a rule it is situated below the origin of the artery for the 3rd frontal convolution which remains unaffected. This branch is the one which supplies the angular and supra-marginal convolutions and the 1st two temporal gyri, Whilst the other branch supplies the Rolandic convolutions. As this latter branch escapes these convolutions are not affected. The softening then affects only the

angular and supra-marginal convolutions and a part of the 1st two temporal gyri, and again this area may be still further restricted in size by the collateral circulation. It would appear then that such a softening should give rise to the aphasia of Wernicke and yet during life it has given rise to the aphasia of Broca. The explanation of this is that if one makes horizontal sections of the hemisphere one discovers that the white substance which unites the parieto-occipital lobe to the region of the central ganglions is the site of a well marked disturbance which one can trace into the retro-lenticular region and as far as the external capsule, involving sometimes to a greater or less extent, the lenticular nucleus itself. This deep lesion, which the absence of a corresponding cortical lesion does not permit one to foresee, is due to the fact that the enclosed branch of the artery before terminating in the angular gyrus, gives rise to a series of small branches which pierce the supero-internal wall of the sylvian fissure and supply the white substance of the isthmus which joins the temporo-parietal lobe with the central ganglions; a certain number of these vessels go to the insula and to the external capsule and approach also the lenticular nucleus and even the internal capsule. The differences in the situation and extent of the regions affected by

the different modes of distribution of the blood-vessels in different individuals.

Finally, individual variations in the mode of distribution of the blood vessels will allow us in addition to make note of some very important facts. It must be admitted that frequently in cases of the aphasia of Broca, the intellectual deficiency although certainly present, is however, less marked than in the aphasia of Wernicke. A consideration of the type of softening which we now come to consider shows us the reason for this difference; since we see that in a certain number of cases it gives rise to lesions of the white substance in the neighbourhood of the temporo-parietal isthmus affecting only to a slight extent the convolutions of the area of Wernicke when the circulation of those parts for example is carried on by the other branch of the sylvian artery. And moreover a lesion even less extensive of the white substance of this zone and of the fibres proceeding from it towards the central nuclei, suffices to cause a sufficient amount of intellectual deficiency to constitute aphasia. This idea of aphasia produced by a lesion of the white substance of the left hemisphere permits of the understanding of the infinite clinical varieties produced by a combination of the aphasia of Wernicke more or less marked, with a greater or lesser degree

of anarthria following a lesion of the zone of the lenticular nucleus and spreading more or less into the white substance of the temporo-parietal lobe; or a lesion of Wernicke's area extending more or less into the zone of the lenticular nucleus. This gives rise to the so called mixed aphasias; but as a matter of fact Broca's aphasia is invariably a mixed aphasia with a predominance of anarthria. There is still the consideration of the individual varieties of distribution of the branches of the sylvian artery, which lets us see why, in certain cases of Broca's aphasia the third left frontal convolution participates in the softening (without that giving rise to any notable difference in the clinical picture).

As a matter of fact the branch of the 3rd frontal may take origin very clearly above the bifurcation of the sylvian artery; it may take its origin at the same point as the bifurcation so that there exists a real trifurcation, or finally the branch for the 3rd frontal convolution may arise after the bifurcation of the sylvian and in consequence on one of the branches of the bifurcation of this artery. The examination of this last method shows that the third frontal will also show softening even when the latter takes the deep form that is, for the parts situated behind the third

frontal convolution.

One can thus understand, says Marie, the reasons which have led to the perpetuation of the error that the 3rd frontal convolution has a special connection with the aphasia of Broca.

As regards cerebral haemorrhage - this may give rise to Broca's aphasia without lesion of the 3rd frontal, the condition extending more or less behind the region of the central nuclei. The commonest site for this being towards the temporo-parietal isthmus and the white substance of this lobe. When the haemorrhage remains absolutely limited to the region of the central nuclei anarthria is produced and not aphasia.

I will make a note here of two cases that have been published in support of Marie's views.

The first case[30] was shown on October 12th 1906 by M. Souques to the Société Medicale des Hopitaux.

The patient was a woman who for a long time had suffered from stenosis of the mitral valve and had been rendered hemiplegic on the right side and aphasic by an attack of apoplexy.

The aphasia in this case affected all the varieties of conventional language, but aphemia and

agraphia were the most noticeable. This patient died three years afterwards from pulmonary complications. On examining the brain one found a distinct area of softening situated in the posterior part of the 1st two temporal convolutions. The third left frontal was intact. On section the integrity of this convolution was equally evident. This centre of softening involved the posterior convolutions of the insula, the most posterior part of the two first temporal convolutions and extended to the postero-external part of the lenticular zone and the adjacent portion of the internal capsule.

The second case (31) was brought before the notice of the same society by M. Marie and M. Moutier.

The case was one of softening of the 3rd left frontal convolution, in the case of a right handed man 55 years of age, without the aphasia of Broca being produced.

On the brain being shown there were clearly seen two separate areas of softening. The one situated at the foot of the 1st temporal convolution, the other at the foot of the 3rd frontal and the two anterior convolutions of the insula, but without any distinct lesion of the subjacent white substance. This man had not suffered from Broca's aphasia but showed on the contrary a tendency to "verbigeration"

and also showed the symptom of coprolalia. These two latter symptoms were clearly due to the softening of the first temporal convolution. For the rest this patient articulated his words perfectly and did not show any sign of dysarthria. This being due to the fact that the lenticular zone was intact.

With regard to word-blindness, Marie admits that certain patients have a difficulty of reading which if not absolutely isolated, is at least definitely predominant. He explains this, however, by stating that in this case the lesion is situated in the area of the posterior cerebral artery and involves injury to the occipital visual centres with hemi-anopsia; and that this word-blindness may be complicated with aphasia if the lesion encroaches on Wernicke's area.

We may then summarise Marie's views on aphasia as follows:-

(1) That the true centre of language is the area of Wernicke and that Broca's convolution has no special function with regard to speech.

(2) That the area of Wernicke must be considered as an intellectual and not a mere sensory centre.

(3) That lesions of this centre give rise to

loss of the power of understanding spoken speech and also loss of the power of reading and writing.

(4) That Broca's aphasia is produced where, in addition to a lesion of the area of Wernicke, there is also a lesion in the zone of the lenticular nucleus.

(5) That Anarthria is characterised by the loss of the power of speech, with preservation of the comprehension of words, of reading and writing. It is due to a lesion in the lenticular zone which interferes with the co-ordination of the movements necessary for phonation and the articulation of speech without giving rise to true paralysis of the muscles.

Before proceeding to take up a critical consideration of Marie's views I shall go on to consider the results of Experimental, Histological and Embryological research with regard to the subject of Motor Aphasia.

(1) The motor area of the cortex:

In the first place before proceeding to consider the localisation of the psycho-motor centre under the above heads, we have to note that the old view which localised the motor area of the cortex in the region of the fissure of Rolando, more particularly in the convolutions bounding that fissure,

has been shown to need reconsideration. In 1901 Sherrington and Grünbaum[32] published the results of experimental work which they conducted on the various species of anthropoid apes. The results of their investigations show, that using uni-polar faradisation as the method of cortical excitation it was discovered that the responsive area was limited to the ascending frontal convolution.

The area included the whole length of this convolution, extending over its free width and continuously round into and to the bottom of the fissure of Rolando. The point to be noted is that their results show that the older idea which also included the post-central convolution in the motor area, was wrong. Sherrington and Grunbaum proved, that not only was this latter convolution silent under stimulation, even with relatively strong currents, but that destruction of it, or parts thereof, did not give rise to any motor paralysis. In addition, histological research bears out the results of the experimental evidence.

Thus Campbell has shown that the general motor area as marked out by the latest experimental work, can also be defined by a study of the histology of the parts in question. That is to say, Campbell has marked out an area of cerebral cortex which has distinctive histological characteristics, and this

area corresponds closely with that defined as the motor area by experimental physiology. It is unnecessary here for our present purpose to take up the detailed consideration of the histological arrangements or their more detailed boundaries. We have to note, however, that this area is termed the "pre-central" or motor area.

We now come to a very important part of the subject.

Adjoining this pre-central area Campbell has defined another area of cortex which is likewise possessed of distinctive histological characteristics and to which the name of "inter-mediate pre-central" area has been given.

In its general construction it bears a structural resemblance to the pre-central cortex, and Campbell holds that this area possesses functions closely allied to the motor area proper, and suggests that it is the part of the cortex concerned in the control of the various skilled movements executed by the motor cells in the pre-central area. It is also extremely interesting to note that this "inter-mediate pre-central" region includes the area of Broca.

I do not propose to go into the histological details but will look shortly at the distribution

of this type of cortex. Campbell gives this as follows:-

(1) "The cortical field to which the name 'inter-mediate pre-central' is attached ranges as a zone between 3.5 and 1 c.m. in width, placed after the manner of a buffer in front of the 'pre-central' area proper and showing an additional extension downwards on to the orbital surface of the hemisphere. Broadest above, the area becomes constructed at its middle and then expands again below."

(2) "Briefly put, it covers the base of the upper and middle frontal gyri, some of the ascending frontal (that not coated by the pre-central type), a considerable portion of the inferior frontal, including the pars basilaris (area of Broca), the pars triangularis (sometimes) and the pars orbitalis of the frontal operculum."

(3) "The calloso-marginal and transverse orbital fissures, at the upper and lower extremities of the area, respectively, form fixed limits, but the anterior boundary is not regularly determined by nuclei."

It may also be noted generally with regard to its histology that "many of the structural characters noted in the pre-central cortex are repeated, thus the general depth is preserved, the difference in regard to nerve fibres chiefly affects the degree

of fibre wealth, and save for the giant cells of Betz the cell lamination is remarkably alike. These resemblances suggest a physiological kinship between the two parts."

With regard to the Embryological evidence, we find that Flechsig (33) working on the basis of myelogenic development has described a field of cortex which he has called the "sensory projection centres". Campbell draws attention to the anterior portion of the great central field, and particularly to the more sparsely dotted zone lying in front of the ascending frontal convolution and its paracentral annexe. He says that the area which Flechsig indicates on the mesial surface of the hemisphere cannot be said to coincide with his; the field mapped out by the developmental method proceeds considerably further forwards and also extents on to the subjacent gyrus fornicatus; but on the lateral surface the agreement, so far as the anterior border is concerned is extra-ordinary; broad over the upper frontal gyrus, the area rapidly narrows at the level of the inferior pre-central fissure, and finally sweeps forward to cover the frontal and orbital opercula in just the same manner as Campbell's "inter-mediate pre-central type of cortex does.

We have now finally to consider what conclusions we are entitled to come to from a review of the large amount of rather conflicting evidence at our disposal with regard to the localisation of the cortical centres which are concerned in the production of spoken speech.

In the first place, let us look at the views of Marie and consider if we are entitled to accept his conclusions either in whole or in part.

As we have seen the first conclusion Marie asks us to accept is:-

(1) That the true centre of language is the area of Wernicke and that Broca's convolution has no special function with regard to speech.

I do not think that the evidence Marie produces to the effect that Broca's convolution has no function with regard to speech, justifies him in coming to this conclusion.

In the first place, he himself admits, that in 50 per cent of the recorded cases of motor aphasia there has existed a lesion of the 3rd left frontal convolution. It is also probable I think that a large number of the remaining 50 per cent of cases without lesion of the 3rd left frontal convolution

were cases of the type known as infra-pictorial motor aphasia.

Again there are cases of motor aphasia on record in which there has existed definite and circumscribed lesion of the 3rd left frontal convolution without any lesion whatsoever of the other parts of the brain. For example, in the 2nd case described by Broca himself, there was present a sharply defined and limited lesion involving only the area of Broca and extending to the posterior extremity of the 2nd frontal convolution. There are numerous other cases on record of this nature which show without any doubt whatever that the region of the foot of the 3rd left frontal convolution is at least intimately connected with the production of spoken speech.

Suppose, however, for the sake of argument that we adopt Marie's statement that the above convolution has nothing to do with speech, what theory have we to replace this view ?

Marie says that Broca's aphasia is produced by a lesion in the area of Wernicke coupled with a lesion in the zone of the lenticular nucleus.

It will be admitted that such a double lesion will give rise to serious disturbance of the power of producing spoken speech. The chances are, however, that in such a case the reception of spoken

speech, would be more interfered with than its production. Marie to my mind does not distinguish clearly between mere inability to articulate words and loss of the memory of how those words are produced.

It is a proved anatomical fact, that lesion of the region of the internal capsule or lenticular nucleus on one side does not give rise to loss of the power of producing words - that is, Anarthria, but merely produces a difficulty in proper articulation or Dysarthria. A lesion in both hemispheres may, however, as we have seen, give rise to complete Anarthria. There is, however, a class of case in which a single lesion in the white substance of the brain may give rise to loss of the power of speech; such a lesion, however, has been shown not to be situated in the internal capsule but between that structure and the cortex of the left frontal lobe. The only explanation one can give of this fact is that such a lesion destroys fibres proceeding from the centre controlling the production of speech to the internal capsule on the left side and to the oral articulative and vocal centres on the right.

Another fact which strikes one is that Marie does not give us a cortical area at all as the centre for the production of spoken speech. His theory, as we have seen, is that lesions of the area

of Wernicke give rise to loss of the power of understanding speech, and also loss of the power of reading and waiting. He does not say, be it noted, that such a lesion gives rise to loss of the power of producing speech. This latter being due he says to a lesion in the white substance of the brain. Such a lesion cannot destroy the centre controlling the production of speech, as there must be a cortical centre for such a function, but it may destroy the fibres coming from the psycho-motor centre, wherever it is situated, to the oral articulative and vocal mechanisms. This centre cannot be situated in the area of Wernicke otherwise a lesion of the cortex of that region alone would produce motor aphasia.

Accordingly we are forced to the conclusion from our previous evidence that the 3rd left frontal convolution is at least intimately connected with the function of speech.

What final conclusions can we therefore come to ?

It has been proved beyond doubt I think, that, in spite of Marie's evidence, the 3rd left frontal convolution has a very close connection with the faculty of the production of speech. There seems however, to be evidence tending to show that we

must take note more exactly of the structures involved in lesions of this area.

There are cases on record which show that a purely superficial lesion of the cortex of Broca's convolution may give rise to temporary motor aphasia, but that if the symptoms are to be lasting the lesion must not only affect the cortex, but also the subjacent white substance. That is to say we do not at present know how far lesion of the cortex in this area gives rise to the production of the interference with speech and how far it is due to underlying associating or centrifugal nerve fibres.

Campbell records a case which supports the view that the lesion must affect the subjacent nerve fibres.

The patient was a man who became affected with complete motor aphasia. This, however, passed off in the course of a few months. Twelve years later the patient again came under observation suffering from alcoholic insanity. He had, however, no speech defect and had not been troubled with a recurrence of the aphasia during these twelve years. He died from an abdominal affection, and at the post-mortem there was found an old standing patch of softening in the left hemisphere. The lower two thirds of Broca's convolution were completely destroyed but on making sections of the brain, it was found that

the destruction did not extend inwards beyond the plane of the surface of the insula, the white substance anterior to the lenticular nucleus and internal capsule was, therefore, intact. It is interesting also to note, that in the 2nd case published by Marie and Moutier[31] which they showed as evidence supporting the view that there may be lesion of the 3rd left frontal convolution without Broca's aphasia being produced, that in this case the lesion was confined to the cortex and did not affect the white substance. What is the explanation of this ? It seems to be possible I think that the psychomotor speech centre is more widely extended in the area of cortex described histologically as the intermediate pre-central area than we have hitherto supposed, and that, therefore, if a lesion is to have the effect of producing lasting motor aphasia it must not only destroy the cortex in the region of Broca's convolution to a greater extent than we have hitherto considered necessary, but must also cut across all the fibres proceeding from this area of cortex to the various motor-executory cells concerned in the complicated movements necessary for the production of speech.

Let us now look at Marie's views with regard to the localisation of the centre for the reception and interpretation of spoken speech.

He states that the area of Wernicke is the centre concerned in this function, and this to a certain extent agrees with current opinion. But we find that instead of ascribing to the 1st two temporal convolutions the pure function of a word-hearing centre, he goes much further and affirms that the area of Wernicke is not merely a sensory, but an intellectual centre.

In addition to including in its functions the power of interpreting spoken speech, he also states that lesions of this centre give rise to loss of the power of reading and writing and of certain didactic accomplishments. Marie lays great stress on the fact that in cases of interference with the power of interpreting spoken speech, there is a marked diminution of the intellectual capacity in general, and that the difficulty the patient shows is not due to word-deafness merely, but to defective comprehension.

It is readily understood, of course, that a patient unable to understand what is said to him should have his inability put down to defective comprehension, and as a matter of fact, this is so. The patient has got defective comprehension of spoken

language but this is not because his "intelligence" is affected but because the centre in his brain concerned in the special function of the interpretation of spoken speech, is destroyed, or cut off from its communications with the primary auditory centres. The case of word-deafness recorded by Giraudeau and quoted by Professor Wyllie to which reference was made in a previous part of this paper affords strong proof of the fact that it is possible to have cases of word-deafness without diminution of the intelligence. In that case the patient, it will be recalled, was a widow, aged 46, who was quite word-deaf. To all questions she answered "what is it you say"? "I don't understand you? cure me". Hearing and sight were intact. She read well and answered questions which were written and shown to her. At the post-mortem"there was found a tumour the size of a walnut, in the posterior part of the left temporal lobe. Above it reached the fissure of Sylvius; below, it involved three-quarters of the breadth of the second temporal convolution. It reached forward to within three centimetres of the anterior extremity of the temporal lobe, backwards, to within one centimatre of the posterior extremity of the fissure of Sylvius. It encroached slightly on the white matter and was easily enucleated". This case is a very valuable one. It not

only shows that a lesion of that part of the cortex of the temporal lobe which we concluded in our study of the subject was the centre for the reception of spoken speech, does give rise to the condition of word-deafness but also shows that this condition can definitely exist without there being any interference with the intellectual faculties in general.

Marie holds that further proof of the affection of the intellect in cases of aphasia is afforded by the fact that in some cases of Broca's aphasia the patients were unable to understand all that was said to them.

That this is so is admitted. The patient, even in cases of motor aphasia, may have difficulty in understanding the meanings of certain words and more particularly of concrete things. This, however, cannot be held as proving that this is due to an interference with the general intelligence. As Professor Wyllie(11) has pointed out, the meaning of words is in direct connection with both the auditory and motor images, and destruction of either of these centres, termed by him the "Primary Couple" is capable of causing a difficulty in interpreting the meaning of words. Though when the motor images are destroyed the interference is not so marked as in the other case. This is because the auditory word images revived by spoken speech are usually able to call up

in the mind the meanings of words without help from the motor images. It is also to be remembered that any severe lesion of the brain, may amongst other effects, involve the speech centres and may also give rise to loss of memory and other cerebral symptoms. These, however, must be differentiated from the results of pure lesions of the speech centres. A case of concussion of the brain, for example, may give rise to some interference with the powers of speech, or may cause the patient to forget the names of things or persons. These symptoms being due to disturbance of the faculties of the brain as a whole. Further, Marie seems to regard the fact that in some cases of aphasia, both motor and auditory, there may be interference with the musical faculty of the patient, or that in other cases there may be loss of the power of understanding or producing gesture language, as additional proof that in these cases there is a siminution of the intellectual faculty in general. This, I think, we shall have little difficulty in seeing presently is due, not to defective intelligence, but to <u>involvement of the specialised centres concerned in these functions</u> in the lesion responsible for the defect of speech.

From a consideration of the above, therefore, I do not think that we are led to alter our convictions with regard to the localisation of the centre for the reception of spoken speech, obtained from the study of the clinico-pathological and histological evidence. We, therefore, conclude that this centre is situated in the posterior part of the 1st two temporal convolutions that it is primarily a word-hearing and not an intellectual centre, and that this area of the cortex is probably also the centre for the interpretation and elaboration of other auditory stimuli such as those of musical sounds.

Marie's views regarding the localisation of the word-seeing centre.

We have seen that Marie gives a very wide extent of cortex as the probable centre for the reception and interpretation of written and printed speech. He says that interference with this function is caused by lesion of the area of Wernicke. This area includes, as we have seen, in addition to the posterior part of the first two temporal convolutions, the angular and supra-marginal convolutions.

Now Marie does not attempt to subdivide this extensive area of cortex into smaller areas and locate in them specialised functions. On the con-

trary, he not only ascribes to lesion of this large area the aphasia of Wernicke, but in addition says, that lesion of this area also gives rise to interference with the power of reading and writing.

On the face of it, it is improbable that such an extensive area, comprising, as we have seen from the histological evidence, various distinct types of cortex, should be considered as a homogeneous centre controlling not only the higher intellectual faculties, but also the power of understanding spoken speech and also the power of understanding reading and writing.

Although it is somewhat difficult from the nature of the case to be absolutely certain that there is not some impairment of the intellectual faculties in a case of auditory aphasia, yet, when we come to look at the condition of word-blindness, we have as we have seen in preceding parts of this paper undoubted evidence to prove that word-blindness may exist by itself without any affection whatsoever of the general intelligence. Marie's explanation of word-blindness is that it is produced by a lesion in the area of the posterior cerebral artery involving injury to the occipital visual centres with hemianopsia, and that word-blindness may be complicated with aphasia if the lesion encroaches upon Wernicke's area.

We have seen, however, in our consideration of visual aphasia that hemianopsia due to destruction of one occipital visual centre does not result in word-blindness. Destruction of both occipital visual centres produces complete blindness and not merely word-blindness. Again, with regard to the power of reading and writing how can Marie explain the remarkable fact that a patient may be able to write correctly and at the same time be unable to read what he has written ? This fact seems to me to afford very strong evidence against the supposition that one controlling intellectual centre, situated somewhere in the area of Wernicke, is responsible for these faculties, and that it also points very strongly to the existence of separate specialised centres for these functions.

If we analyse our cases of word-blindness we are immediately struck by the fact that when there has existed a lesion situated in the angular gyrus the patient has invariably exhibited the symptom of word-blindness, and in addition has lost the power of writing.

Further, that a lesion in the white fibres conducting impressions from the primary visual centres to the angular gyrus gives rise to word-blindness without loss of the power of writing. These facts, I think, afford us good grounds for concluding that

the centre for the reception and interpretation of written and printed speech is situated in the region of the angular gyrus. Other varieties of visual stimuli are probably elaborated in that part of the cortex which has been described under the term "visuo-psychic".

The localisation of the centre for the production of written speech.

This centre is called the graphic-motor centre. Some authorities have questioned the existence of such a centre. They explained the loss of the power of writing that was so frequently present in cases of aphasia by saying that as the patient could not remember words owing to amnesia verbalis it was impossible for him to be able to write them. This explanation is right in so far that the symptom of agraphia is present in many of the varieties of aphasia, but it is wrong, as we shall see, in so far as it denies the existence of a special centre for the control of the production of written speech. In his experimental work Ferrier locates the cortical centre for movements of the hand in the middle of the ascending frontal and ascending parietal convolutions opposite the posterior extremity of the second frontal.

As a result, however, of the recent work of Grünbaum and Sherrington previously referred to, we have seen that the motor area is not so widely extended as was formerly supposed, and in consequence we have now to localise the centre for the hand in the middle of the ascending frontal convolution only. The hands are unilaterally represented in the cortex that is to say there is a centre for each hand in the opposite cerebral hemisphere.

This centre, however, is not the part of the cortex which has stored in it the memory of the complicated movements requisite for writing, any more than are the oral articulative centres the real centres for the production of spoken speech.

It is true that we are able to write more or less with both hands, and this seems to point to the possibility of the existence of a centre for writing in both hemispheres; but when we come to consider the matter closely we see at once what a difference there is between the writing produced by the hand trained for the purpose as compared with that produced by the other. When we write with the left hand we laboriously and slowly copy the visual image of the letters we are writing. We are writing. We can do this if we attach a pencil to any movable part of the body. But what a

difference there is between the easy and purpose-like way in which we can write with the right hand, as compared with the manner in which we write with the left.

It is more than mere practice which leads to this facility in writing. It is true, however, that if one practises writing with the left hand, there is also gradually evolved a controlling centre in the right hemisphere which exerts the same function as that on the left.

Let us here look briefly at the effects produced by the different varieties of aphasia on the power of writing.

(1) Lesion of the auditory word centre:

In this case the patient in spontaneous writing shows the same defects that he exhibits in his speech. That is to say, his writing shows the symptom known as Paragraphia.

Further as the patient cannot understand words spoken to him, he is unable to write to dictation.

(2) If the lesion is in the psycho-motor speech centre:- the patient is able to write very little.

Gairdner (34) puts the state of affairs shortly as follows:- "The aphasic writes at least as badly as he speaks; and when he speaks not at all, he

writes not at all".

There are, however, cases of motor aphasia in which the power of writing is preserved.

In these cases the lesion is a sub-cortical one and does not interfere with the fibres connecting the psycho-motor speech centre with the centre for writing.

(3) Lesion of the visual word centre: Gives rise to marked loss of the power of writing. The patient in such a case is either not able to write at all or else he writes very badly.

This fact shows that if there is a special graphic centre, this centre cannot perform its work properly without the aid of the visual word centre. This, it seems to me, may be due to the fact that since these centres are joined by connecting fibres and always work together, that destruction of one of them may give rise to great interference with the powers of writing.

Just as in the case of the psycho-motor and auditory word centres lesion of the auditory word centre damages the powers of producing speech and gives rise to the symptoms of amnesia verbalis, articulative amnesia, and paraphasia. As regards the power of copying; in this case the patient may be able to copy words as he would a drawing but

cannot copy printed matter into writing or vice versa.

(4) If the lesion is situated so as to cut across the fibres proceeding from the cortical visual centres to the visual word centre (that is in infra-pictorial visual aphasia), the patient will be able to write voluntarily and to dictation but will not be able to copy perfectly.

Lastly, it must be stated that, so far as I can find, there is no recorded case of a lesion giving rise to uncomplicated interference with the power of writing. This has been held to prove that a specialised graphic-motor centre does not exist.

This, however, I think can be shown to be incorrect.

Let us look briefly at the subject of what is known as "Mirror writing".

Mirror writing is produced when the patient writes from right to left instead of from left to right. It is easily read when looked at in a mirror.

Elder (35) holds that the phenomenon of mirror writing proves the existence of a special graphic motor centre. For this reason, that "if the homologous muscles were acted on in the same way in the

left hand as would have been the case if the impulses had passed to the motor centres for the movements of the right hand, then the left hand would move in the opposite direction to the right, in its relation to the mesial line of the body - that is, all movements from side to side would be in an opposite direction, whilst antero-posterior movements would be in the same. In other words, adduction movements on the right side are in the opposite direction to adduction movements on the left, and abduction on the right to abduction on the left."

Elder also points out that although the representation of the hands in the cortex is unilateral, yet it is easier for them to act in the same way in movements similar to each other, such as in twirling the thumbs, than to act in the opposite direction.

This seems to point to the conclusion that there are commissural fibres joining the centres for the hand, and these fibres probably make it earier to make homologous muscles act in the same, rather than in the opposite direction.

These facts then tend to prove the existence of a special graphic-motor centre.

What evidence have we then as to the exact localisation of this centre.

From the fact that there is no authenticated case of pure graphic-motor aphasia we might be led to believe that this centre is not pathologically

separable from the centres for the hand.

In 1881, however, Exner (36) stated that the graphic-motor centre was situated in the posterior extremity of the second frontal convolution, and it is acknowledged that in a great number of cases of agraphia there has existed a lesion of this area.

It has to be remembered, however, that in these cases there has also as a rule been a lesion of Broca's convolution, and as Lichtheim has pointed out, in such cases where recovery takes place, the power of speaking is always recovered before the power of writing, and that it is possible that the agraphia which persists may not be due to lesion of the centre above-mentioned but to lesion of Broca's convolution.

Exners localisation of this centre is, however, supported by the histological study of the parts in question.

If we again look at the work of Campbell we find, that just as Broca's convolution was included in the type of cortex designated as the intermediate pre-central type, so also is the foot of the 2nd frontal convolution included in this area.

And further it is very significant that this part of the intermediate precentral cortex is situated exactly on a level with that area of the pre-central or motor cortex which has been shown

experimentally to be the centre for the muscles of the hand.

From a consideration then, of the whole subject, I think we are entitled to conclude, that the posterior extremity of the second left frontal convolution is the centre for the graphic-motor images used in writing.

Before bringing this paper to a close I should like to note briefly what conclusions we can come to with regard to the localisation of two functions closely allied to that of speech, I refer to the Musical Faculty and to Gesture Language.

The Musical Faculty:

A theoretical study of this subject entitles us to assume I think, from its close relation to the faculty of speech, that there are the following centres concerned in the reception and production of music in its various forms.

(1) A centre for the reception of musical sounds in their various forms.

(2) A centre for the production of musical sounds by the voice, that is singing.

(3) A centre, or centres, for controlling the production of musical sounds by means of the various musical instruments.

(4) A centre for the reception and interpretation of musical notation.

(5) A centre for the writing of musical notation.

In the first place we have to note that disturbance with the faculty of music is generally accompanied by some interference with the mechanism of speech.

Edgren has made an analysis of 52 cases of interference with the musical faculty and shows that these cases can be divided into three groups.

(1) Cases in which there was some form of aphasia present, but no amusia.

(2) Cases in which in addition to one or more forms of aphasia there was also one or more forms of amusia.

(3) Cases where there was present some form of amusia but no aphasia.

Out of the 52 cases Edgren found that

of type (1) there were 24 cases.

of type (2) there were 22 cases.

of type (3) there were 6 cases.

This shows that there were very few cases where there was amusia without some form of aphasia.

This naturally leads us to expect that the centres for these different functions are situated near each other.

Let us look briefly at each centre in turn with regard to its localisation.

(1) The centre for the reception of musical sounds in their various forms.

Edgren describes a case which is of interest in this connection.

In it the patient was rendered unable to appreciate musical tones. At first there was also word-deafness but this disappeared, leaving the tone-deafness.

At the post mortem there was found a lesion of the anterior two thirds of the first, and anterior half of the second left temporo-sphenoidal convolutions.

Bernard has described a case of tone-deafness which is quoted by Edgren and in this case the lesion was situated in the same position as the above.

This tends to show that the centre for the reception of musical sounds is situated in the anterior two thirds of the 1st and the anterior half of the 2nd temporal convolutions on the left side. Now it is an interesting fact that this area is included in that part of the temporal cortex which we came to the conclusion from the histological evidence was concerned in the interpretation of various auditory stimuli and which was described under the

term "audito-psychic" area. There are, however, cases on record in which with lesions affecting this part of the left temporal cortex there has been no evident interference with the reception of musical sounds. This leads us to suppose that the opposite hemisphere may probably also be connected with this function.

The centre for the production of singing:

In this connection we have to note:-

(1) That a patient unable to speak may yet be able to hum a tune correctly without words.

(2) That there are cases which illustrate the striking fact that a patient, totally unable to speak, may yet be able to sing a song with the words correctly and distinctly.

The fact that a patient may be able to hum a tune, although he cannot sing the words, shows that the centre concerned in the control of the laryngeal movements necessary for humming a tune has not been involved in the lesion giving rise to the aphasia.

The fact, however, that a patient affected with motor aphasia may yet in rare cases be able to sing a song, with the words correctly, is a very puzzling one.

Professor Wyllie makes mention in his work on "The disorders of Speech" of a case of this kind,

and suggests that the condition may be due to the action of the "uneducated" cortex on the opposite side.

While house-physician in Professor Wyllie's wards in the Royal Infirmary, I had the good fortune to have, in the female ward, a woman aged 30 who was affected with right-sided hemiphegia and motor aphasia. This patient had no word-deafness, she understood everything that was said to her and could read fairly well. She had absolute loss of the power of speech except for the recurring utterance "in" which she always said when asked a question. The curious fact about the case, however, was that if part of the song "The Anchor's weighed" was sung to her she could join in and also sing the song, producing both the tune and the words correctly though the latter were evidently produced with a slight effort. On trying to get her to speak the words of the song immediately after singing them, it was found that she could not do so. This patient, however, by means of training with the physiological alphabet was in the course of a few weeks after her admission to the Hospital able to pronounce several sentences correctly. She left the Hospital able to speak a fair number of sentences spontaneously and correctly.

What are the possible explanations of this interesting case.

It seems to me that there are three possibilities.

(1) That the opposite hemisphere may in some cases possess the centre for storing the psycho-motor images of the movements necessary for singing.

(2) That there is another psycho-motor centre in the left hemisphere distinct from that for ordinary speech having the sole function of controlling the movements necessary for musical speech.

(3) That the lesion in such cases is situated so as to leave the psycho-motor speech centre itself intact, but to involve the nerve fibres carrying the impulse for spoken speech from this centre to the oral articulative and vocal centres - thus producing the motor aphasia; whilst it leaves unaffected other fibres passing from this psycho-motor speech centre and conveying the impulses for musical speech to the oral articulative and vocal mechanisms.

What the real explanation of this fact is we do not at present know. Further investigation on the subject is needed.

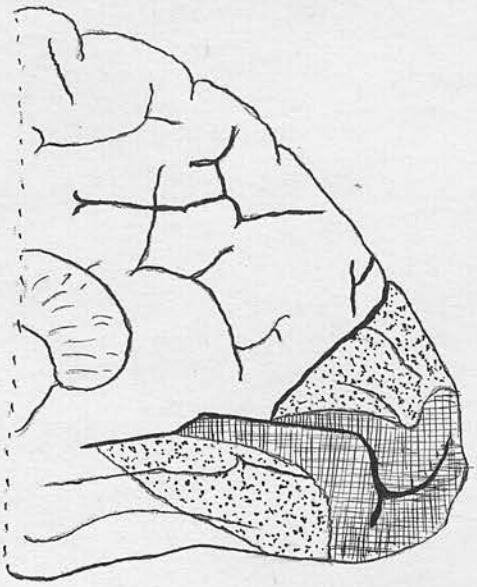
The centre or centres for controlling the production of musical sounds by means of the various musical instruments.

With regard to this subject it seems to me to be very probable that the centres controlling the movements necessary for the playing of any of those instruments are situated in the area of cortex possessing the "intermediate pre-central" type of obstructural arrangement at a corresponding level to that of the motor centre in the "pre-central area" of the cortex, responsible for the execution of the particular movements required.

The same explanation may be given with regard to the centre for controlling the writing of musical notation.

The centre for the reception and interpretation of musical notation.

We have seen in our cases of word-blindness, that in some of them the patient retained the power of reading arithmetical figures when he was quite unable to read words. This shows that either the visual images of arithmetical figures are stored in both hemispheres, or else in a part of the cortex separate from the word seeing centre. The same condition may be produced with regard to the reading of musical notation and it seems probable that the



Drawing to illustrate the distribution of the visuo-psychic and visuo-sensory areas. (after Campbell.)

visual images for this function are stored in the area of cortex in the occipital lobes which we studied under the term " visuo-psychic" area.

The centres for the interpretation and production of Gesture language.

We have seen from our previous studies of disturbances of the faculty of speech that although a payient may be unable to speak, yet he can as a rule, express himself, more or less clearly, by means of gesture language. On the other hand, an aphasic patient may exhibit loss of the power of understanding the gesture language of others.

Others again misunderstand the meaning of the conventional signs used for this purpose, such as the shaking of the head as an indication of negation.

Lastly some patients may produce the wrong gesture, shaking their head when they mean "yes" for example.

From the fact then, that a lesion confined to one side of the cerebrum, is sufficient to produce an interference with the faculty of understanding or producing gesture language it is evident that the centres concerned in this function are, for the most part, situated in one hemisphere.

The localisation of the centre for the interpretation of Gesture language.

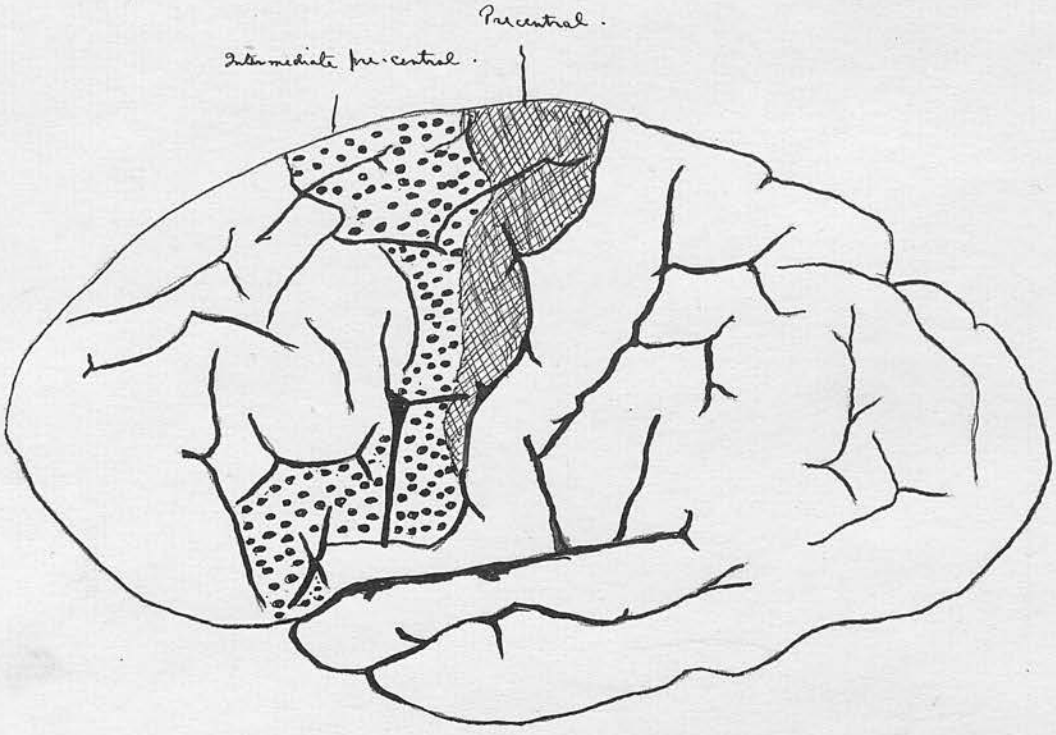
This centre must be, I think, a highly specialised one. For this reason, that though the most simple signs used for the purpose such as nodding or shaking the head, or beckoning with the arms, may be very readily understood, yet we have to remember that some of the signs used in this method of conveying thought are extremely delicate and complicated.

The interpretation of the complicated movements of the lips in speaking as is accomplished in the case of trained deaf persons, shows that the centre for the interpretation of these signs must be quite as highly specialised as the ordinary word-hearing centre. The various other combinations of movements connected with the expression and interpretation of thought as for example, by means of facial expression, or by the fingers as in deaf-mutes must also be received and elaborated in this centre.

In what part of the brain then is this centre situated ?

It seems very probable,I think, that the centre for this function is situated in the region of the occipital lobe possessing the visuo-psychic" type of cortex.

Our previous studies have shown us that this area is in all probability connected with the



Drawing showing the distribution of the pre-central and inter-mediate pre-central types of cortex. (after Campbell.)

function of the interpretation and elaboration of various visual stimuli. This centre may be partly located in both hemispheres but there is evidence to show that to a large extent at least it is situated on the left side of the cerebrum.

The centre for the production of the language of gesture.

From what has been said with reference to the production of music by means of the various musical instruments, and our conclusion as to the localisation of the centres concerned in these functions, we are here also led to the conclusion, that the higher centres concerned in the co-ordination of the movements required for the illustration of thought by means of gesture language are situated also in that region of the brain which is possessed of the type of cortex which we have seen reason to believe is connected with the control of the centres in the motor area proper, and which has been described as possessing the "intermediate pre-central" type of cortex.

REFERENCES.

(1) Schenkins: Trousseau Clin. Med. Vol.I. p.240.

(2) Bouillaud: Treatise on Encephalitis.

(3) Broca: Bulletins de la Societe Anatomique August, 1861.

(4) Trousseau: Clin. Med. Vol. I. New Sydenham Soc., 1867.

(5) Russell: The Disorders of Speech, Wyllie, 1894.

(6) Wernicke: Der Aphaische Symptomen complex, 1874.

(7) Ferrier: Med. Chir. Trans. Vol.LXVII. 1884.

(8) Lichtheim: Brain, Jan. 1885.

(9) Dejerine: Compt. Rendus de la Soc. de Biolog. 1891 and 1892.

(10) Pierre Marie: La Semaine Medicale, 1906, pp.241, 492 and 555.

(11) Wyllie: The Disorders of Speech. 1894.

(12) Campbell: Histological studies on the localisation of cerebral function, 1905.

(13) Serieux and Mignot. Abstract in neurology Centralblatt p.495, 1901.

(14) Mills: Brain p.468, 1891.

(15) Kaufmann: Berlin-klinwochenschr. p.540, 1886.

(16) Ferguson: Journal of Anatomy and Phys. p.292, 1890.

(17) Giraudeau: Rev. de Medo. II. 1882, p.446.

(18) Ferrier: Philos. Trans. Part II. 1884 & 1895 and Brain 1888.

(19) Sanger Brown: Brain, Jan. & April, 1888 and Prac. of Phys. Soc. No.2, 1887.

(20) Henschen: Klinische und pathologische Beitrage zur Pathologie des Gehirns, 1890, 1892, 1894.

(21) Sequin: Contribution a l'etude de l' hemianopsie d'origine corticale Arch. de neurol. Tome XI. 1886.

(22) Vialet: Les centres cerebraux de la vision.

(23) Serieux: Compt. rendus de la Soc. de Biolog. 16th Jan. 1902.

(24) Serieux: Comptes rendus Soc. de Biolog. Dec. 1891.

(25) Horsley and Semon: Ibid.

(26) Risien Russell: Brit. Med. Journal August 1895.

(27) Horsley and Beevor: Phil. Trans. of Roy. Soc. Lond., 1890.

(28) Broca: Bulletins de la Soc. Anatom. de Paris.

(29) Ogle: St. George's Hospital Reports, Vol. II. p.105.

(30) Souques: La semaine medicale, 24th Oct.1906 p.514.

(31) Marie and Moutier: La semaine medicale, 21st November 1906, p.563.

(32) Sherrington & Grunbaum: Observations on the Physiology of the cerebral cortex of some of the higher apes Pro. Roy.Soc. Vol. LXIX, 1901.

(33) Flechsig: Gehirn und Seele, Tafel IV. 1896.

(34) Gairdner: Proc. of the Phil. Soc. of Glasgow 1865 - 1868 p.104.

(35) Elder: Aphasia and the cerebral speech mechanism.

(36) Exner: Untersuchungen uber die Localization der Functionen in der Grosshirnrinde des Menschen, 1881, p.57.

(37) Edgren: Amusie Deutsche Zeitschrift fur nervenheilkunde VI. 1895.

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