"A STUDY IN APHASIA".

CONTENTS.

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INTRODUCTION.

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The Cerebral localisation of the faculty of speech is a subject which has only engaged serious attention within the last half century. The credit of having accurately located the centre for articulate speech in the 3rd. left frontal convolution is generally ascribed to M. BROCA, who in 1861 gave the result of his primary researches on this subject. Previously to this , however,more than one observer had though in a more general way, directed attention to the same region. BOUILLAUD, in 1825, basing his views upon observations in 114 cases of disease of the frontal lobes, affirmed that these were the parts principally concerned in the production of language. MARC DAX, in 1836 , pointed out the frequency of loss of speech in association with right sided paralysis and came to the conclusion that the organ of language is situated in the left hemisphere of the Brain near to the Island of Reil. These two observers , therefore , made valuable contributions to the ultimate solution of the problem. The subject,however, did not receive much more attention until 25 years later. In 1861, M.BROCA, who had made careful record of the now historical cases of Leborgne and Lelong, precisely defined the posterior part of the 3rd. left frontal convolution as the cerebral centre for articulate speech. His observations have since been fully confirmed both in his own and other countries, and in honour of this teacher the region of this particular centre is often referred to as " Broca's Convolution "

To the clinical condition produced by loss of the cere-

bral faculty of speech , the term APHEMIA (aphémie) was applied by M. Broca ; this designation had however been since superseded by the term " APHASIA " (aphasia) suggested by M. Trousseau,while Aphemia has a more restricted meaning as I shall afterwards point out.

Aphasia is now recognised as of two kinds - " Motor " and " Sensory " - according as what Ross terms the " emissive or " apperceptive " departments of speech are at fault. In motor aphasia the patient may understand perfectly both written and spoken language, bur has himself lost the articulate mechanism of speech; such a condition would be produced by a lesion in Broca's area. "Sensory Aphasia " is of differents kinds according as it affects the auditory or visual word centres. " Word Deafness " is the term applied to those cases in which the person cannot comprehend spoken speech although for actual sound the hearing is perfect. " Word Blindness"" is the term applied to those cases of Aphasia in which the person cannot comprehend the meaning of written or printed speech although for form the vision is as good as ever. It is now known that in these cases of Sensory Aphasia the lesion is situated in the cerebral area supplied by the posterior branches of the Sylvian Artery ; thus a lesion of the Angular Gyrus will produce " word - blindness " while a lesion of the upper tempero-sphenoidal regions (" Wernicke's Convolution ") will produce " word-deafness ".

In cases of gross lesion we may find various combinations

of Motor and Sensory Aphasia ; and in cases of injury to the subcortical speech tracts or commisural fibres we may find aphasic symptoms of a complex and even baffling character. From what I have said in these brief introductory remarks it will be rightly inferred that the subject of Aphasia is a vast one. In a Thesis of this kind one can only pretend to touch a fringe of the subject ;and even that , I feel only too conscious in my own case, but imperfectly at the best.

I wish more particularly in this paper to draw attention to some of the " TRANSITORY MANIFESTATIONS "of Aphasia. Under this term I include not only cases of " Functional " Aphasia in which the loss of speech is usually of a very temporary character, but also some Organic types of the disease in which a rapid recovery is by no means infrequent. A knowledge of such transitory manifestations is a somewhat necessary equipment from the " prognostic " point of view ; and I purpose making the cases quoted here a basis for an incidental discussion of the factors which determine recovery in cases of Aphasia. Most of the cases which I give in illustration of the subject have been personally met with in my own practice ;all of them will I trust prove intrinsically interesting, and some of them,possibly, merit more than passing notice. Cases of Aphasia occurring as an Initial symptom of Tubercular Meningitis, and cases occurring during the puerperal period (" Puerperal Aphasia ") appear from the literature of the subject to be somewhat infrequent. I would also draw attention to the case of " Amnesia for Proper Names of Persons "which presented

features of a somewhat unique character, and which may possibly throw further light on the existence of a separate " Grapho-Motor Centre ". With these remarks I at once proceed to the discussion of my subject under the following sections :-

1. Transitory Motor Aphasiae of "Functional" Origin

2. Transitory Motor Aphasiae of "Organic" Origin.

3. Transitory Sensory Aphasiae of "Functional" and "Organic" origins with special reference to some cases of " AMNESIA VERBALIS ".

(Section 1. over)

TRANSITORY APHASIA IN EPILEPSY.

In discussing the so called " functions
of the Brain which give rise to Aphasia. Now
" most common functional [illegible]
" the condition which [illegible]
" in some cases the [illegible]
" of a sudden inability to speak [illegible]
by will come out [illegible]
also the follow[illegible]
[illegible]
[illegible]
seizure, but also a very slight & [illegible]
[illegible]

SECTION 1.

ON TRANSITORY MOTOR APHASIAE of FUNCTIONAL ORIGIN

S. W.(with illustrative cases)

[illegible] the age of 18. I saw him for the first time on [illegible] when he was brought into my surgery in a fit and I had [illegible] an opportunity of making further enquiry into the nature of his condition.

The " aura " or warning in this case invariably took the form of an inability to speak - at first partial and later complete. His friends at once notice an unnatural ness come over him. He himself feels the warning not only on account of his inability to speak but also because he then finds himself beginning to think very vividly of some previous event in his life[illegible] usually football or cricket match in which he played [illegible]

TRANSITORY APHASIA IN EPILEPSY.

In discussing the so called " functional " affections of the Brain which give rise to Aphasia, Ross remarks " The " most common functional lesion that gives rise to Aphasia is " the condition which precedes or follows an epileptic attack. " in some cases the warning of an epileptic attack consists " of a sudden inability to speak " Almost any case of Epilepsy will bear out the former part of this statement ;but I give the following instance as illustrating not only a condition of aphasia preceeding and forewarning the convulsive seizure, but also a very distinct condition of Amnesia following on recovery.

F. W, aet 17 years, has been subject to epileptic fits since the age of 13. I saw him for the first time on 21st. April 1899, when he was brought into my surgery in a fit, and I had then an opportunity of making further enquiry into the history of his condition.

The " aura " or warning in this case invariably takes the form of an inability to speak - at first partial and later complete. His friends at once notice an unnatural quietness come over him. He himself feels the warning not only on account of his inability to speak but also because coincident with this he finds himself beginning to think very vividly of some previous event in his lifetime - usually a football or cricket match in which he himself plays a prominent part. At this stage, however, he his still quite conscious of what is going on around him, and often tries but unsuccessfully , to battle against and shake off the aproach-

-ing seizure. For instance he may keep on determinedly at his work, but if this is anything of an engrossing nature he has to giveit up. He is quite able to walk about and has a rule plenty of time to make for a place of safety. As an immediate precedent to the convulsive seizure his head his drawn round towards the left side, and he falls over unconscious.

The following is the history of the attack on 21st. April when I first saw him. He was bowling for his side at a cricket match at Higham Hill , Walthamstow, when he felt the usual aura of an inability to speak. He immediately threw the ball to the ground, and without any possible explanation to his companions left the field and made straight for home. He does not remember passing certain well known landmarks on the way ;but it is certain he walked over mile and a half before theactual " fit " brought him to the ground. A man walking behind him stated that he was going along steadily and at a fair pace till he suddenly fell over unconscious close to our Surgery. I saw him while yet in the epileptic " " fit " which lasted about ten minutes. He then recovered his senses sufficiently to sit up and drink a glass of water , but remained slightly dazed and distinctly amnesic. He could remember neither name or address although he volunteered the information that he had been at a cricket match and added that he should be allright presently. In about half an hour's time he thought he could find his way home although he still failed to remember the address and could not give his name.

He started off under the care of two constables and walked briskly and without help for about three hundred yards. The trio then came to a dead stop as the lad did not seem to remember the streets. The inevitable crowd gathered but fortunately a passing lady recognised the lad and sent word to his father. Before the arrival of the latter, however, the patient's memory had returned and he was able to write down his name and address . By this time close on an hour had elapsed since recovery from the actual " fit ". After reaching home he went off into a sound sleep from which he awakened with speech and memory both perfect.

It will be noted therefore that in this case the convulsive seizure was preceded by an aura of Aphasic nature and followed by a distinct period of Amnesia. The Aphasia and Amnesia were concomitants of the status epilepticus and did not exhibit themselves at any other time ; the case therefore may be taken as exemplyfying a Transitory Aphasia of Functional Origin.

I ought to add that although in cases of Epilepsy the speech usually returns soon after the recovery of consciousness, Kussmaul remarks that the Aphasia may persist for " hours, days, or weeks at a time ".

ON TRANSITORY APHASIA IN MIGRAINE.

Transitory Aphasia is well known as an occasional associate or Megrim or Migraine. The Migraine may be of what is called the " accompanied type ", i.e.the headache is accompanied by a paresis of one side of the body or by ophthalmic phenomea of various kinds: but in this form of migraine the occurrence of aphasia, although uncommon , is not so rare as in what may be termed the"unaccompanied type" when ophthalmie and other phenomena are absent. Of this latter form I lately came across a case in which the loss of speech was so marked as to be indeed the outstanding feature of the man's illness ; and I give it here as a good example of the occurrence of a Transitory Aphasia during an attack of Megrim or Migraine. (notetaken Feby.1900.)

A. R. aet 35 years, is a man of good physical constitution and temperate habits, and is employed in one of the large City Banks in a position of some responsibility. Of late he has had additional stress of work and felt more than usually worried about business. He has suffered for some time from periodic attacks of headache, but never of such a severe character as the one I am about to refer to.

On Friday,February 3rd. he began to complain of dull frontal headache , more severe on the right side and latterly most intense in the region of the right temple. The pain asserted itself so much on Monday 5th.February. that he was compelled to stay home from business. About 11 P.M. on this date he was sitting in the easy chair & talking to

his wife with his usual freedom of speech;He had no ophthalmic disturbances of any kind. All at once his wife noticed him put his hand up to his head as if in severe pain, and he began to stutter in the middle of a sentence. He made several fruitless attempts to express himself further although as his wife says " the words were on the tip of his tongue ! He remained quite speechless for 2½ hours - from 11.30till 2 A.M. when he fell asleep. I saw him in the latter portion of this interval and found an aphasia such as his wife had described. There was no paralysis nor paresis of any kind , and the tongue was protruded in a straight line. There was no loss of sensation. He was quite conscious but slightly dazed and distressed. He apparently understood what was said to him but could only respond by signs.The face was flushed and the pupils dilated.

I advised him to get off to bed as soon as possible and he was able to walk upstairs and undress without help. A draught of Bromide was administered and evaporating lotions applied to the head. He slept soundly and next morning awakened to find that speech had returned. When I saw him later in the forenoon he told me the headache was much better, but that he now feels exactly as if he had received a severe blow on the head the day previous. In the afternoon a severe attack of sickness supervened ,and subsequent to this a more marked improvement in the headache became apparent. There was no recurrence of the speech difficulty and otherwise he soon felt as well as ever.

In the above case of A. R. the outstanding features in order were :- atonic condition of patient, unilatered headache of increasing intensity , aphasic attack, and finally complete recovery.

Dr. Clifford Allbutt has compared Migraine in the symptoms of its onset, outburst, and departure to Epilepsy and allied disorders, and points out that there is usually a regular cycle of events. In some cases , however, part of the cycle may be awanting ; thus we might have a hemicrania without the exhibition of accompanied symptoms ; or we might have the latter e.g. aphasia, haemiopia &c without the hemicrania. Such cases have been termed by Trousseau " Larval "types of Migraine. Allbutt points out that if we find Aphasia in a person apparently free from organic disease, and on enquiry get a neurotic family history, then it is possible that the Aphasia is actually the evidence of such a " Larval " migraine and in that case would be transitory in character. Dr. Clifford Allbutt refers in his paper to a case where he had reason to suspect such an occurrence, and the favourable prognosis which he gave was fully justified by subsequent events.

The actual pathology of Migraine is not yet quite clear, although the condition is believed to depend on some intracranical change. For the present , therefore, it is convenient to class the Transitory Aphasia of Migraine, like those of Epilepsy, under the Section comprising cases of " Functional Origin ."

TRANSITORY APHASIA IN CASES OF TOXÆMIA.

An Aphasia of a transitory character is an occasional concomitant of certain forms of poisoning e.g. snake-bite, plumbism, the Uraemia of Bright's Disease, and the Acetonaemia of Diabetes. So important indeed are the two latter that Wyllie would urge an examination of the urine for both sugar and albumen in all cases where there is a history of temporary attacks of aphasia.

In Diabetes a transitory attack of aphasia sometimes manifests itself at an early stage of the trouble while the patient is yet going about and able to follow his occupation. More commonly it manifests itself towards the end. Of the latter incident I can distinctly remember two instances in cases under my care - the one when Resident in the wards of the " Western Infirmary " and the other while assistant to Dr. Fraser of Carlton. Both patients referred to were men between 20 and 30 years of age and able to go about till within a few days of death. Then preliminary symptoms such as pains in the loins and in the calves of the legs, diminished secretion of urine " air hunger " &c. heralded the graver condition of diabetic coma. It was in the period subsequent to the appearance of these preliminary symptoms, and previous to the onset of actual coma (when of course speech is non est) that the aphasia of which I speak was manifested in my two cases. The patients could recognise the friends summoned to see them and understand all that was said but appeared themselves powerless to articulate a syllable in

reply. Acetone was present in the urine of both cases.

In the transitory aphasia which is known to occur after certain forms of snake - bite, Dr. William Ogle has put forward the theory that the poison produces spasm of the middle cerebral arteries. It is possible that a similar selective influence might be exerted by the poisons engendered in the sysyem in cases of Bright's Disease and Diabetes Mellitus, and so be productive of a transitory aphasia such has been described in those two conditions.

ON TRANSITORY APHASIA IN TYPHOID FEVER.

The occurence of an aphasic condition as a passing incident in the course of one of the continued or specific fevers has been noted by many writers on the subject ;and it seems to be generally agreed that of all the fevers, Typhoid is the one in which the complication is most likely to be met with. I do not know that any statistics have been made to indicate the frequency of the occurrence ;personally I have only noted one case of the kind in about 300 cases of Typhoid seen in private practice. The loss of speech in Typhoid or other of the continued fevers may be either of a functional , or of an organic origin. In the former case it is in all probability due to exhaustion of the higher nerve centres : in cases of organic origin the aphasia may follow embolism or thrombosis of the cerebral arteries

supplying Broca's area. Clarus seems to have made a special study of the subject so far as Typhoid is concerned , and his deductions as expressed by Wyllie are so much in accord with the history of my own case, that I give them here by way of introduction. " It,"(the aphasia)"appears usually " in the later period of a severe case, after other severe " nervous symptoms. The functional form is not accompanied " by hemiplegia . It occurs more frequently in children than " in adults , and seems commoner in boys than in girls. It " usually passes off in a few days. Sometimes it is prolonged " for weeks during the patient's convalescence from the fever : " but in such cases it is always ultimately recovered from. " When death occurs, nothing material is found in the Brain " to account for the Aphasia "

CASE of H.R. aet 5 years (Transitory Aphasia during Typhoid fever.

This boy came under my care on 28th. Sept. 1899 and in a few days presented undoubted symptoms of Typhoid Fever. The patient was not over-robust to begin with, and the attack soon began to tell severely on his system generally and on his nervous system in particular. The Aphasia to which I wish to draw attention made its appearance about the 12th. or 13th. day of the fever and continued present for the long period of 33 days. During all this time (with one exception to be referred to later)the boy did not utter a single articulative sound , and yet in the latter portion of this interval his temperature was absolutely normal and convalescence steadily going on.

At the end of the 33 days his recovery was only partial and for a further fortnight his vocabularly remained limited to the very simplest words. Progressive improvement however was slowly maintained andultimately he could speak as well as any boy of his age.

I have referred incidentally to one exceptional utterance which broke the otherwise silent period of 33 days. He was lying in bed examining in an apathetic kind of way a new toy which his mother had given him. His little sister began to make herself very agreeable , but whether or not he thought she had ulterior designs on his toy, it was soon apparent that he preferred her room to her company ; and when she ventured to become still more inquisitive about his new possession he suddenly came out with the expression " Be off " in such a way as to prove thoroughly effective . This expression , then, of a distinctly emotional nature, was the only articulative utterance for a period of close of 5 weeks. I made many attempts during this time to get him to speak. I tried bribing him with current coin of the realm and in every other way I could think of , but all to no purpose. He could put out his tongue when asked: and look at any person or article mentioned ;he could therefore understand what we said to him ;but he could neither say anything himself nor repeat it after us. On account of the loss of speech his mother felt it her duty to sit with him night and day for he could not draw attention to his ordinary wants except by signs which she intuitively understood. His parents were most anxious about his condition ,

and readily agreed to take note for me of any signs of return -ing language. His speech made its first reappearance on the morning of 12th.Novr. On awakening he said " Mamie " in his usual tone but at the sound of his own voice " he looked round frightened-like and quite flushed up ". Later he said " Ta " on receiving his breakfast of bread & milk ;and in the course of the day several other simple words were made use of . During the first week of recovery the Vocabulary did not exceed about 20 of the very simplest words ;but in the following week progress became more marked and he could soon speak as well as any other lad of his own age.

One other point in this case I wish to note, and that was the coincident loss of expression present along with the loss of the faculty of speech. Throughout the whole 4½ weeks of his aphasic comdition the boy was never noted to smile nor shed a tear. An occasional grunt he gave when displeased, and once or twice I detected a corrugation of the eyebrows equivalent to a frown . As a rule , however , he had an apathetic, expressionless, look about him, even when playing with his toys. On Tuesday 13th.November. the day after the first reappearance of speech,his mother reported that he shed a few tears, the first natural cry for weeks. His other various powers of expression,smiling , laughing , &c.returned at a later stage.

These observations seemed interesting inasmuch as the expressions can be best studied in a child of this age who has not yet learned to mask his feelings, and because the

relations between the evolution of speech , gesture , and expression have been shown to be intimate by many writers. In my patient the whole nervous system seemed to have become somewhat exhausted during the Typhoid attack ;and while the lower nerve centres certainly did suffer,they were not in abeyance to anything like the same extent as the higher centres for speech and expression . These last, being of later evolution and more delicate texture naturally suffered most of all.

In connection with the aphasia of the specific fevers, it may be noted that another form of speech disturbance , not strictly of an Aphasic nature, is sometimes met with in Typhoid Fever. This affection is described under the name of " DYSARTHRIA ". It consists of a slurring or of a staccato utterance which is generally regarded as due to a purely motor disturbance of speech. The " Slurring " form is recognised to be of grave import :but the staccato or stammering type is usually recovered from .

Case of A. C. aet 30 years.(Transitory Dysarthria In Typhoid Fever.)

Mr. A. C. was under my care in the autumn of 1899 suffering from,a somewhat severe attack of Typhoid Fever. Towards the height of the fever, headache was much complained of,and there was marked delirium at night. The temperature was frequently as high as 105 . 5°. During the second week a distinct difficulty of speech made its appearahce,the words be-

-ing pronounced in a slow staccato form of utterance with a distinct interval between each . Sometimes when I asked him a question he would stare at me for about a minute without being able to utter a syllable in reply : finally the words would be discharged as if each came out of a pop-gun. The case was not one of pure stammering ; there was no breaking up of individualswords and no sliding of the sibilants. The affection remained present about a week and towards the latter part of this period quickly disappeared. Speech was normal at the close of the fever.

Themabove two cases illustrate different transitory types of speech affection met with in Typhoid Fever. In regard to the occurrence of an Aphasia without Hemiplegia in the specific fevers, a good prognosis can generally be given ; but it ought to be remembered that in some few cases a thrombosis has apparently set in at a later stage and been the cause of a more permanent condition.

(over)

ON THE TRANSITORY SPEECH AFFECTIONS OF HYSTERIA.

A consideration of the various forms of Transitory Aphasia would be incomplete were reference, however brief, not made to the affections of speech met with in Hysteria. These affections are usually of a passing nature although exceptional cases of aphonia and mutism have been recorded in which the conditions lasted for years. In hysterical Aphonia, as the name indicates, the patient loses his or her voice but not whispered speech ;whereas in Mutism both vocal and whispered forms of speech are absolutely lost for the time being. The latter form (Hysterical Mutism) presents many characters identical with aphasia, inasmuch as the patient can understand spoken language perfectly & can write down an answer in reply, but is unable to articulate a single word of spoken speech. Indeed the affection has been located by so distinguished a neurologist as M.Charcot among the " Functional Motor Aphasiae ";but although this opinion is shared by many observers, there are others (notably Wyllie and Bastian in this country) who only accept it with very considerable limitations. Into these views however I do not purpose to enter here. It would take me beyond the scope of this Thesis. I prefer to give a few examples of the class under consideration ;and in passing , to point out how closely these speech affections of hysteria simulate aphasic types resulting from actual organic disease.

Case of Miss B. (Convulsive Hysteria with loss of speech during the"fit") .

Miss B. aet 24 years has been for some time under our care suffering from painful dysmenorrhoea. She has occasional hysterical convulsions of a minor type and the occurrence of these is usually coincident with the period of her monthly illness. The " fit " lasts from 5 to 10 minutes. During this time she is to all appearances unconscious and makes no response nor indication of any kind to signify that she is aware of being spoken to. For the time being she is quite bereft of speech. Yet she herself has often assured us afterwards that during these seizures she knows perfectly well who is speaking to her and understands all that is said, but at the same time feels perfectly helpless to articulate a reply of any kind whatever. She at once recovers her speech when the " fit " passes off.

2 Case of Miss H. (Convulsive Hysteria with loss of speech persisting after recovery of consciousness.)

Miss H. aet 22 years, also suffers from Dysmenorrhoea and Ovaritis, and has occasional hysterical seizures of a very violent type. She cannot of course speak during the convulsive attack, but excepting on the following occasion, has always recovered her speech immediately on the return of consciousness. On this occasion (15th.Feby. 1900) she was recovering from her menstrual period which had at the onset been even more painful than usual, and the atate of nervous tension was no doubt still further heightened by the excitement of her approaching marriage. About 6 P.M.

on the above date , she complained of faintness,and after some premonitory twitching about the angles of the mouth, went off into a violent convulsive seizure. The " fit " lasted about 15 minutes and she then recovered sufficiently to sit up. Contrary to her usual experience , the speech on this occasion did not come back with returning consciousness, and this so alarmed her parents that they sent for me. She remained absolutely mute for over an hour although from her manner and gestures it was plain she understood all that was said. She couldalso write, although somewhat unsteadily , when tested on a slate. She did indeed make attempts to speak but beyond a grunting sound,and the vowel sound " ah " which was uttered once or twice after considerable effort, no sound was produced , and certainly nothing in the way of either whispered or vocal speech.
In rather over an hour's time speech returned quite suddenly. She complained of a numbness affecting her whole body, but this feeling likewise passed away in a further few minutes time!.

3 Case of Mrs. E. (Non-Convulsive Hysteria :Condition of Aphonia alternating with periods of Mutism. Paresis of Arm and Leg & exaggerated Reflexes)

Mrs.E.(aet 36 years) married 6 years ago but has had no family. She attributes her hysterical attacks to a period of domestic trouble 2 years ago when her husband left her. Shortly after that event she first suffered from an attack

of aphonia similar to the present one. She also suffers from occasional ovaritis. Menstruation is regular but scanty. At no time has she had anything approaching a"fit";in other words the hysteria has always been of the non-convulsive type.

On 22nd.Jany.1899.she suddenly lost her voice without preceding catarrh or other throat ailment ;but remained able to speak in a horse whisper. Coincident with this aphonia she developed a paresis of the left arm and leg. I saw her on the 12th.Feruary.and found the conditions described. The grasp in the left hand was very weak,and the power in left lower extremity was lost to such an extent that it was impossible for her to walk without some very material support. The left Knee-jerk was exaggerated and ankle-clonus readily obtained. The Phalangeal flexors of left foot were in a state of tonic contraction causing the toes to arch downwards towards the sole. She complained of numbness in the left upper and lower extremities. There was no wasting of the paralysed limbs. Physical examination of the Chest revealed nothing abnormal ;in the abdomen great tenderness was found to exist over both ovarian regions but especially the left.

Laryngoscopic Examination on this and subsequent occasions shewed that there was distinct paresis of the adductor muscles and that the vocal cords could not approximate each other in phonation. From the whole history of the case as well as from the symptoms present it was clear that Mrs. M.suffered from hysterical paralysis.

The aphonia remained present more or less for a period of six weeks. On one occasion the aphonia was replaced by a condition of absolute mutism when both whispered & vocal speech were lost. She remained throughout able to express herself in writing if desired to, do so.
The paralysis ultimately disappeared as suddenly as it came. I need not detail the treatment.

The above 3 cases are I think, fairly illustrative of some of the Transitory Speech Defects met with in Hysteria. In case 1. (Miss B.) the speechlessness was only present during the actual convulsive seizure. In case 2.(Miss H.) the loss of speech was present not only during the convulsive seizure but also for some time after ; the inhibition or disability of speech being as it were carried over into the period of succeeding consciousness. Such a condition might, I think, be aptly compared with an aphasia following certain cases of epileptic convulsion. In case 3. (Mrs.E.), we have an illustration of aphonia and mutism as met with in hysteria of the non-convulsive type. In hysterical mutism the patient cannot utter a syllable of either whispered or vocal speech, but can express herself in writing; the condition exactly resembles that type of Motor Aphasia in which while spoken speech is lost , written speech is retained (Aphemia); Such a condition is brought about by actual organic disease.

Hysterical speech defects are usually of a passing or

transitory nature ;but some caseshave been known to persist for years. It is impossible therefore in a prognosis to give any approximate date of recovery ;and even after the patient is apparently well a recurrence may take place at the most unexpected times.

This concludes my section on Transitory Motor Aphasiae of Functional Origin.

The cases which I have referred to in the first part of my paper have been grouped under the heading " FUNCTIONAL APHASIAE " Such Cases are without [illegible] more frequently of a transitory nature than are [illegible] actual organic lesion such as [illegible] Nevertheless many cases of the [illegible] under favourable circumstances, tend to recovery [illegible] we not have a transitory [illegible] of organic origin [illegible] similar cause.

SECTION 2.

ON TRANSITOR MOTOR APHASIAE of ORGANIC ORIGIN

(with illustrative cases)

The factors which determine recovery in [illegible] numerous [illegible] extent of lesion. In dealing with lesions in [illegible] with other parts of the cerebral cortex, it has to be remembered that we have to do with a grey mantle [illegible] tissue of delicate structure and supplied by [illegible] having little, if any, anastomotic connections. If [illegible] of these " end arteries "as they have been termed [illegible] ed, permanent damage of tissue will certainly result unless removal of the obstruction is quickly effected. The [illegible] of recovery would naturally be greater where [illegible] plastic and where the lesion involves [illegible] arteries supplying only a limited area [illegible] observations bear this out. Thus [illegible] aphasia [illegible] simply loss of speech [illegible]

The cases which I have referred to in the first part of my paper have been grouped under the heading " FUNCTIONAL APHASIAE " Such Cases are without doubt much more frequently of a transitory nature than are those depending on an actual organic lesion such as cerebral haemorrhage or embolism. Nevertheless many cases of the latter class, do, under favourable circumstances, tend to recovery, and thus we may have a Transitory Aphasia of organic origin just as we may have a transitory monoplegia or hemiplegia due to a similar cause.

The factors which determine recovery in these cases are numerous, including age of patient, nature, situation, and extent of lesion. In dealing with lesions in Broca's area, as with other parts of the cerebral cortex, it has to be remembered that we have to do with a grey mantle of nervous tissue of delicate sructure and supplied by Bloodvessels having little, if any, anastomotic connections. If, then, one of these " end arteries ";as they have been termed, gets blocked, permanent damage of tissue will certainly result unless removal of the obstruction is quickly effected. The chances of recovery would naturally be greater where the vessels are still elastic and where the lesion involves one of the smaller arterioles supplying only a limited area; and clinical observations bear this out. Thus we find that cases of Motor Aphasia involving simple loss of speech and accompanied by Agraphia or other form of paralysis, not uncommonly end in recovery:

whereas

whereas in cases of Aphasia presenting a combination of all those symptons the chances of recovery are more remote. The term " Aphemia " is generally applied to cases of motor aphasia in which there is loss of speech but in which the patient still retains the power of expressing himself in writing. This is a distinctly recognised but rare form of affection, and only a moment's reflection is necessary to remind one that the lesion in such a case must be extremely limited. The fact that in these cases a fatal termination is most uncommon except as the result of some inter-current affection, explains the scanty records we possess of the post mortem conditions found in Aphemia ; but it is generally believed that the lesion is a very limited one affecting either a portion of Broca's cortical area, or the subcortical fibres leading downwards from that centre. Such a conclusion is also supported by the clinical facts of recorded cases taken in conjunction with the light thrown on them by experimental physiologists. I will give several illustrations of this class of affection, one of them occurring in my own practice, and it will be seen from these how often this ailment is of a temporary or transitory character.

Case of Dr. Samuel Johnson:

The case of Dr. Samuel Johnson, as reported in his life by Boswell, may be taken as a classical instance of the affection under consideration. The attack of Aphemia from which Dr. Johnson suffered, occurred on June 17th. 1783, in his 74th. year. The loss of speech lasted over a period of twenty hours but

but during this interval his mental faculties were unimpaired, and he was also able to express himself quite well in writing as shewn by the letters which he wrote on the same day. He himself describes his attack in a letter to Mrs. Thrale 2 days later :-

" On Monday, the 16th. I sat for my picture, and walked a
" considerable way with littleinconvenience. In the afternoon
" & evening I felt myself light and easy, and began to plan
" schemes of life. Thus I went to bed, and in a short time
" waked and sat up, as has been long my custom, when I felt
" a confusion and indistinctness in my head, which lasted
" I suppose about half a minute. I was alarmed and prayed God
" that, however he might afflict my body, he would spare my
" understanding. This prayer, that I might try the integrity
" of my faculties, I made in Latin verse. The lines were not
" very good, but I knew them not to be very good; I made them
" easily and concluded myself to be unimpaired in my faculties
" Soon after I perceived that I had suffered a paralytic
" stroke, and that my speech was taken from me. I had no pain
" and so little dejection in this dreadful state, that I won-
" dered at my own apathy, and considered that perhaps death
" itself, when it should come, would excite less horror than
" seems now to attend it. In order to rouse the vocal organs,
" I took two drams. Wine has been celebrated for the pro-
" duction of eloquence . I put myself into violent motion,
" and I think repeated it ;but all was vain. I then went to
" bed, and strange as it may seem, I think slept.
" When I saw light, it was time to contrive what I should do

" Though God stopped my speech he left my hand.....My
" first note was necessarily to my servant, who came in talk-
"ing, and could not immediately comprehend why he should
" read what I put into his hands.

" I then wrote a card to Mr. Allen that I might have a
" discreet friend at hand, to act as occasion should require.
" In penning this note I had some difficulty: my hand, I knew
" not how or why, made wrong letters. I then wrote to Dr.
" Taylor to come to me, and bring Dr.Heberden..... I
" have so far recovered my vocal powers, as to repeat the
" Lords prayer with no imperfect articulation.

The loss of speech was of short duration for on June 25th.
" he writes " Before night (on the 17th.) I began to speak
" with some freedom, which has been increasing ever since,
" so that now I have very little impediment in my utterance"

These extracts from Dr.Johnsons letters illustrate a typical attack of Aphemia. It is, I think, somewhat notable that we should find in lay literature so lucid an account of an affection which, at the time of writing , had not even been assigned its place in medical nomenclature, and which even at the present day is only recognised as a comparatively rare occurrence. Truly Boswell did well to preserve permanently these accounts;even from a medical point of view one cannot deny him.homage as the"Prince of Biographers"

Case of Mrs. M. (Personal Observation)

The following case of Aphemia I think worthy of record for several reasons :-

(1) It is a typical illustration of the form of aphasia

under consideration, viz :- loss of motor articulate speech while the power of written speech is retained.

(2) It was transitory in character.

(3) It occurred during the latter months of pregnancy in a woman of apparently sound physical constitution.

(4) Restoration of speech was gradual and I had the opportunity of taking some notes on the return of the various articulate sounds, interesting I hope from a philological point of view, as well as possibly helpful in the education of other cases. I must therefore claim a little indulgence if it has been necessary to give the case in some detail. The history of the case which I saw for the first time in September 1896 with my then principal, Dr.Longwill, of Rutherglen, was only obtained in disconnected form owing to the patients inability to speak ; but I have endeavoured to give it as far as possible in narrative form as less likely to prove wearisome to the reader.

At the time of the attack Mrs.M. was seven months pregnant. During the most of the time she had been carrying the child, she had suffered more or less from the usual sickness & vomiting, but apart from this had made no special complaint. Her family history was good, and excepting the attack to be described, she had herself always enjoyed fair health.

On the evening of 16th.September 1896, Mrs. M. retired to bed feeling as well as usual. She rose next morning between 7 and 8 A. M. and went about her household duties; having kindled a fire, she was about to put the kettle on, when she suddenly noticed her little boy half awake and rolling right over

almost on the edge of the bed. She became terrified lest he should fall over and attempted to shout out to him to take care, but was astonished to find" the words would'nt come " and that she had absolutely lost her speech. Her husband awoke at this time and found her unable to speak - only " grunting " and making signs. He was somewhat alarmed and advised her to go back to bed and rest a little. She did so, and in the course of a few minutes her speech began to slowly to return; by the time she got breakfast (9 am.) she could speak again as well as ever. This first attack of speechlessness was therefore of very short duration.

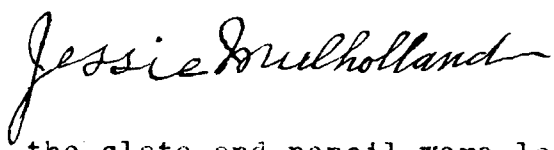
After breakfast she called next door to tell her neighbour Mrs. P. about the peculiar loss of speech from which she had suffered that morning. Mrs. P. says that at first she spoke as well as ever to her, but had not proceeded far when the speech got " thick " and indistinct; in a further few minutes Mrs. M. had completely lost her speech for the second time, and could only make herself understood by signs. This attack however, was likewise of short duration, for she was seen both by Dr. Longwill & myself at the Surgery at 11 a.m. and she then spoke perfectly. On her way home from the Surgery she felt some momentary twiching of the right angle of mouth, but continued to speak quite well till late in the afternoon. About 3 or 4 p.m. a " thickness " of speech again manifested itself and her neighbour Mrs. P. who seems a particularly observant old lady, states that she also spoke more slowly than usual. Gradually the speech became more difficult and indistinct until about 9 p.m. when she was

unable to articulate a single syllable. She retired to bed hoping the condition would again pass off, but as on awaking next morning she was still speechless, we were summoned to see her. This third attack I will now describe in more detail, as it lasted for a somewhat considerable period and I had the opportunity of keeping the case under observation for some months.

On the 18th. September when I saw her at her own home, Mrs. M. exhibited all the symptons of Aphemia. Loss of speech was absolute. She could not even say simple words of one syllable. When answering a question requiring an obvious negative she would attempt to say "no" and finding this impossible would finally shake her head. Sometimes after failure to speak she would make a gesture of annoyance, and then perhaps an amused smile as much as to say " well this is strange" —"I cant understand it ! When she required anything she would ask for it in pantomine'. Her mental faculties were as clear as ever ; she noted any conversation going on in thee room and would correct (again by pantomine) any obvious mistakes. If anything ridiculous were mentioned, her face at once locked amused ; her other expressions were also noted to be natural. I ought to say that on one or two occasions the words " no " and " yes " came out as it were automatically in answer to questions, for if asked to repeat these two same words after me she found it impossible to do so.

We learned from her signs and by putting questions to her requiring a nod or shake of the head for answer, that she had some frontal headache and some tingling in the right forearm,

There was however,no obvious paralysis of the mouth or face ; She was thoroughly tested to ascertain these points. The senses of smell and taste were normal. Knowing the close connection between right-sided paralysis and aphasia the condition of the right arm and hand was also carefully noted, but at this time there was no appreciable loss of power in this extremity. I accordingly tested her power of writing - it was as good as ever. She put down her name,copied a letter and wrote to dictation in as good a hand as ever. I give here a fascimile of her name as she wrote it onthis date :-



At my suggestion, the slate and pencil were left constantly beside her : she continued to ask for things in pantomine as being the most rapid method, but in the event of not being understood ,at one wrote down any conversational blanks on her slate.

Physical examination of the heart , lungs, and excretions on this date revealed nothing abnormal. There was no specific indication for treatment beyond gastic disturbance attributed to pregnancy. For this last we prescribed Cerium Oxalate combined with Bismuth and Antipyrin. She was also ordered complete rest in bed.

On the morning of the following day,19th.September. I found the aphemic condition much the same. Distinct paresis had, however,made its appearance in the right hand, the grasp

of which was distinctly less than that of the left, and there was also marked anaesthesia of the right wrist and hand. The patient had consequent difficulty in performing finer movements such as knitting, sewing or writing ;nevertheless she could still write quite legibly though slowly on the slate whenever she found it necessary to do so. The paresis was most marked in the muscles of thumb and forefinger.

The cutaneous sensibility was tested by Weber's method -i.e.by noting the shortest distance at which two points of the compasses could be recognised : and I found that although more or less anaesthesia was present over the wrist, palm, and fingers,yet it was most marked over the rad--ial aspect, notably the thumb, forefinger , and ball of thumb The distal phalanges suffered more than the proximal ones. The details of the cutaneous sensibility tests made on this and subsequent occasions, are given later in the tabulated form. A further incident, the importance of which I will point out later, falls to be recorded on this date (19th. September) Mrs. M. complained of pain in the lower part of right leg,and on examination I found very distinct tenderness from apparent phlebitis and thrombosis in the venous radicals over the lower part if right tibia. Fomentations gave relief but it was fully a week before the induration and tenderness had disappeared.

I also tested the vision at this time. There was no refractive error, the visual fields were normal & colour sense intact. On.O. E. the fundus presented nothing unusual. The hearing was perfect.

There was numbness and slight anaesthesia over the right cheek in proximity to the angle of mouth but this was not accompanied by any obvious paresis of the muscles.

Generally speaking, the examination confirmed the association of aphasia with Right Sided Paralysis, and indicated that in cases of very limited lesion with aphasia the parts most affected are - Right angle of mouth, muscles of right thumb and forefinger, and to a lesser extent the muscles of other fingers and forearm. The association of these areas as observed clinically is interesting in view of the similar cortical grouping already demonstrated by experimental physiologists.

On the evening of the above date (19th. September) I again visited the patient and was pleased to find a slight but notable return of speech. Mrs. M. could say the words " No " and " Yes " more frequently, although as a rule she simply answered by nodding or shaking the head. When I asked her if she felt better, she immediately answered " Better " speaking the word somewhat disconnectly. Again, in answer to another question she got the length of saying " I had ---- " suddenly stopping short and giving her head a decided shake, clearly expressing annoyance at her inability to complete the sentence.

On Sunday, 20th. September, I asked her to count 1 ,2, 3, 4, holding up the fingers of my hand. She could not do so but was able with considerable effort to repeat the numbers after me, thus :- " w.....ūn; " " t...oo..oo "; " th - ee ":, " f-f...four "

This simple exercise of language was to her most difficult and the effort unfortunately set up a severe headache.

The gradual improvement in speech continued and two days later she could voluntarily count up to twelve although with several mistakes in the consonant sounds to be summarised later. These indications of returning speech were most encouraging and enabled me even at this early stage to feel quite justified in giving a favourable prognosis. As a matter of fact, speech perceptibly improved every succeeding day - at first slowly - and in the course of a few weeks, very rapidly. I took careful notes of the words as they returned so that I might make make an analysis of these which might prove interesting from a philological point of view.

In testing the patient's powers of articulation I made use of several methods - such as pointing out available objects of furniture &c; also noting any deficiencies in her own voluntary speech. What I found most valuable and complete test, however, was Sir Isaac Pitman's phonographic alphabet which contains of course all the phonetic sounds in the English language. I was not at this time aware of Professor Wyllie's Physiological Alphabet; but Pitman's was equally good and had the additional advantage of giving numerous examples which proved of great value in testing the patient.

The English Language, as pointed out by Pitman, contains but 23 useful letters (rejecting **c**, **q**, and **x** as equal to s, k, and **ks**) to represent the 41 distinct sounds of the language.

PITMAN'S ALPHABET.

Consonants.

Explodents.

P	= pee	as in	rope
B	bee	"	robe
T	tee	"	fate
D	dee	"	fade
CH	chay	"	etch
J	jay	"	edge
K	kay	"	leek
G	gay	"	league

Continuants.

F	= ef	as in	safe
V	vee	"	save
TH	ith	"	wreath
TH	thee	"	wreathe
S	es	"	hiss
Z	zee	"	his
SH	ish	"	vicious
ZH	zhee	"	vision

Nasals

M	= em	"	seem
N	en	"	seen
NG	ing	"	sing

Liquids

L	= el	"	pall
R	ar	"	air
R	ray	"	raise

Coalescents.

W	= way	"	way
Y	yay	"	yea

Aspirate

H	= aitch	"	hay
H	"	"	

Double Consonants

WH	= whay	"	where
KW	kway	"	quick
GW	gway	"	anguish
MP	emp	"	hemp
MB	emp	"	embalm
LR	ler	"	feeler
WL	wel	"	wail
WHL	whel	"	whale

LONG VOWELS.

AH	"	Pa	
EH	"	may	
EE	"	be	
AW	"	thought	
OH	"	so	
OO	"	poor	

SHORT VOWELS

a	"	that	
e	"	pen	
i	"	is	
o	"	not	
u	"	much	
oo	"	good	

DIPTHONGS.

I as in My.OW as in Now. OI as in Oil,U as in New.Wi asin Wide.

I have made use of the above alphabet in compiling the following summary of my patients recovery:—

The long vowels and dipthongs, with one exception, were recovered very early in the first week, and " Eh " (as in may) first of all. The exception referred to was " U " (as in new) which the patient continued to pronounce "oo " (as in wool) until about the third week of recovery. The Triphthong " Wi " (as in wide) was also recovered among the earliest sounds but for some days she had a tendency to place too much stress on the initial " W "

The Consonants gave by far the most trouble. Some of these were,it it is true, recovered almost as soon as the vowel sounds, but many others were not mastered until after the lapse of several weeks. Of the consonants,the nasals " M " and " N " caused the least difficulty of all and were the first in the process of restoration. This is not to be wondered at since " m " is a very simple sound and easily acquired. Thus if a person can say the long vowel " ah " he has only to commence the sound while the mouth is being opened to produce the result " ma " -- a combination of nasal and vowel which is as a matter of fact one of the earliest words in baby speech.

The Explosives " B " and " P " were also among the first to be recovered, but even after their restoration, the

patient had for some days the habit of pronouncing them with much more " explosive " force than natural to educated speech. This was doubtless due to the great effort entailed in her endeavour to pronounce even simple sounds.

" T " and " D " were likewise soon acquired, but for some days there was a tendency to prolong the sounds too much especially in " D " terminal e.g. in fade .

" Ch " and " J " could also be pronounced towards the end.of the first week.

" K " as in " leek " could be pronounced about the 6th. or 7th. day. A combination of " K " with another consonant e.g." Kl " (as in "clock") caused more difficulty than a single "K" in direct combination with a vowel sound. " G " as in league was only managed with difficulty in the course of the second week of recovery. At first the patient invariably pronounced it " K " e.g." leak " instead of " league " , "Gl" was more difficult of acquirement than "Kl" thus the patient could say " Kl " as in "Clock" towards the end of the first week,but could not say "gl" as in "gloves" for many days later - not even after it had been repeatedly pronounced by myself in her hearing " Gloves " she usually called " Kloves " ;indeed the combination " gl " was not acquired properly until well on in the second week. As a matter of fact this combination "Gl" was more difficult to recover in the process of restoration of speech than any other in the alphabet excepting perhaps the dipthong "U"(as in new) .

The Coalescents " W " (as in way) and " Y " as in " Yell

were learned in the first week of recovery.

" Yes " was one of the first words to return but this was probably owing to its being a word of common occurrence. Thus while she could say " yes " she could not say"year" . The numeral, (" wun ")could after some endeavour be pronounced after me on the 20th. September.and voluntarily a couple of days later ;but there was during the first week too much stress laid on the initial " w "

The Continuants." f " and " v " were often misplaced the one for the other e. g. " save " pronounced " safe "

If anything the " v " caused more difficulty than the " f " These consonants could also be pronounced in the first week of recovery.

The Sibilant." s " was easily reacquired. Like " f " and "v" the consonants " S " and " z " were often transposed the one for the other. " SH "(as in vicious)caused little difficulty but in cetain combinations mistakes were apt to occur . Thus " vicious " was during the first two weeks invariably pronounced " Vishoush "

The Liquid Consonant " R " could be easily pronounced in certain words, and only with difficulty in others, depending apparently on its position as well as on its combination with different consonants. Thus initial " r " as in " Ray " was easily articulated when " r " terminal was pronounced with a burr owing to the patients inability to bring the word to an end - e.g. " four " pronounced " four. r .r.r. " the " r " was also pronounced"l" on many occasions during the first two weeks e.g. " lug " for " rug ":lubber for " rubber &c.I also noticed that more particularly with " r "

than with any other consonant, the acquirement of its correct pronunciation depended greatly on the combination of letters forming the word. This was markedly illustrated by asking her to pronounce the numerals " three " ," Thirteen " and " thirty ". " Three " during the first week was invariably pronounced " Th-ee", yet she could say " thirteen " and " Thirty " quite well. The apparent explanation seems to be that the intervening vowel " i " in the latter cases acted as a kind of articulative crutch. As late as as 28th. September she could not read words like "strong", "shrill", and only pronounced them with great difficulty after I had repeated them in her hearing several times. Sometimes, in large words, " r " was missed even when only in combination with one other consonant eg. " penet-ate " for penetrate, " f- equency" for frequency .

The Liquid Consonant " l " gave little trouble.

The Double Consonants were later of recovery than the simple ones:, and were at first given with too much explosive force as if only produced by much effort ; this was especially noticeable in the case of "gw" (as in anguish) and "mb " (as in embalm)

As far as my observations in this patient went, the latest and therefore most difficult to be acquired were the following combinations :- the gutteral " g " with an " l " as in " Gloves "; treble consonants occurring together without an intervening vowel especially with a component " r " e.g. "shr ", "str ", "thr" as in "shrill" " strong " "Three " &c. and the latest of all was the dipthong " U "

(the ordinary " u " of the English Alphabet) This last for ~~last~~ for several weeks was invariably pronounced "oo".

Having pointed out in the above summary the mode of recovery so far as the component vowels and consonants were concerned, I will now briefly indicate the manner of recovery for words, groups of words , and sentences. During the earlier part of the first week the vocabulary remained strictly limited to a few simple words of common usuage and mostly of one syllable. Even these were only articulated with much effort and the patient often preferred to make use of pantomine ' e.g. Shaking or nodding the head for " no " and " yes " respectively. At this time as well as later she could in many cases repeat a word after hearing it pronounced although she could not articulate it spontaneously. During the second week, larger words of two and more syllables were attempted, but at first these could only be pronounced by breaking them up into their component sounds. She also began to say small groups of words , but there was for some weeks a very distinct interval of time between each word as if each required a separate effort. Even as late as the third week I have seen beads of perspiration standing out on the patient's forehead from the intense effort required to read a few lines.

About 4th. October (17 days from date of attack) the patient began to string together short sentences, and from this time recovery was more rapid. Yet although she now found herself able to articulate any word or sound in the English language, it was several months before her speech was

at all free and unconstrained. I may therefore say that in this case of aphemia the order or recovery was as follows :-

(1) Simple words of common usage e.g. No, Yes, the ordinary numerals &c.

(2) Simple words of less common usage ; words of two and more syllables.

(3) Disjointed groups of words.

(4) Small sentences.

(5) Larger words & complete sentences without the necessity of any accompanying pantomine'

In short the patient regained her language in the same order as a child learns its Baby - Speech. If we can imagine a child's initial speech training of several years compressed into the short period of as many weeks, we should have an exact picture of Mrs.M 's. recovery.

I shall afterwards point out that in the recovery of words in the condition known as " Amnesia Verbalis ;"
(the " Aphasia of Recollection " of Kussmaul) there is a recognised order of return for the different parts of speech viz :- Verbs, prepositions , & pronouns first, nouns later, and proper names last of all. This order however does not seem to hold good in cases of Aphemia such as the above. Recovery of words in the latter cases of motor aphasia seems to depend rather on the words themselves - in other words on the complexity of their sound units. The reason for the distintion is at one apparent; in amnesia the recall of words to consciousness is interfered with; in motor aphasia

(aphemia) the words are actually present in consciousness but the difficulty is for the injured motor mechanism to translate them into articulate sound

Note on Paresis and Tactile Anaesthesia

Of Right upper extremity.

The paresis noted in the right hand and fingers in this case quickly disappeared for I found that on 7th.October (3 weeks from the onset of aphemia) the grasp in the right hand was as good as that of the left. The numb feeling in the vicinity of the right angle of mouth had also disappeared. The tactile anaesthesia in the right upper extremity remained deficient for many weeks and I had abundant opportunity to note its manner of improvement. The cutaneous sensibility was tested from time to time by Weber's method and I give some of the results on the accompanying table :-

TABLE OF TACTILE SENSIBILITY.

	Left Hand	Right Hand (Affected by Lesion)					
	Sep19	19	24th	30th	Oct7	Nov6	Jan4
Ball of Thumb (middle)	7mm	22mm	22mm	10mm	10mm	8mm	8mm
Thumb (Proximal Phalanx) middle	6mm	11mm	11mm	10mm	8mm	7mm	7mm
Thumb (Distal ") "	4mm	0	0	0	0	12mm	(9mm at tip) 4mm
Forefinger (Prox. ") "	6mm	11mm	11mm	10mm	10mm	9mm	9mm
Forefinger (Mid. ") "	5mm	0	0	11mm	10mm	8mm	6mm
Firefinger (Dist. ") "	4mm	0	0	0	0	12mm	10mm
Middle Finger(Dist. ") "	4mm	0	0	0	8mm	5mm	5mm

In taking the above tests I found the somewhat interesting fact that improvement took place in sections. Recovery of Tactile Sensation was

first most noticeable in the palm : next in the proximal, and finally in the distal plalanges. The Skin over the proximal part of any individual phalanx. It was therefore essential to take all my observations by placing the two points of the compasses transversely to the long axis of each finger.

From the grouping of the symptoms in this case, the causative lesion must, I believe , have been situated either in a portion of the third left frontal convolution, or in the immediately adjacent sobcortical fibres. The fact that cutaneous sensibility was distinctly affected by the lesion would go to support the belief of sensory as well as motor representation in the grey cerebral cortex ; in other words that the so-called " motor area " is actually " sensori motor " as held by Horsley and Hughlings - Jackson. The mode of recovery of the cutaneous sensibility in this case in segments would also appear to give clinical support to the view of Horsley that not only is the tactile sense represented in the above cortical area, but " that its representation,, is, like that of movement, segmental in character ."

The pathology of this case in its relations to the fact of the woman's pregnancy I shall discuss later in a subsection on " Puerperal Aphasia ".The after history will be referred to at the same stage.

(Wyllie)

Wyllie , Bastian , Trousseau , and other writers on Aphasia give examples of this class of affection. Wyllie gives a case ("Patrick Keaney") which in many of its features resemples that of Mrs. M. reported above. It was formerly believed that in cases of Aphasia the loss of speech was invariably accompanied by a coextensive loss of the faculty of writing ;such cases as the above appear antagonistic to such a theory. The point, however, to which I wish to draw attention here , is that the lesion in Aphemia must necessarily be a limited one, and that recovery, either partial or complete , is often the ultimate result. While therefore a prognosis in these cases should always be of a somewhat guarded nature, we are justified in regarding Aphemia as one of the most favourable types of Aphasia, and as by no means unlikely to prove transitory in nature. The cases which I have given by way of illustration happily ended in complete recovery.

(Motor Aphasia with Agraphia --over)

MOTOR APHASIA with AGRAPHIA.

In cases of loss of speech accompanied by agraphia or by more or less extensive paralysis usually of the hemiplegic type, the chances of the condition being of a transient nature are much more remote . Such cases as a rule have not a bright outlook, and recovery , when it does occur , is generally but partial and only effected after a long period of time. In some instances restoration of speech seems to be brought about by a clearance of the vascular channels and the gradual return of function to the damaged convolution in other instances it seems to be effected by means of the education of the corresponding convolution in the opposite hemisphere. As an example of the latter incident, a case reported by Br. Barlow is a somewhat notable one and quoted by several writers on Aphasia. Dr. Barlow's patient, a boy aged, 10 years was the subject of aortic regurgitation and had an attack of right hemiplegia with aphasia, due, as afterwards shewn , to embolism of the left Anterior Sylvian artery. The boy made a good recovery and in course of time regained his powers of speech. Four months later the Right Anterior Sylvian artery got likewise plugged by an embolus and aphasia resulted a second time :, no recovery ensued on this occasion. The conclusion arrived at in this case was that after destruction of Broca's convolution in the first instance , the corresponding convolution on the right side of the Brain acquired the lost function by a process of education; and speech was restored. The occurence of embolism of the right

Anterior Sylvian artery at this stage destroyed the new centre and so speech was irreparably lost. Death occurred later from the effects of the aortic lesion and post mortem the emboli referred to, were demonstrated. I mention this case as interesting because it shows how a Transitory Aphasia (as in the first attack here) may be due to the education of a new speech centre on the opposite side of the Brain. Such an education would be more likely to occur in a young subject like Dr. Barlow's patient than in a person more advanced in life. In youth not only are the bloodvessels more elastic and the demand for an increased vascular supply to a new centre likely to be promptly met, but the "receptivity " of the Brain cells to new impressions is a distinctly favourable element. This " receptivity " becomes less as a person advances in years, but exceptionally we find even old people in whom such an endowment is present to a much grester degree than in others of a like age. The presence of such " educability " would constitute a favourable element in the prognosis, and goes to explain how even in advanced life a new speech centre may be ultimately evolved in certain cases of Aphasia.

I have already incidentally mentioned that the SITUATION OF THE LESION may determine the production of a transitory Motor Aphasia. There is good reason to believe

that a quick recovery of speech in cases of extensive , cortical lesions involving Broca's area is sometimes due to the fact that only the margin of the lesion encroaches on the speech centre or its efferent fibres. In this way might be explained many cases of temporary loss of speech at the time of an apoplectie attack involving permanently an arm or a leg. I lately came across a good illustration of such a Transitory Aphasia in one of my parish patients :-

Case of Mrs. Masson (aet 75 years).

On the 23rd. February 1900 I was called to see this patient and got the following history, At 7.30 P.M. while sitting in her armchair, she felt a twitching of the right side of face and slight convulsive movements in the right arm and right leg. A few minutes later she lost her speech and was unable to say anything for fully half an hour. Nevertheless she remained quite conscious and nodded assent when her daughter proposed sending for the doctor. She was also able to make known her other wants by means of signs. I saw her an hour later. Speech had returned, but not freely, and the patient seemed also to have some difficulty in knowing what to say. The tongue was pro-truded towards the right side. The pupils were equal. There was distinct paresis of right arm and right leg. The arteries were distinctly atheromatous, and the arcus seniles well marked: Heart sounds normal.

On the following day (24th. February) Speech was as good as ever.

On 25th. Feby: a more severe attack supervened; the right arm

and right leg were completely paralysed and there was again absolute loss of speech. On this occasion the aphasia was likewise transitory in character for by the following day, 26th.Feby: the speech was almost perfect although the limbs remained powerless. On the evening of 27th February the patient had a third transitory attack of aphasia lasting about an hour. The writing could not be tested. This patient has since been under observation for several months. Speech has remained intact , but the limbs are still paralysed and rigidity has set in. There was therefore in this case three distinct attacks of Transitory Aphasia accompanying a hemiplegia which has remained permanent. I think there is good reason to believe that a haemorrhage involved premarily the leg and arm cortical areas and that the speech centre was only involved in the confines of the lesion;consequently with retraction and absorption of the clot the speech area was the first to be relieved from pressure and the aphasia passed off. This case seems to me a good illustration of how a " Transitory Aphasia " may be produced in this way.

The considerations which I have referred to in cases of embolism and haemorrhage ,apply in a general way also to cases of THROMBOSIS. If the thrombosis be limited and a collateral circulation established , or a removal of the thrombus effected in suffieient time, we might have an

Aphasia of Transitory character. Such a recovery would likewise be more probable in younger patients. The Thrombosis might also be related to some constitutional disturbance affecting the vessels locally. It might be due to syphilitic endarteritis , of to atheromatous degeneration ; or again it might be dependent on the condition of pregnancy . Such constitutional conditions would have to be taken into consideration in forming an opinion as to whether the Aphasia would probably end in recovery or otherwise. (See subsection on Puerperal Aphasia).

Besides cases of Transitory Aphasia, due to cerebral embolism, haemorrhage & Thrombosis, we may occasionally find the same condition produced by extraneous forms of pressure on Broca's area. Cases have been recorded where in fracture of the skull a spicule of bone has impinged on the speech area and produced Aphasia ; such an aphasia has passed off subsequent to a trepanning operation.

Cerebral Abscess or, tumour might conceivably set up a similar train of events. Cerebral Tumours often give rise to stmptoms of a temporary character according to the varying conditions of intracranial pressure ; in this way we might have Transitory attacks of Aphasia just as we find transient convulsive attacks affecting an arm or a leg. Where the growth is remediable by operation an entire disappearance of the attacks might be anticipatd . Similar pressure might be exercised by a syphilitic gumma. Indeed in all

cases and especially in Transitory types, particular enquiry should be made for any evidence of a syphilitic history. Attacks of temporary loss of speech are by no means uncommonly due to the presence of a syphilitic endarteritis . Remedies directed to the constitutional ailment will as a rule prove successful and the aphasic attacks disappear . A good example of this class of affection is recorded by Professor Mc.Call Anderson in his work on"Syphilitic Diseases of Nervous System ";this case is so apropos of my subject of " Transitory Aphasia " that I have taken the liberty of quoting it here :-

Case 69. " Attacks of Temporary Aphasia with Confusion of Mind "

" A gentleman ,aet 38, consulted me on the 31st. August. " 1886,Between the 3rd. & 5th. August, wrote Dr. Archibald " Brown of Mount Florida, who conducted the treatment , Mr,A. " experienced the first symptoms of his illness,a slight " difficulty in pronouncing certain words. On the 6th. " August on landing at Greenock after a sail on the river " he found himself unable to speak :but on arriving at " Glasgow , the power of speech had returned. For about ten " days afterwards he felt an occasional numbness of the left " cheek and point of the tongue, and experienced a distinct- " ly metallic taste. On the evening of 16th. August, he sud- " denly suffered from a choking sensation, followed by con- " traction of the left side of the face and loss of speech. " He was quite intelligent at the time , but felt some con- " fusion of mind. The power of speech returned in about an

" hour, but from the 19th. to the 31st. he became speechless
" four or five times. During all this time he was unable
" to make the simplest calculations , and could not spell
" words correctly with the single exception of his own name."
" When I saw him on the 31st. August. there was a little
" permanent aphasia, he could not write very accurately and he
" was suffering from severe pain , chiefly in the back of the
" head, which set in some time after the onset of the aphasia.
" Dr. Mc,Call Anderson for reasons stated in his book sus-
pected these symptoms had a syphilitic basis , and put the
patient on inunction of 1 drm of Shoemaker's mercurous ole-
ate ointment daily.
" In a few days after beginning its use, improvement in pro-
" nounciation and power of speaking set in. In three weeks
" all the symptoms had disappeared !

I will conclude my section on Transitory Motor Aphasia with a reference to two other conditions in which such a loss of speech may occasionally be found.---- I refer to the occurrence of Aphasia as an initial symptom of Tubercular Meningitis , and to loss of speech during Lactation and the latter months of pregnancy.

The literature of the two incidents would appear to indicate that they are by no means common, I trust therefore that the cases I wish to put on record here will not be altogether without interest as a further contribution to each subject;

ON APHASIA AS AN INITIAL SYMPTOM OF TUBERCULAR MENINGITIS.

CASE I :(Personal observation) HEMIPLEGIC APHASIA [illegible] as a prelude to TUBERCULAR MENINGITIS.

TUBERCULAR MENINGITIS is a disease which as every clinical student knows, is protean in its manner of onset; In reference to this affection, Bristowe in work on medicine writes " Sometimes the first indications of disease are " furnished by dulness, strangeness or wildness of manner, " by impairment of memory or defect of speech " The pathology " of the disease, moreover, is such that the occurence of an aphasia as a primary symptom might at first sight be regarded as no unlikely contingency; yet while I have no doubt many such cases must at one time and another have been under observation, I find the records of them remarkably few. I have only been able in the course of my reading to find two similar histories illustrating the occurrence of a definite aphasia as the initial Symptom of Tubercular Meningitis. One of these is recorded by Schutz in the Prager Med Woch, for 1881, and the other by Déjerine in the Rev. de Médicine for 1885. The latter is further interesting in that like my own case the aphasia was ushered in by several attacks of a Transitory character; but I shall give a resumé of both cases after first detailing the one which came under my own notice.

CASE 1 :(Personal observation) TRANSITORY APHASIA occuring as a prelude to TUBERCULAR MENINGITIS.

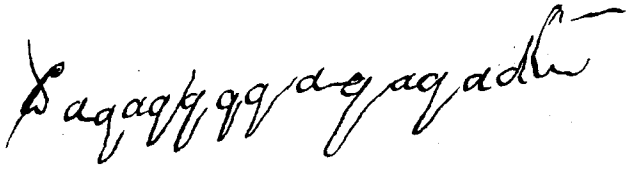
The patient, J Bowes. aet 28 years, came under my notice in October 1898, suffering from a slow form of Phthisis Pulmonalis. He had occasional attacks of haemoptysis, but for the first nine months he was under our care, always recovered sufficiently to undertake light work between the exacerbations of his illness. Latterly the wasting and Hectic fever be-

came more marked. On 7th. Decr. 1899 we were called suddenly to attend him for another attack of haemoptysis somewhat severe in character. Under treatment he rallied, but naturally remained somewhat blanched in appearance. In the course of a few days time any traces of Blood had left the sputum and on the 11th. of December he had every promise of recovering, temporarily at least , from the immediate dangers that threatened him. Three days leter, however, (on the 14th. Decr.) we received another urgent message from his wife stating her husband was apparently going out of his mind. Mrs. B. shortly after awaking that morning, had become strange in his manner, and spoken in a peculiar way ;his wife at first attributed this to delirium such as he had had previously, but when she could not understand her husband's speech he became more incoherent and excited than ever. At last it struck her that he wanted to know what time it was - a common question of his in the morning - and so apparently he did, for on holding the clock in front of him he looked at it , nodded assent, and quietly went off to sleep again. He had a similar incoherent attack on awakening later in the forenoon.

I saw him at 11A.M. and found him sitting up in bed ;and first appearances did not indicate anything unusual apart from his previous history. I was surprised to find that he could answer my questions as to his general health, as to how he had slept, &c. in quite a natural tone and manner. He then complained of severe frontal headache extending above the eyebrows and back over vault of the cranium, and said he

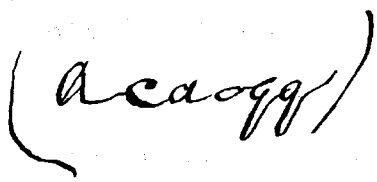
had not slept well for two nights. We continued talking for some minutes as I purposely wished by so doing to bring out any speech defect such as his wife had indicated. In a short time the flaw became apparent. He had kept up an intelligent conversation for I should think three or four minutes when his words became indistinct and blurred, running into each other and getting so jumbled up that, failing to understand him, I turned to his wife and looked questioningly at her. She, being more accustomed to his talk, could still make out the drift of his conversation while I was hopelessly lost, but in a few moments more his talk had degenerated into such ah unintelligible jargon that not even his own wife could make out a single word. Seeing from our looks and remarks that we failed to comprehend him, Mr. B. endeavoured all the more to say what was on his mind but only a jumble of sounds was the result ; among these sounds something like " Gug "- ah - yuh " seemed prominent. his inability to make us comprehend , made him come out with the jargon in such an eager, persevering, and excited manner, that I at once saw how his wife had imagined he was going off his head earlier in the day

I now told him we could not make out his speech but that as he seemed anxious to tell us something, perhaps he could write it down. He perfectly understood what we were saying and he took pencil and paper in a way that suggested he had a very easy task to perform. This however, was what he wrote :-



Looking puzzled, he commenced a fresh line more slowly-again the result :-

at which he looked in a helpless kind of way and then handed it over to me to see if I could make anything of it. He then started off again with his mumbling attempt at speech. I allowed him to go on for a few minutes , and then turning over a blank sheet of paper so that he should not see what he had already written, I again made an experiment with his writing , and told him to write down what he wished to tell us. This time he wrote in a more deliberate manner,but with a very similar result :-



and then looking puzzled , gave up attempting further. It was plainly a case of what Professor Gairdner has termed " intoxication " with a group of letters, and evidently nothing else was to be got out of him. As the patient was by this time somewhat exhausted I desisted from further examination and ordered absolute rest till I saw him again.

His wife reported to me later in the day that he remained quiet for about an hour after I left ; by that time he had recovered his speech just as he did after the previous attacks. He now voluntarily commenced a conversation with his wife, remarking that the doctor had been seeing him and that he

knew he had been unable to express his meaning properly at the time. He also knew that he had attempted to write something down and would like to see the paper only he saw me taking it away. His remarks therefore proved that he was fully alive to all that had taken place at the time of his aphasic attack. Dr. Clarke saw the patient with me later in the day ; our observations confirmed those of the morning's visit, and after speaking correctly for a few minutes his speech quickly degenerated into mere "gibberish."
We also determined the fact that when he spoke quite well (e.g. at the commencement of our conversation) he wrote quite well: His hearing was good and there was no " Word Deafness " nor " Word Blindness ".

On the following day, 15th. Decr. I again saw him, and there now a very material improvement. He conversed with me for a very prolonged period without any flaw becoming apparent I also shewed him the writing he did yesterday. He said he recognised the paper he had written on - otherwise he could hardly believe he had written it. He seemed highly amused at the composition but could give no explanation of what the letters stood for. He remembered perfectly well what he wished to tell me at the time his speech failed him ; it was in regard to the details of a previous illness which, however, had no material bearing on the present attack. It is clear therefore that in his aphasic attacks he knew what he wanted to say but was unable to say it.

The temperature of the patient was now only 100°, and the pulse 80 : the headache had disappeared and his general

appearance seemed to promise at least temporary recovery, but fortunately no prognosis was given.

I say"fortunately" because on the same evening insidious symptoms made their appearance, then more alarming ones, and within a week the man was dead from a clear attack of Tubercular Meningitis. First there was a return of the Headache ;then his memory at times became defective;like amnesia generally, this first exhibited itself in the non-remembrance of proper names, that of his only child " Dolly" having to be constantly recalled to him. I need not further describe the further progress of the case , the disturbance of vision, the alterations in pulse & temperature, the subsultus tendinorum, The curled up posture as he lay in bed like a ball of wool, the ensuing paralysis,irregular breathing, deepening coma, and then the end. These were all but a common variety of symptoms of this fatal malady. I quote the case mainly to shew how all these symptoms may be ushered in by initial attacks of an aphasic nature.

I regret that in this case a post mortem was absolutely denied me and that I had to rest content with the history above set forth. We may however form a fair estimate of the lesion in this case;but before doing so , a resumé of the other two cases I have referred to may prove of interest.

(over)

CASE 2 (Schutz) - APHASIA as an initial Symptom of Tubercular Meningitis.

F.W. Elsner gives the following summary of this case which was apparently believed to be the only example of the kind recorded up till that date.

" In the Prager Med. Woch No 31. 1881. Schutz relates the case of " a man, who, without any other disorder than an enlarged elbow " joint, suddenly got disturbance of his speech without other " Phenomena of Paralysis. He could understand and write down " words but could sometimes not articulate words even if spoken " out for him. The Aphasia increased and other symptoms appeared- " headache, paralysis of facial nerve and apathy, alternating with " great disturbance and excitement. On the 12th. day stiffness & " pain in the nape of the neck appeared: the right eye was turn- " to the right :, and its pupil was dilated. There was facial hyper- " aesthesia, rales in both lungs, slight cyanosis, and incon- " tinence, complete coma, sterorous breathing, retention of urine, " and finally death. The temperature was elevated at night to " 38.6 C. (101 . 5 F) It was at first thought that there was " embolism of the artery of the Sylvian Fissure ; but as there was " no cardiac lesion the idea was abandoned and tubercular mening- " itis resulting from tubercular chronis bone disease , with tub- " ercles in the Brain substance near the centres of speech (3rd. " left frontal convolution) was thought possible. The Brain " however was found free at the post mortem examination whilst " there was an aggregation of Tubercles and copious exudation on " the Pia Mater of the left aperculum, as well as in the Sylvian Fissure ; less on the right side.

(the)

"The Aphasia must therefore have been connected with the "primary appearance of Tubercles in the Pia Mater of the left "hemisphere, and the histological changes which such an affect "ion must cause in the Brain substance in its neighborhood. "All the other rare cases which have had Aphasia in tubercular "Meningitis had the affection better marked on the left side ; "no other case gives it as an initial symptom, it usually ap- "pearing late. The post mortem examination further shewed "acute general tuberculosis,right lobular pneumonia,and "and caries of the Right Ulna, which was regarded as having "been the starting point of the malady."

Case 3 (Déjerine) Aphasia in a case of limited "Tubercular Meningitis!

(Rev. de Med, 1885 p.175.)

This case is quoted by Bastian as an illustration of Aphasia resulting from damage to the commissural fibres in the vicinity of the Island of Reil. It will be noted that there were several transitory attacks of loss of speech and that the symptoms were due to a developing Tubercular Meningitis :

" A compositior , aet 20, suffering from Phthisis, was ad- " mitted to the Hôtel Dieu , May 6th. 1884.On July 16th. at " 8 A.M.the Sister when speaking to him,noticed that he had a " great difficulty in pronouncing words. Twelve hours after- "wards the difficulty had disappeared. The next morning " Déjerine says :At the hour of the visit a certain amount of " motor aphasia was noticed in the patient; he pronounced

" with difficulty certain words and certain letters." D " for
" instance could not be pronounced. There was also a certain
" degree of paraphasia, but no sensory aphasia :the patient
" easily understood what was said to him,and what he read.
" He wrote spontaneously and from dictation without mistakes:
" His face was slightly paralysed on the right side.
" Speech again became normal during the day, but in the
" evening the Aphasia was found to have returned . The next
" morning (18th.) it was yet more pronounced, though still
" only of motor type, and without agraphia. The right arm
" as well.as the right side of the face was now weak. By the
" following day this was still more marked as was also the
" aphasia " .....".On July 20th. the patient
" had an attack of epileptiform convulsions: many other
" attacks followed at short intervals during the day, and he
" died comotose at 2 AMm.on the following morning."
" The necropsy was made with grest care, The lesion was found
" to be a limited tubercular meningitis, involving the Island
" of Reil and parts of the ascending frontal and parietal con-
" vulsions, (while the convolution of Broca was intact throughout its whole extent.
" The whol e of the Island of Reil was covered by a fibrino-
"purulent exudation, containing inumerable granulations attach
2 ed to the vessels which penetrated into the brain sub-
" stance so that the subjacent white fibres must also have
" been effected.

It will be noted that in all the above cases of Aphasia occurring as an initial symptom of Tubercular Meningitis the cerebral trouble was secondary to a tuberculosis pre-existing elsewhere. In the case of Schutz it was secondary to tubercular bone disease: in my own case, and that of Déjerine, to Phthisis Pulmonalis. In the two latter the initial aphasic attacks were also of a Transitory character; and in both cases a rapidly fatal termination ensued within a week.

In the cases of Schutz and Déjerine there was aphasia but no agraphia and the damage was apparently due to the tubercular deposit affecting the commisural fibres in the neighborhood of the Island of Reil. In my own case loss of the faculty of writing was present with the loss of speech, and the lesion probably affected the corresponding cortical centres. I think the most probable explanation is that a tubercular deposit or exudation near the foot of the Sylvian Fissure affected the vascular supply to Brocas' area. In the case of a diminished blood supply to any portion of the cerebral cortex the cells of that area might still receive sufficient nutriment to remain structurally intact, and even after periods od rest to carry on their allotted functions' but in the event of an excessive demand they would fail to respond. If If Brocas' convulution were the area concerned we might thus find a loss of speech occurring after a moderate conversation. This was exactly what happened in my patient. The condition indeed, reminded me at the time of an affection described by Dr. Hinshelwood under the name of "Dyslexia " in which the

patient became word-blind after reading a few lines of print, Adopting the same nomenclature my case might fitly be termed one of " Dysphasia " rather than Aphasia.

The results of an interferance with the vascular supply to the cortex by a tubercular deposit or exudation along the vessels, might also be compared in its results with those sometimes produced by a syplilitic endarteritis or by atheromatous degeneration.

As a deduction from the above cases, it is clear that occurrence of an aphasia even of a temporary character, in a patient already suffering from any form of tubercular disease, should be looked upon with the gravest suspicion, and regarded as the possible precursor of furhher meningeal trouble.

ON " PUERPERAL APHASIA "

The late Sir Frederick Bateman of Norwich, in his notes of A case of Aphasia occurring during the puerperal period, remarks on the few references to this subject in our literature and pays a no doubt well deserved compliment to the paper of a Continental observer, M.Pompon. He further refers to the rarity of the affection by pointing out that in a record of 8000 puerperal cases by M. Stroganoff, not a single instance of the complication is mentioned, and adds that he can find no reference to the subject in any of our classical works on Midwifery.

The affection is certainly one seldom met with & Bateman does not mention it in his great work on the Disturbances of Speech ; and the contributions to the subject elsewhere might ~~also~~ almost be reckoned on the fingers of one hand. Nevertheless I think Sir Frederick Bateman's remarks hardly do justice to his fellow countrymen, and especially - if I may be pardoned for saying so - to those of our own Glasgow School.

I find that on referring to Dr.Leishman's System of Midwifery, published in the same year as Bateman's Lectures, a distinct note of the condition , and also a further reference to a most valuable paper by Dr.Finlayson published in the Glasgow Medical Journal for 1874, a paper , by the way, which even in the matter of priority seems one of the earlier contributions to the study of this affection.

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To return to the subject, it is at once apparent why cases of loss of speech during the puerperal period should

be of only rare occurrence. Women during the Childbearing period are in the prime of life : the bloosvessels are ~~as~~ *yet* elastic and without the signs of degeneration which make their appearance in the decades following the climacteric . Unless therefore a childbearing woman exhibited some definite cardiac or vascular lesion , the occurrence of an aphasia might be looked upon as a very remote contingency . The factor which is believed to act in producing a puerperal aphasia and the only satisfactory one to my mind put forward to explain its occurrence in the absence of cardiac or other apparent lesion, is the greater tendency to coagulation of the blood at this time. This factor is one which we have good reason to know is present not only during what is strictly termed by obstetricians " the puerperal period " but also during the later months of pregnancy. Possibly this was what induced M. Poupon in his paper on the subject of Puerperal Aphasia to include under that term not only cases of loss of speech occurring soon after delivery, but also cases occurring during the period of pregnancy. I think it can at any rate be shewn that such a classification has a sound pathological basis, and I also follow it with the greatest pleasure in that it gives me the opportunity of bringing forward a most interesting case of my own - a case in which the aphasic symptoms manifested themselves towards the end of the 7th. month. of pregnancy and continued present in a greater or less degree right over into the strictly puerperal period.

Case of Mrs.M.(Puerperal Aphasia " occurring during the later months of pregnancy)

I have already referred to this case in detail in the previous part of my thesis as an illus-tration of that form of Aphasia in which ,while spoken speech is lost, the power of written speech ispreserved (Aphemia). It is therefore unnecessary for me to do more than summarise the case here so far as it bears on the subject of Puerperal Aphasia, and to refer the reader for additional particulars to the preceding part of my paper.

Mrs. M. aet 27 years, with no evidence of cardiac or vascular disease, and with a good family history ,was suddenly affected with total loss of speech in the latter half of the 7th. month.of her fourth pregnancy. She retired to bed in her usual health on the evening of 16th. Sept. 1896 ; On the following morning after sudden emotional excitement she lost her speech for the period of less than an hour's duration ; later in the same forenoon she had a second very transient loss of speech, this time more gradual in its onset but also passing off in less than an hour's time. At 3 p.m.of same day embarrassment of speech again manifested itself ; the patient was observed to speak more slowly and with increasing difficulty until at 9 p. m. this culminated in total loss of speech for the third time.Next morning she remained in statu quo and at the time of my visit I found a condition of complete motor aphasia without agraphia (" aphemia ")She could not articulate the simplest words such as " yes" or

" no " nor could she repeat them after having them pronounced. Yet she apparently understood everything that was said to her, and could make replies either in pantomine or written speech. There was no other paralysis on this date although definitely looked for , and it was only on the following day, 19th. Septr. that we found a distinst paresis with loss of sensation in the right upper extremity, and a numbness in the region of right angle of mouth. On this day, also the patient had an attack of phlebitis and thrombosis of the veins in the right lower extremity.

Speech returned slowly but progressively from about the 3rd. or 4th. day after the attack and in a few weeks time she had begun to string together simple sentences and to dispense altogether with the help of pantomine.

On the 26th. Novr. 1896 (10 weeks after the attack) she was confined of a healthy full time child. Labour was natural and easy. Mrs. M. at this time still spoke in a slow hesitating style, and if flurried , stumbled in her speech ,but it could not be said that any relapse in her condition followed delivery. With rest and care she progressively improved, and although she could not speak with her former freedom of utterance for several months, she ultimately made a perfect recovery : Coincidently with the improvement in speech the numbness and paresis of right upper extremity also gradually disappeared.

There were no disorders of lactation.

There was no evidence of Syphilis in the personal or

or family history. I have already noted the absence of cardiac disease and there was no history of rheumatic fever. Pyhsical examination of the patient revealed nothing of consequence apart from the lesions referred to.

After History of Case. Two years later (in 1898) Mrs. M. again fell in the family way. She began to exhibit at an early stage signs of great nervousness, and a possible later renewal of her former aphasic illness was anticipated with some apprehension . Dr. Longwill of Rutherglen under whose care she remained decided „ and I think wisely, to terminate the condition. Abortion was purposely induced at 2½ months. I shall afterwards give my reason for stating that this was not only the right course to pursue, but indeed the only justifiable in the circumstances. Mrs. M. made a good recovery ; the signs of nervousness disappeared ; and there has never been any recurrence of the Aphasia.

The only other contributions to the subject of Puerperal Aphasia which I have been able to find are those of Sir Frederick Bateman, Leith Napier, and Finlayson in our own country :, as those of Lewandowski and M.Poupon in continental records. In the following table I have endeavoured to summarise the principal records of these observers so far as the literature has been available to me. I trust this epitome of the subject will prove not only intrinsically interesting , but also serve as a basis for the pathological discussion of a somewhat unusual affection.

RECORDS OF PUERPERAL APHASIA (VARIOUS AUTHORS)

Author of Case	History of Attack	Remarks
Case 1. (Finlayson)	2nd. Confinement at age of 32 years followed 10 days after delivery by complete aphasia and slight Paresis of Right Side. Partial Recovery of Aphasia. 3rd. Confinement at age 35 years followed by complete loss of of speech: numbness & paresis of Right Hand but no agraphia ; aphasia persistent.	No Cardic Lesion Note partal recovery in the first instance and the recurrence of the Aphasia in a subsequent pregnancy.
Case 2. (Finlayson)	9th. Confinement Labour natural but followed 3 weeks after delivery & during convalesenne by aphasia & Rt. Hemiplegia . Aphasia passed off in a few days : Hemiplegia in 3 weeks. 10th. Confinement, aet. 39 years. Labour tedious, followed on 20th. day by complete Aphasia & marked Hemiplegia of Rt. Side : Aphasia persistent ; ensuing rigidity of paralysed limbs.	No Cardic Disease Note Transitory nature of first attack and again the recurrence of the Aphasia after the subsequent confinement.

(over)

Case 3. (Lewandowski)	Confinement followed on 10th.day after delivery by sudden loss of speech without unconsciousness : Next day could say "no" : recovery of both speech and writing in 3 days time.	Note Transitory nature of attack.
Case 4. (Lewandowski)	Confinement (difficult Labour) followed on 9th.day by rigor headache pyrexia & slight tetanic convulsions ;about the 13th.day.loss of speech although still conscious & able to move limbs and tongue freely :Death on 17th.day.	P.M. Inflamation of Brain and membranes & in the left anterior convolution a partly decomposed focus the size of a nut;also several purulent foci in lungs & suppuration of the veins of the Ovario-uterine Plexus
Case 5. (Leith-Napier)	Confinement :- followed on 17th. day after delivery (on being subjected to considerable excitement) by sudden Aphasia : followed 3 days later by partial paralysis which culminated in complete hemiplegia 2 days afterwards,The patient slowly improved and eventually recovered.	Case attributed by Napier to Embolism of left mid.Cerebral artery: Note eventual recovery:

Case 6. (Bateman)	1st. Conft: (aet 22 yrs) natural labour, At the 7th. month of this pregnancy "she had some obscure symptoms with some embarrassment of speech, which lasted about a month." 2nd. Conft: (aet 23 yrs) natural labour & unattended by any straining or unusual effort: Early in 7th. month of pregnancy, paresis of rt. arm & rt: leg ; principally former : At beginning of 9th. month. embarrassment of speech noticed, culminating in conplete aphasia 6 days after delivery: no loss of consciousness : On 8th. day after delivery Batemen noted Rt. Hemiplegia: Died from exhaustion 6 weeks after confinement.	No Cardic disease and no history of rheumatic fever: allways enjoyed good health: Note occurrence of Aphasia during pregnancy ; Transitory nature of first attack: and recurrent attack in subsequent pregnancy.
Case 7. (Poupon)	Conft. (Aet 24 yrs) ; Second day after delivery patient seized with Aphasia and rt. Hemiplegia : also Word - Blindness but not Word - Deafness:	Attributed to Embolism of left mid: cerebral artery.
Cases 8 & 9. (Poupon)	Puerperal Aphasia associated with Lacteal derangements (see later)	
Case 10. (Gignoux)	The Aphasia in this case recurred in two successive pregnancies.	

Case 11. (Self.)	4th. Conft: (aet 27 yrs) In latter part of 7th. month of 4th, pregnancy sudden onset of total motor Aphasia (without Agraphia) : two days later slight paresis of Rt. hand: Phlebitis & Thrombosis of Veins in Rt. Leg. On the 3rd. day could say " No " and " Yes ";& progressive improvement with ultimately complete recovery : Labour at term natural : No Lacteal derangements: Abortion purposely induced in subsequent pregnancy & no return of Aphasia:		No Cardic lesion : Note Transitory nature of attack.

In connection with the above summary of cases the following points of interest might be noted. : -

1. NATURE OF LABOUR: This has apparently little to do with the occurrence of the aphasia: In only two of the cases is labour stated to have been difficult ; one of these was followed by utero-ovariam trouble and the occurrence of a secondary pyaemic focus in the left anterior convolutiob. In one of Dr. Finlayson's cases the labour was also difficult, but this is counterbalanced by the fact that in the same case a transitory aphasia followed a previous confinement when delivery was natural & easy. In dther cases the aphasia manifested itself long before natural term.

We may therefore fairly conclude that with the possible ex-

ception of pyogenic cases, the severity of the labour has little if anything to do with the occurrence of Puerperal Aphasia.

2. LACTEAL DERANGEMENTS. M. Poupon mentions that in two of his cases there was an evident relation between the lacteal secretion and the appearence of the Aphasia ;in one instance however, the lacteal disturbance preceded the cerebral accidents and in the other it followed them : in the second case it is stated from the moment the left hemiplegia & aphasia were observed , the lacteal secretion ceased in the right breast, while in the left (the paralysed side), it appeared to increase and was much more abundant than usual. M. Poupon considered this increase was due to " Vaso-Motor" paralysis . Bateman , after noting the above observations , goes on to say that in his own case there was galactorrhoea in both breasts on the second day after delivery : next day the supply was perceptibly diminished , and on the 5th. day the child had to be weaned because the supply was gone : In the other recorded cases I find no note of any lacteal derangement & in my own case there was no trouble with the breasts: The flow of milk was natural hut at the end of the first week I purposely weaned the child to give the mother every additional chance ofrecovery.

I have referred to the lacteal functions , because stress seems to have been laid on them by both M. Poupon & Bateman. At the same time I cannot help remarking that disorders of lactation are of by no means uncommon occurrence,in the prac-

tice of every obstetrician , and I do not see that the incidence of a galactorrhoea or the reverse helps as much in our researches as to the origin of Puerperal Aphasia .

3. TIME OF OCCURRENCE, and its reference to PATHOLOGY.

In all the cases quoted above the loss of speech manifested itself either during the three last months of pregnancy or in the month succeeding delivery. This period corresponds with that in which numerous analyses of the blood have shewn it to be most profundly altered. These analyses are referred to by most recent writers on Midwifery and Leishman in his work gives a brief epitome of the researches of M.M. Andral & Gavarret which are still recognised as authoritative on the subject; " In the earlier months of pregnancy it would " appear that the blood deviates little from the normal stand- " ard . ... .. In the later months however, the blood is char- " acterised by a remarkable diminution in the number of Red " Corpuscles, and a considerable increase in Fibrin, while the " proportion of albumen is somewhat diminished ... .An " estimate has been made by the same observers according to, " which they assume that, if we suppose the average number of " red blood corpuscles in the blood of healthy women we are not " pregnant to be represented by the number 125, the average in " women towards the end of pregnancy is probably not more than " 115. If, in like manner, we take 300 as representing the " physiological average of the fibrim, the proportion of that " constituent up till about the sixth month may be set down at 250, while from this period onwards, during the last three

" months of gestation, It steadily increases in quantity and reaches as high in extreme cases as 480 .....These phenomena adds Leishman," are further interesting in connection with the Occurrence of Thrombosis in the pregnant state " The observations regarding the increase of the fibrin and extractive matter are also quoted in the most recent volume of Playfair's work and in regard to them he adds that they go far " to explain the frequency of certain thrombotic affections observed in connection with pregnancy and delivery. I hope to show inthe next paragraph that these researches throw direct light on the causation of Puerperal Aphasia ; and the fact that the condition of the Blood undergoes profound alteration during the latter 3 months of pregnancy as well as during the period succeeding delivery, was my reason for affirming that M.Pompon's classification has in fact a sound pathological basis.

4. NATURE OF LESION. The Aphasia does not as a rule depend on any pre-esisting cardiac or vascular lesion. In most of the cases it is distinctly stated that valvular heart affection did not exist, and there was no history of Rheumatic Fever. The general consensus of opinion seems rather to point to thrombotic influences resulting from the altered composition of the blood in pregnancy. Some of the cases have it is true been referred to embolism of the left middle cerebral artery, but in connection with the primary formation of such an embolus it is just conceivable that thrombotic influences were at work in some side eddy of the blood stream

and even in the cases attributed to embolism the later history often points to at least additional thrombotic extension from the original point of blockage . Thus in Leith Napier's case, attributed to embolism , the lady 17 days after confinement suddenly lost her speech; this was only followed 3 days later by partial paralysis, and again two days later by complete hemiplegia . Clearly to my mind secondary thrombotic extension affected the neighbouring cortical areas and accounted for the later paralysis. Again, in regard to his two cases Finlayson remarks " In neither could we detect " evidence of any valvular disease of the heart : neither of " them had a history of rheumativ fever; the age of the first " woman was 35, and of the second 39 :we may therefore fairly " suppose that the mischief was due to the tendency which puer- "peral women have to the formation of clots in their vessels, " and so to the subsequent occurrence of embolism in various " forms." Bateman is inclined to attribute his case to a " thrombotic origin and his concluding remarks are as follows:- " From the comparitive rarity of the association of loss of " speech with the puerperal state I hesitate to venture upon " any decided opinion as to the pathology of the above case. " The symptoms could scarcely be due to any merely transient " cause, for they had existed in a modified form for 3 months " before parturition & became intensified a few days before " labour ; the absence of any cardiac lesion & the gradual de- " velopment of the nervous symptoms would rather point to cere- " bral thrombosis than to embolism."

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In my own case the history of the attack is somewhat peculiar. There were two very transient attacks of Aphasia on the forenoon of 17th. Septr: the former of these immediately following on sudden emotional disturbance ; in a third attack later on the afternoon of same day the embarrassment of speech was unquestionably gradual in its onset ; only after the lapse of several hours did total loss of speech supervene, and it was not till about 36 hours after this again that paresis and loss of sensation in the right arm could be detected although definitely looked for from the beginning. Whether the two very transient attacks of speechlessness in the forenoon were due to only a partial blockage of the blood stream , to congestive attacks , or to a local ischaemia resulting from temporary constriction of the cerebral vessels, there can, I think , be little doubt that the gradual onset of the symptoms in the third instance point distinctly to a thrombotic origin. This diagnosis receives support not only from the manner of onset, from the absence of cardiac lesion, and from the recognised features of the blood at this time , but also from what I might term strong corroborative evidence . It would be noted in my narrative of this case that on the 19th. Septr: i.e. 2 days after the onset of the aphasia , Mrs. M. had an attack of phlebitis & thrombosis of the veins in the lower part of the right leg. Now this occurrence took place in the absence of any varicosity of the veins, and in spite of the fact that at this time my patient was according to orders laid up in bed in the recumbent position. The fact that undoubted thrombosis

took place in the venous radicals of the lower limbs in circumstances most favourable to a perfect circulation is, I think, good reason for the inference that a similar thrombotic accident in the cerebral vessels was the determining cause of the aphasia. In short , I think it is fully proved that Thrombosis dependent on the altered condition of the blood , is the main factor in the production of Puerperal Aphasia.

5. PROGNOSIS OF PUERPERAL APHASIA.

One cannot help being struck with the Transitory nature of many of the primary attacks of puerperal aphasia and their usual termination in complete or at least partial recovery. In some instances this was a matter of weeks or months ; in others complete restoration of speech took place in only a few days time. It would seem either that the minute thromboses in the cerebral vessels had quickly become resolved, or else that the opening up of collateral channels had early relieved the circulation, and brought about a restoration of function in the affected centres.

The table however clearly indicates the probability of a Recurrent attack in the event of a subsequent pregnancy. In both of Dr.Finlayson's cases , in Bateman's case, and in that of M. Gignoux reported by Poupon, we are told the interesting fact that the aphasia, recovered from in the first instance , recurred in the following pregnancy. It looks as if the primary attack had left histological changes in the vessel wall which formed , as it were , a seat of election

for the formation of a fresh thrombus when the vascular conditions were again favourable to such an accident. The secondary or recurrent attack of Puerperal Aphasia invariably turned out of a more severe nature than its predecessor, and doubtless depended on a more extensive lesion. In both of Dr. Finlayson's cases the secondary aphasia remained persistent, and in one case this was accompanied by permanent rigidity of the paralysed limbs. Dr.Bateman's case , without the development of fresh symptoms, terminated fatally in six weeks. While therefore we may look upon a first attack of Puerperal Aphasia as not unlikely to prove Transitory in character, or at least to tend to ultimate recovery, yet such an attack should act as a danger signal inasmuch as the aphasia will probably recurr in the event of a subsequent pregnancy, and such a recurrence will as a rule be of a very grave character. Indeed M. Poupon goes so far as to advise that where Puerperal Aphasia has once manifested itself, means should be taken to avoid a future pregnancy. Basing my conclusions on a larger number of recurrent cases than were at the disposal of this observer, I can only add that I am in entire accord with the views which M. Poupon has expressed. In the event of the patient unfortunately becoming enceinte the only justifiable course to my mind is to produce abortion at the earliest opportunity. Such a proceedure was actually adopted in my own case , and up till date there has fortunately been no recurrence of the Aphasia.

There are several recognised forms of SENSORY APHASIA - ie. cases in which the loss of speech is due to some lesion of the apperceptive faculties. Thus a person may, notwithstanding the possession of perfect vision, be unable to read a single line of printed or written language. He can see the letters even to the dot on the " i " and the stroke on the " t " yet be unable to understand what the printed symbols represent in visual language. To use an old expression " It is all Greek to him " Such a condition is usually due to a lesion in the Angular Gyrus, and the defect is known by the term of " Word Blindness "

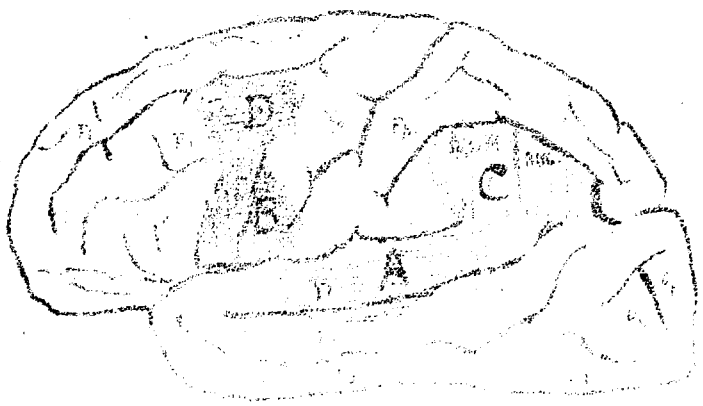
SECTION 3.

ON TRANSITIRY SENSORY APHASIAE

with special reference to some cases of

AMNESIA VERBALIS

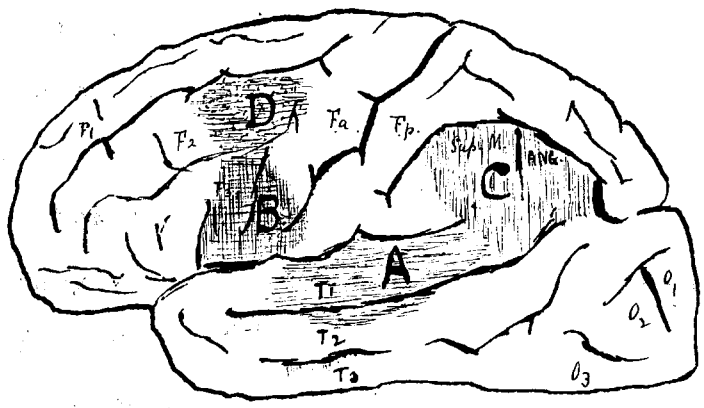
(The " Aphasia of Recollection" of Kussmaul).



A = Auditory " Word Centre " = Wernicke-Lichtheim Word Centre.

There are several recognised forms of SENSORY APHASIA - ie. cases in which the loss of speech is due to some lesion of the apperceptive faculties. Thus a person may, notwithstanding the possession of perfect vision, be unable to read a single line of printed or written language. He can see the letters even to the dot on the " I " and the stroke on the " t " yet he fails to understand what the printed symbols represent in actual language. To use an old expression " It is all Greek to him " Such a condition is usually due to a lesion in the area of the Angular Gyrus, and the defect is known by the term of " Word Blindness "

Again, a person may be in possession of perfect hearing-so perfect as to hear the proverbial pin drop ;yet when spoken to he fails to understand a single word. His sensorium cannot appreciate the meaning of the sound symbols and hence the words cannot be translated into conscious knowledge. Such a condition is usually due to a lesion in the region of the 1st. Tempero-Sphenoidal Convolution and the defect is known by the term " Word Deafness "



A = Auditory C = Visual D = Graphic-Motor Word Centres.

Word Deafness and Word Blindness are the two principal types of Sensory Aphasia, and the annexed diagram shews the position of Brain cortex in which a lesion will be productive of these two conditions.

Now it would be beyond the scope of my Thesis to go into these conditions fully, and in regard to Transitory Types of those affections I am not able from my personal observations so far, to bring under notice a sufficiency of suitable cases. I may however remark in passing, that many considerations stated in regard to Transitory Organic lesions of the Motor Area apply equally in regard to similar lesions of the Sensory Centres of Speech. For instance an obliterating endarteritis might produce Transitory attacks of Word-Blindness after prolonged reading ; and in these cases it is always well to make enquiry for Syphilitic antecedents just as in the case of motor aphasic affections: With these remarks I pass on to the more particular consideration of a third form of Sensory Aphasia. I refer to what has been termed by Kussmaul " THE APHASIA OF RECOLLECTION ". This is an affection involving the productive processes of thought and speech ; it is one in which the independent recall of words from consciousness is to a more or less extent interfered with.

Now the words " AMNESIA " and " AMNESIA VERBALIS " have by many writers been applied in a somewhat loose way to any of the above forms of Sensory Aphasia. In this paper I use the term Amnesia in its more restructed meaning as applied to the Aphasia of Recollection only. I follow the

definition of Amnesia Verbalis given by Wyllie as " a failure "to call up in the mind the appropriate word images for ideas "that are seeking embodiment in words,whether for the pur- "poses of silent thought or for those of audible speech." "Taking Amnesia in this sense let us enquire how such a con- "dition may be produced.

To begin with, it must be admitted that there are great individual differences in the powers of recollection for names, places and events,even in people who are in the possession of perfect health. Some are endowed with what is termed " a good memory ", others " a bad memory ". The personal equation is therefore a distinct factor in the production of amnesia. A certain historian is reported to have boasted on one occasion that were Milton's " Paradise Lost " consigned to the flames he would undertake to reproduce it from memory ; one reading of a poem often sufficed to to stamp it indelibly on his mind.

Converse cases in which persons are unfortunate in having an equally bad memory are perhaps more common. A friend of my own has a capital repertoire of yarns, but invariably spoils a good story by his constant failure to remember the names of the individuals concerned. Moreover in persons endowed with an average memory there is no doubt that the excitement of the passions -fear, anger, and the like - may suffice to produce a temporary amnesia of greater or less severity. He is a fortunate student who has not experienced this condition in the throes of a professional examination : it

It is a curious fact that of all the various parts of speech, the one which plays the traitor most of all to our memory is the noun ; and of nouns it is found that PROPER NAMES are far and away the greatest offenders. We have only to refer to our own individual experience to confirm the truth of this statement. How readily for instance do we forget the names of people to whom we have been introduced but five minutes previously ; loss of memory on these occasions sometimes puts one in an awkward position. Seldom , however , do we hear of such a flagrant case as that of the Ambassador who actually forgot his own name. His Excellency, according to Crichton, was at the time in St. Petersburg, and having occasion to call at a house where he was not known to the servants, wished to give his name ,but found to his consternation he was not able to remember it ;whereon he turned to to his companion and said to him with much earnestness "For God's sake tell me who I am." Later in this paper I shall refer to a similar but even more severe case which lately came under my own notice - one in which the patient temporarily forgot not only his own name but the names of all his relations. In my patient's case amnesia affected little else than proper names. In this connection it is also interesting to observe that savage races seldom employ proper names in the sense that we do. They have the habit, which comes more easily, of calling a person by a cognomen or nickname founded on some personal characteristic or peculiarity. Readers of Rider Haggard will remember how the Allen Quatermain of his books was called by the natives "Macumazahn" i.e.

i.e. " he who sleeps with an eye open " ;and further examples might be quoted to show how Kaffirs and other uncivilized races invariably steer clear of the difficult process of thought involved in remembering proper names. In an amnesia affecting proper names it might therefore be affirmed that one was reverting to the mental type of his barbarian ancestors.

Next to Proper Names the parts of speech most frequently affected in Amnesic conditions are Concrete Nouns ;after these , Abstract Nouns ;much more rarely Verbs,Pronouns , Adjectives and Prepositions. It is a somewhat significant fact that this order of events which actually occurs as a matter of clinical observation , is in exact agreement with with the law previously laid down by logicians and grammarians. These latter had already pointed out that the naming of an object involves a complex mental process, and one only acquired at a late stage in the evolution of language. The question is fully debated in works on philology and I need not further discuss it here ;but the views I refer to have been put very tersely by Mr. Crosskey in his discussion of Professor Gairdners Paper on Aphasia before the Philosophical society of Glasgow. On that occasion this gentleman is reported to have stated " that naming objects "was an exceedingly difficult thing , and involved a process "of thought difficult for a child to accomplish ;and there - "fore it was found that the verb, of all utterances ,was the " first object, and that to give a name to an object ,shewed "a considerable amount of predication and will ; so that the

" arrest of the faculty of the mind to grasp proper names,
" instead of being an arrest of a primaeval quality of the
" mind was an arrest of one of the latest and most elaborate
" actions of it ". These remarks, will, I think, be found a
" fitting introduction to a case of " Amnesia for proper
" names" which lately came under my notice.

CASE OF " AMNESIA VERBALIS " with LOSS OF PROPER NAMES OF PERSONS.(Personal Observation)

The following is a somewhat remarkable case of Amnesia Verbalis and it particularly illustrates a condition somewhat analagous to that drawn attention to by Sir William Broadbent in his paper " On a particular form of Amnesia ; Loss of Nouns " in the Medico - Chirurgical Transactions for 1884. The Amnesia in the case I am about to relate was of a Transitory nature inasmuch as it was only present for 4 or 5 days during the boy's convalescence from Influenza, and then disappeared as suddenly as it came.

I saw the patient , Robert Jones , aet 11years, on 28th. Novr. 1899 and was told that he has been somewhat feverish and"out of sorts" for about a week. He had also complained of severe headache and pains in the limbs. In spite of the general malaise he had regularly gone to school , being anxious to obtain an attendance medal;but on the day previous to my seeing him he was - much against his will- obliged

to stay at home on account of the severe headache. I attributed the symptoms to influenza which was at that time prevalent in the district, although at the time of my seeing him the feverishness had almost disappeared . Excepting for the resulting weakness, and the very severe headache, I saw nothing particular in the case until his mother casually remarked that it was very funny that he had forgotton all their names since the day previous (27th. Novr 1899). I at once made further enquiry regarding this and found the condition was first noticeable by his referring to his brother Fred (employed in a confectioner's shop) as " The Toffee Man " or as " The one who makes the toffee " His reference to his brother in this way at first caused some amusement ;but it then became apparent that he was equally at sea regarding the names of the other members of the family. He could only designate them by some attribute or personal characteristic. Thus his younger sister " Lizzie " who is his most constant playmate, he calls"the little girl ";his elder sister " Daisy " " the big girl " his brother Tom who has been recalled to his regiment " the one who is away ";his sister's sweetheart " Bert " as " her man " ;and so on. This amnesic condition for proper names remained present for almost four days ;and during the latter three of these I had the opportunity of making the following observations.

In testing his condition I was fortunate in having sufficient material for the purpose, for Mrs. Jones has no fewer than thirteen of a family. There was therefore no lack of proper

names which ought to have been perfectly familiar to him. The members of the family at home I brought individually before him but he could not name one. A common reply was "that is - - - ", here he would hesitate , and often put his hand to his head as if in deep thought. Unable to find the name he would sometimes add " I know quite well ;I will tell you in a little " ;but ultimately would have to give it up the attempt as useless. Thus in the case of his favourite sister " Lizzie " I said " Who is that ? " he tried hard to remember her name but failed and finally added " She is the little girl, tell me her name and I will tell you if it is right" So I asked him if it were " Mary " or " Tommy " (at which he laughed) or " Agnes " to all of which an immediate negative was given. He at once assented when I said the right name,and repeated it after me ;yet in only a few seconds more he had again forgotten it and the name was completely wiped out of his remembrance. His mind for proper names was a veritable " tabula rasa "

I also wrote down a number of names on the slate and he invariably pointed to the correct name of any individual indicated ;he could also point correctly to the possessor of any name mentioned in his hearing.

I asked for the Photograph Album containing present and absent members of the family as well as many near relatives. I went over the Photographs in order from start to finish. He could not name one. The loss of memory for names was absolute : yet he recognised the faces perfectly and could always indicate who they were in some round about way e.g. " He lives

at

Southend ": He is the one that does so & so" " He is coming home from India ". It will be noted in passing that he remembered certain geographical names.

I have already referred to the case of the Ambassador who temporarily forgot his own name and had to request the assistance of his friend. When asked his name my patient immediately replied " I'm BOB " ;but what his surname was he utterly failed to remember. Yet when I asked whether it was Brown, Jones or Robinson, he at once picked out the middle name ,which was of course,correct; I may say that his defective memory was put to the test somewhat severely by his younger sister Lizzie who could not quite forgive him for referring to her as " the little girl ". She threatened to bring him no breakfast to his bedroom this morning until he called her by her right name. She had asked a manifest impossibility and his patience at last getting exhausted he shouted angrily " Girl bring my breakfast at once " adding some little threat of his own which proved successful in its object.

The amnesia , I may say was almost solely confined to proper names. I tested him in order to find out any defect of memory for other nouns. I pointed out a number of articles in the room - knives, table, vases, pictures, nick-nacks, & c.and he named them all correctly. His mother at my request took note of any words he forgot, and in this way the loss of only one or two common names was detected. He is very fond of home - made " lemonade " but could not remember the name when he wished it ; at last he made his

mother understand by asking for " what she made in the jug " He also forgot the names of some flowers on the table. Among these the " Rose " ought to have been perfectly familiar to him. It is curious that although he forgot the name of this flower he associated it in his mind with one of his sisters, who has the same name. After failing to recall the name of his sister " Rose ", he pointed to a flower in the vase and said to his mother - " Tell me the name of that : that is her name ".

I think I have shewn that the case was a good and almost pure example of the loss of memory for proper names ; and the case would be interesting from that point of view if from no other. But a more curious part of the case is yet to come.

In cases of Aphasia I generally make a point of testing the patient's writing ; but I had already tired the boy with a somewhat prolonged examination, and moreover the writing test in Amnesic cases is usually regarded as superfluous. I had therefore omitted this test at my first visit,However it occurred to me as I was leaving the house to ask his mother to keep pencil and paper beside him and to ask him to put in writing any name he could not say. I was more than surprised when his mother reported to me later that while he could not write down some of the names which he had forgotten yet he certainly could write down others. I fully confirmed this report at my second visit of 29th. November, when I made a prolonged writing test. It certainly does seem a curious thing that a boy should have absolutely lost the

memory of his brother's name and yet be able to write it down; The two statements appear absolutely contradictory ; but I can only register what actually occurred in this boy.

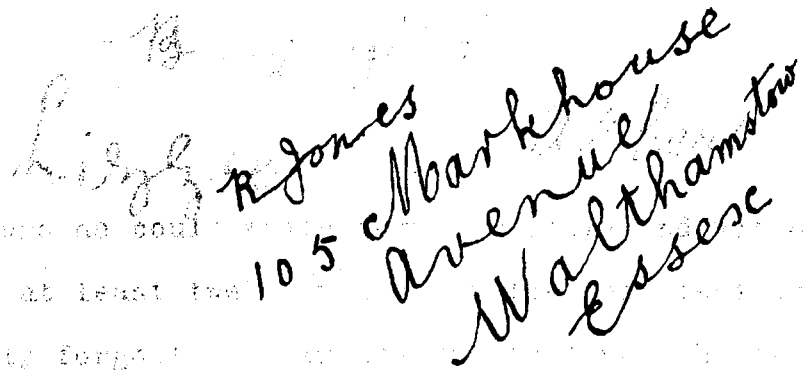
Having ascertained that such a state of matters existed, I will give an illustration of one test I put him through , with an exact tracing of his writing. He has an elder brother called " Harry " who lives in the city and whom he only sees at intervals - the last time 3 weeks ago. I shewed my patient Harry's photograph & asked who it was. He immediately said " my brother ". He failed to remember the proper name and looked steadily at the opposite wall as if endeavouring to recall it from thought. At last he gave it up, but added that it was his brother who was home three weeks ago, and that he would remember the name presently. I asked him if he could spell the name, or if he could tell how many letters were in it. Both replies were in the negative .As far as ordinary tests could go it appeared clear that his brother's name was completely lost to memory. Yet when I asked him to look at the photograph and then write the name down he at once wrote

without the slightest hesitation . I now asked him to read his own writing and he at once read " Harry " - adding emphatically, " Yes " - ' Harry '- that's it " as if he had

just made a welcome discovery. I took the slate away and asked him to repeat the name. He said " Harry ", the name being still fresh in his memory. I now purposely changed the conversation for about a couple of minutes and then returned to the same photograph ;he had already completely forgotton the name, but could again arrive at it by means of the writing test above indicated.

In a similar manner I ascertained that he could write down the names of the following members of the family quite correctly:- Tom, Will,Will taylor, George,Bert, and Daisy. There are two in the house having the same name " Will " one a brother and the other a brother - in - law of the patient's; to distinquish them they aregenerally called " Will " and " Will Taylor " respectively. This distinction came out in writing.

I now asked the lad " What is your own name ?"and got the reply " I'm Bob ",but he could not remember the surname. I told him to write it down. He at once wrote name and postal address without hesitation as follows :-



By now reading what he had written he arrived at a knowledge of his own surname.

He often talked about his school teacher with whom he was apparently a favourite, but could never remember his teacher's actual name. I asked him to try and write it down, and putting pencil to paper at once wrote

Bob

teacher

Mr Cook

In this case he had no photograph to help him. I give here some further examples of his writing on this occasion:-

Lizzie Daisy

Charley Nell

Rose Taylor

Big girl

Lizzie Albert

I found he could write down correctly and without hesitation at least twelve names which but for this test he had absolutely forgotten. In several instances he failed to write the correct name, and in these cases he only made the attempt when urged to do so.

Thus we pointed out his sisters Lizzie and Rose and asked him to write down their names : he invariably hesitated and with pencil in hand kept staring at the paper. Then when urged to try, he would write " Albert " for " Lizzie " and oddly enough " Lizzie " for his sister " Rose "; and these mistakes were uniform on repeating the test. In the case of his brother"Fred" (the " toffee man ") he also hesitated, (at last writing "Tommy" and adding doubtfully " I think that must be it ". Yet when I wrote out the following list of names - Tom, Daisy , Rose, Fred, Lizzie, Mary, - he at once pointed correctly to " Fred" as the right name.

The above notes were taken on the 29th. Novr. and confirmed by repeated tests. Out of about fifteen names he only failed to write three; There was therefore more than an element of chance in his invariably writing the remaining twelve correctly. I have no doubt that if I had continued testing him I could have got further names which he could not write down. The mystery of the case is however , not that he failed to write down some , but that he was able to write down any. It was quite clear that he could do so in the greater proportion of the names of members of his own family circle.

On the 3rd. day of my observations (30th. Novr.)an improvement was noted in his condition ;he remembered the names of his sisters " Daisy " and " Rose " He could not recall the names of the others, but when I pronounced them to him he would remember them for at least several minutes

before they again passed from his memory. Yesterday he could not remember a name more than half a minute from the time of hearing it. There is therefore in this respect a distinct improvement .

In the evening of this same day I called to make an O. E. He had just wakened up from a 3 hours sleep. The Amnesia had disappeared. His own name was the only one which tripped him up. He first called himself " James " but immediately corrected it to " Jones " - this mistake being one which might be placed in the transition stage of " Articulative Amnesia ". Subsequently to this date the memory for names remained perfect.

In order to complete the notes on above case it will be necessary to point out shortly

(1) The general Symptoms accompaning the Amnesia Condition.

(2) The After History of Case.

(1) GENERAL SYMPTOMS.

(a) Temp 99.5 on 28th. Novr.(2nd. day of Amnesia) Normal on all succeeding observations.

(b) Respirations normal

Pulse slow, 56 to65 per minute.

(b) Headache severe during first 2 days of Amneaia and completely relieved on latter day by 5 grain doses of Antipyrin. Situation of headache frontal

& extending into right temple.

(c) General feeling of Malaise during whole period of Amnesia and for some time afterwards.

(d) Sensory Disturbances of Sight and hearing On Sunday evening 26th.November, the night previous to onset of Amnesia, the patient complained much of ~~one~~ imaginary green and red lights in the room and on this accpunt asked his mother to light the gas, after which he was no more troubled . Later the same evening he woke up in a fright and said someone was staring at him in his sleep. On Monday and Tuesday 27th. & 28th. Novr. (corresponding to the first two days of the Amnesia)he had hallucinations of hearing and told his mother he had noises like someone shouting in his ears. He started up in his sleep on account of these noises and his mother had to sit constantly beside him till he fell asleep again. The presence of these sensory disturbances of sight and hearing is interesting in view of the pathological relations of the Auditory word centre to be afterwards discussed.

(e) Sound conduction in both ears normal. Sight good with only slight refractive error ;nothing abnormal noted on O.E.

(f) Intelligence of the boy good. Memory for everything but proper names perfect. When tested with simple arithmetic he could add or substract the sums almost as soon as the figures were put on the slate.

(2) SUBSEQUENT HISTORY OF CASE.

The boy was kept in bed till the disappearance of the Amnesia and then allowed up. He was first somewhat shaky on his legs but the general health gradually improved.

On 6th. Decr. there was a recurrence of the Sensory Disturbances of Sight and Hearing - green and red lights:, noises in the ears like people shouting at him or again like bells ringing :- but no loss of memory. Altogether I was pleased with his progress and anticipated his complete recovery until on January 7th.1900. an unfortunate change came over the lad's mental condition. The frontal headache returned, ~~returned~~, and although the boy appeared physically stronger than ever, his mental tone was completely altered. Formerly frank in manner , he gradually became irritable and morose : at times most mischevious and destruct -ive: behaving like a spoiled and overgrown child instead of a sensible lad of eleven years. His former respectful bearing was changed to a defiant impertinent manner and he strutted about the room full of his own importance. He was very destructive at times and had therefore to be carefully watched. One day he would be frightened for small boys, the next he would act towards them like a regular bully. On one occasion he temprarily disappeared and after much searching was found fast asleep on the top of a large wardrobe, where he"wished to die " In spite of these vagaries he shewed much intelligence on general subjects and took the greatest interest in the daily reports of the war then proceeding. There was no return of the loss of memory for proper names; The above morbid changes were noted over a period of about seven weeks:

and resulting imbecility was feared :but towards the end of March the Brain cells hed apparently taken on a healthier action and a distinct mental improvement set in. The destructive tendency disappeared also his forward and overbearing manner ;but he remained afraid of going out of the house except accompanied by one of his sisters for protection. General improvement continued until in a few weeks he expressed a desire to return to school. We did not however consider this advisable until the stability of the nerve centres was more established. The last time I saw him he was in good physical health and the mental balance was sound ; but he is not the same " smart " lad he was previous to his illness.

ON THE PATHOLOGY OF AMNESIA VERBALIS.

I have already indicated that many cases of Amnesia are of a functional origin ;but there is no doubt that recognised pathological changes often play a part in its production. The most common cause of loss of memory for names, nouns, and recent events is an amnesia of the cerebral tissue as exemplified in the amnesia during convalesence from delibitating diseases and in the amnesia of old age. The anaemia may be due to impoverishment of the blood itself, or to local ischaemia from degenerated bloodvessels as instanced by atheroma. If of specific origin we may find de-

generated bloodvessels producing Amnesia in early manhood. The case of R. B. mentioned by Ross as a good axample of the aphasia of recollection was apparently due to a syphilitic endarteritis, and the loss of memory for words rapidly disappeared under specific treatment.

A partial Amnesia is a not uncommon sequela of severe cranical injuries. I have under my care such a case at present. A young man, aet 23 years, received two months ago a severe blow on the head which rendered him unconscicus for some hours. No paralysis ensued and he was able to return to work in a fortnights time ;but from the date of the injury he has suffered from distinct loss of memory as well as a loss in the faculty of attention. He forgets many recent details in his work, and this is to him the cause of incessant worry.

A Transitory condition of Amnesia very commonly follows an epileptic seizure. I have already under the section on " Functional Aphasia " given an instance of this kind. The lad after recovery of consciousness could not remember his name nor address for a very considerable period. Such cases may be due to exhaustion of the cerebral nerve cells following the outbreak, or to some temporary anaemia of the cerebral word centres.

We now Pass from the general to the particular , and enquire what is the exact location of the cerebral centre, pathological changes in which will produce these defects of word

memory. It was first shewn by Wernicke that destruction of the upper tempero - sphenoidal region("Wernicke's Convolution") is intimately connected with what is termed " Word Deafness " As a result of his and subsequent observations it is now generally agreed that the storehouse for auditory word images is in the posterior $\frac{1}{2}$ or $\frac{3}{4}$ of the 1st.tempero - sphenoidal convolution.

Destruction of this area will produce not only " word deafness " on the receptive side of speech but also " Amnesia Verbalis on its productive side ;in other words the patient can neither understand what he hears nor can he himself recollect words which he wishes to make use of for purposes of conversation. Whether the auditory word centre is subdivided into two parts - the one concerned with the receptive and the other with the productive side - remains an open question. The cases of pure " Amnesia Verbalis " ultimately studied at the post mortem table,are few in number. Broadbent found an interesting result in his case of " loss of nouns " already referred to. Careful examination of the Brain shewed that " the 2 posterior of the 6 convolutions of the Island of Reil "had entirely disappeared :....The Angular Gyrus round the ex- "tremity of the Sylvian Fissure and the supra - marginal lobule "forming its upper margin posteriorly ,were undermined by an "extensive area of degeneration continous with that which had "destroyed the posterior insular convolutions " In order to account for the conditions found present , Broadbent advanced the

theory of a " naming " as well as a " propositioning " centre He looked upon the lesion in above case as interfering with the longitudinal commisural fibres running from the assumed naming centre in the sensory cortex to the motor speech apparatus in Broca's region. Broadbent's theory is open to much difference of opinion and Ross has criticised it at some length in his work on Aphasia. It is possible that Broadbent might claim my case of the lad Jones as further proof of his theory. In regard to the commisural band of fibres apparently connecting the visual & auditory word centres posteriorly with the motor centres anteriorly I would remark that possibly these were the conducting paths of the impressions giving rise to the hallucinations observed in that case. It would be noted that the amnesic condition was heralded and accompanied by vivid sensory disturbances of sight as well as hearing - red and green lights - ringing of bells and other noises in the ears.

Another case bearing on the pathology of Amnesia has been recorded by Rosenthal. This patient was unable to name ob- "jects at sight and had therefore lost the use of concrete "nouns ". Post mortem " besides evidence of a chronic leptom- "eningitis, an old focus of softening was found in the 2nd. "and 3rd. tempere-sphenoidal (convolution)" convolution " "being free from the " disease "

Ross in his book on Aphasia quotes this case and goes on to deduce from it that the 2nd Tempero -Sphenoidal convolution has probably to do with the recall of words to

consciousness, while the 1st.Tempero Sphenoidal Convolution being more directly connected with the auditory centripetal fibres would according to his view be the area concerned in cases of "word -deafness ". These two areas are certainly closely connected functionally ;but whether we can subdivide them into two distinct topographical centres is I think somewhat doubtful. The likelihood is that the different cortical centres overlap and merge insensibly the one into the other, especially when their functional activities are closely co-related as in the present instance. For my own part I prefer to look upon Wernicke's (1st. & 2nd. tempero sphenoidal areas) as a concrete whole and having to do with.

(a) primarily with the perception and understanding of spoken language.

(b) the recall of word memories into consciousness as occasion demands.

The latter function , being the higher of the two and the later of acquirement would in the event of damage to the centre be the first to disappear. We might thus have a minor lesion of the centre causing loss of recollection for words although the person could still recognise these same words when he heard some other person repeat them. An incoming audible word would prove a stronger stimulus and revivor of word images in the affected centre than would an idea or meaning seeking independent expression from within. There might thus result an " Amnesia Verbalis " without any accompanying " Word Deafness ". If the lesion were of a more severe form we should have " Amnesia Verbalis " plus " Word

deafness " ;if of a less severe form we should have only a limited or partial " Amnesia Verbalis ": In this last instance Proper names and next concrete nouns would first disappear according to rule. The case of Jones (loss of Proper Names) illustrates such a contingency.

This way of regarding the twofold function of Wernicke's convolution is further supported by two cases reported by Magnan and Schmidt to which I will only refer in a few lines.

(1) Magnan's Case.

This patient suffered first of all from an Amnesia of Nouns and Prepositions. Two years after the first appearance of this amnesia, marked word deafness was developed. It will therefore be noted that in what was apparently a progressive lesion affecting the auditory word centre , the higher function of the independent recall of words to consciousness was the first to be lost, and only at a much later date did the apperceptive functions become involved.

(2) Schmidt's Case is the converse of the above but illustrates the same point. Schmidt's patient was the subject of both Word Deafness and Amnesia Verbalis. During convalesence the former defect was the first to disappear while the independent recall of words to consciousness (the higher function) was only reacquired at a still later stage.

In other words the higher function is the first to be lost in cases of injury to the centre,

and in process of recovery it is thelast to be regained. The clinical history of these two cases taken together is I think strong proof of the view stated above viz :- that the Auditory word centre is not subdivided ;that it has a two - fold function ;and that the loss of one or both functions depends on the nature and severity of the lesion affecting the centre

In the case of Jones it is difficult to say what the nature of the lesion actually was, but we know that severe nervous derangements are no uncommon sequelæ of an influenza attack. The lesion, whatever its nature, apparently cleared up so far as the Auditory word centre was concerned, for the Amnesia totally disappeared . It is possible that the later mental changes were due to a spread of the lesion in the direction of the adjacent posterior cerebral convolutions.

DEDUCTIONS from the JONES CASE on the existence and functions of a GRAPHO - MOTOR CENTRE.

The facts observed in the case of Jones raise some very interesting speculations on the existence and functions of the so-called Grapho - Motor Centre. Does destruction of Broca's region entail agraphia ? It need hardly be said there are good grounds for such an assertion seeing that

many of the older as well as many of the present writers on Aphasia answer the question in the affirmative. Nevertheless there are I think very valid reasons justifying the belief that a separate grapho-motor centre does not exist ;and that destruction of Broca's area does not necessarily involve destruction of the grapho-motor images. Briefly I may summarise the reasons thus :-

(1) When Aphasia and Agraphia are simultaneously present in a case,they may not be co-equal in their extent ;in a not inconsiderable number of cases the loss of speech is more marked than the agraphia : in others the agraphia is the more apparent of the two. Moreover in the course of recovery from such a lesion the aphasia and agraphia may not be simultaneous in their improvement. It may therefore be argued that the lesion causing the one does not necessarily determine the other.

(2) Certain recorded cases with post - mortem examination seem to favour though not actually prove the existence of a lesion in the 2nd. left frontal convolution, (Exner's Centre) as the determing lesion in cases of agraphia .

(3) The occurrence of Complete loss of speech apart from any affection of writing e.g. in cases of aphemia (such as that of Mrs. M. recorded above),or in cases of Hysterical Mutism ,would also seem to favour the view of two separate centres.

(4) The fact that a pure agraphia apart from any aphasic

condition can be produced by Hypnostic suggestion as shewn by the experiments of Binet and Féré.

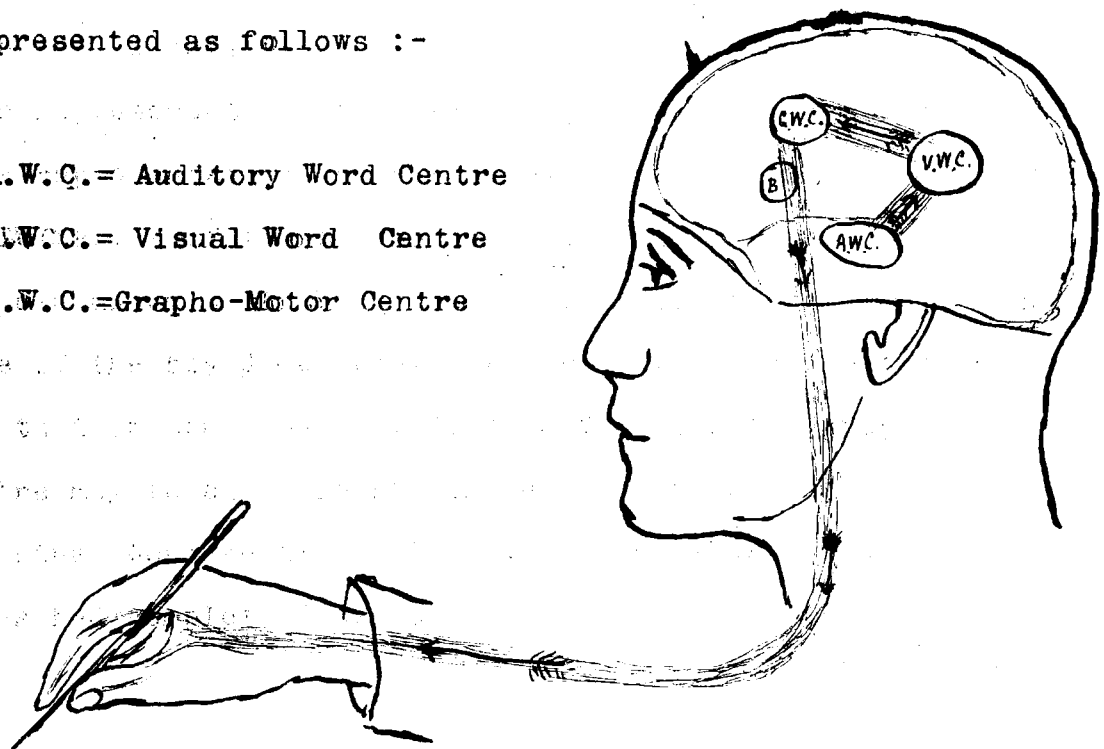
(5) To the above four reasons I would add the deductions which I draw from my observations in the case of Jones. Having thus summarised what I think are very valid reasons for the views I follow, and taking it in the meantime as assumed that there does exist in the Brain a separate " Grapho Motor Centre " -or as Bastian would term it a " Cheiro - Kinaesthetic " centre ;the question arises " how does such a centre work " ?

It is believed in most people that for writing spontaneously or from dictation a primary revival of word images takes place in the Auditory word centre, and secondarily in the visual word centre, from which latter impulses pass to the grapho- motor centre to call up the necessary groups of movements required in writing. This may be graphically represented as follows :-

A.W.C.= Auditory Word Centre

V.W.C.= Visual Word Centre

G.W.C.=Grapho-Motor Centre



But it is also a recognised fact that some people are what is termed " Visual " in type as opposed to others who in techical language are termed " anditifs "In " Visuals " the primary revival of words takes place in the visual word centre :, some such people according to Galton see mentally in print every word that is uttered ;they attend to the visual equivalent and not to the sound of the words, and then read them off usually as from a long imaginary strip of paper". Now in a " Visual" or in one approaching the visual type , the auditory word images might be awanting or destroyed and yet the person would still be able to write perfectly seeing that the grapho-motor centre receives the necessary impulse direct from the visual centre in which the word images are now primarily received. Let us now proceed in thought a stage further and suppose the visual as well as the auditory word centre is incapable from disease or otherwise of recalling to consciousness the various word memories ;can sucha person still write ? As a rule I believe he cannot : for writing may be called a mere representation of internal speech and in amnesic cases this representation would naturally exhibit all the characteristic faults of the original. But in this paper I wish to urge the old axiom that there is an exception to every rule ;and the case of the boy Jones would appear to force us to the conclusion that in some cases of Amnesia Verbalis the grapho-motor centre may to an at least limited extent act independently of the other word centres. It will be remembered that the boy Jones had complete Amnesia

for all proper names of persons. The Auditory word memories could not be recalled to consciousness. As far as testing could prove, the visual word memories for these names were likewise awanting , for he could neither tell the letter with which a name began, nor could he indicate how many letters it contained. Yet notwithstanding the absence of both auditory and visual word memories ,he in a number of instances - too many instances to be explained by any mere element of chance - correctly and without hesitation wrote down the exact name required. The grapho - motor centre seemed in this boy to have reached such a high stage of developement that it was able of itself to record the graphic symbols corresponding to the names of certain individuals. I am not aware that such an endowment has been credited to this centre by any previous writer on the subject :,but it seems to me to offer the only feasible explanation of the above case.

Charcot in his explanation of Aphasic cases ,divided persons into four groups " anditifs "," visuals"," moteurs " and " indifferents "—" anditifs " in whom the auditory word memories are particularly strong :" Visuals " in whom the visual word memories are most prominent ~~are most prominent~~ : "moteurs " in whom the motor centre for speech seems independent of the sensory centres ;and " indifferents " in whom no one form of word memory predominates .The existence of the class " moteurs " has been questioned by some writers

on Aphasia ;for my own part I am inclined not only to accept Charcot's application of the term to Broca's area, but to extend its application in a limited manner to the grapho-motor centre as well. For the very exceptional cases in whom the grapho-motor images are particularly strong I would suggest the term, "<u>GRAPHO + MOTEURS.</u>" The recognition of such a class would, I need hardly add, further favour the existence of such a centre as Exner has attempted to locate.

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