begins at the anterior edge of the coronary vein, then passes forwards on the right side of the auricular septum, below the foramen ovale, lying close on the auriculo-ventricular septum, which passes obliquely from left above to right below. Just above the place of insertion of the median flap of the tricuspid valve the bundle forms a special knot-like thickening which sends processes down on either side of the ventricular septum under the endocardium, ramifying in the walls of both ventricles.⁶ The band contains not only nerve fibres but nerve ganglia, so that conduction by muscle alone is as yet unproved.

Erlanger and others have found that pinching this bundle in dogs increases the ventricular pause. A greater tightening of the clamp lengthens the pause till three or four or more auricular contractions occur for every one in the ventricle. A cut through the bundle is followed by complete diastole of the ventricle, and no stimulus can be made to pass from the auricle to the ventricle. What interests us more as physicians is that all cases of true bradycardia which have come to an autopsy since the band of His was discovered have shown extensive degenerative lesions in its structure.⁷

I would suggest, then, that tachycardia is a pure neurosis, characterized by a great increase in auricular contractions from disturbed nervous influences. After a time, from over-exertion, fatigue in the conductivity of the band of His occurs; missed impulses result, causing longer or shorter delays in the ventricular contraction. When these delays are sufficiently prolonged, cerebral anaemia is caused, followed in due course by the nervous symptoms described. Tachycardia being a neurosis, it is, like other neuroses, commonly recovered from. The condition may persist for years, and recovery may be complete, or relapses may take place from time to time.

In bradycardia we cannot be so hopeful, the ventricular pauses being due to serious structural changes in the band of His. Sclerosis of the vessels, fatty degeneration of the fibres, and an abnormal increase of fibrous tissue in the bundle have all been found. The condition is never recovered from, and the chances of sudden death from prolonged cessation of the ventricular contraction should not be forgotten. Life in true bradycardia, according to Balfour, is rarely prolonged for more than three or four years.

As regards treatment, *rest*, both physical and mental, so far as possible, is of greatest importance. In my case, strict confinement to bed prevented convulsive attacks. In another recorded case the patient "dodged" these when threatening by getting up in bed on his hands and knees, and keeping his head down. The head should be kept low, and the foot of the bed raised. Any deviation from health, such as indigestion, constipation, gout, rheumatism, etc., should be attended to. Massage also may be employed if the conditions allow.

In tachycardia I have pushed bromides till bromidism was marked, without good results—rather the opposite. They certainly have no effect in controlling the fits. Nerve tonics, such as valerianate or oxide of zinc, seemed to do good. The glyco-phosphates or glyco-formates might be tried, but I have no experience of these in this connexion. Strychnine or nux vomica, atropine or belladonna, and alcohol are better avoided.

In bradycardia strychnine hypodermically, with heart tonics such as digitalis or strophanthus, seem to have been of use. Osler records a case of recovery after treatment with iodide of potassium.

With a better knowledge of the causes at work in producing both conditions, it is not too much to hope for more successful methods of treatment in the near future.

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THE fourth Congress of Climatotherapy and Urban Hygiene will be held at Biarritz in Easter week (April 20th to 25th), 1908, under the presidency of Professor Pitres, President of the Medical Faculty of Bordeaux. Information relative to the Congress may be obtained from the General Secretary, Dr. P. Gallard of Biarritz.

BILATERAL LESION OF THE AUDITORY CORTICAL CENTRE: COMPLETE DEAFNESS AND APHASIA.*

By F. W. MOTT, M.D., F.R.C.P., F.R.S.

A FULL clinical account of this case is published in the *Archives of Neurology*, vol. iii, p. 401, but since then I have had the opportunity of making a more complete examination of the extent and limits of the lesion by means of serial sections through the right hemisphere in the region of the lesion and by a separation of the left hemisphere into two portions, so that the exact condition of the convolutions lying within the Sylvian fissure could be studied. Before proceeding to give a full description of the anatomical findings I will give a short account of the clinical notes of the case:

E. M. H., admitted to Claybury March 18th, 1897, married; age on first attack, 22; now aged 25.

Family History.—Father was a drunkard, and lived a fast life. Sister and brother were said to be hysterical. Some time prior to her marriage in 1894 she had an attack of hemiplegia and aphasia. The practitioner who attended her was communicated with and wrote as follows: I cannot remember the year I saw her, but I can the principal features of the case. When called to her she presented the ordinary symptoms of a hemiplegia with aphasia, but as I could not find any cause for the condition. I put it down to hysteria, and I think the subsequent course proved I was correct. If I remember rightly she had complete hemianaesthesia. The paralytic symptoms disappeared rapidly, but the aphasia remained complete for a week or more, and partial for something like a month. She completely recovered under tonics and valerian. She was married in July, 1894, and remained in good health, mentally and bodily, until the first miscarriage, February, 1895 (about six months fetus), in Beckenham Cottage Hospital, when she suddenly lost her hearing, and three or four days later developed paralysis in right leg and arm, had convulsions, and lapsed into unconsciousness. Later on she became a patient at the National Hospital, Queen Square. The following notes were sent from the National Hospital, Queen Square:

Notes from National Hospital, Queen Square.

E. H.—In November, 1894, had a fright followed by some fairly differences; seemed unnaturally worried. A fortnight later was vacant and absent-minded and stupid in the evening. Next morning, at 7 a.m., her husband found her quite helpless; had passed her water in the bed, could not understand what was said to her, but nodded appropriately in answer to written questions. During the day had another (?) fit, cried violently, but had no definite convulsions, but her husband noticed the bed shaking violently.

She was admitted to Beckenham Cottage Hospital completely paralysed on right side. After about a month began to recover her speech. The arm recovered rather rapidly and completely. On coming home the husband noticed she could not feel a prick or a pinch on the right leg (which was oedematous) but she could one on her arm. She was able to read throughout her illness. Her leg has rapidly recovered power; complains of pains in right shin. Memory unaffected, no alteration in disposition. Said not to be emotional.

Personal History.—Married in July, 1894. Had premature birth two months after onset of hemiplegia.

Family History.—No history of paralysis. One brother died of fright, aged 27; said not to be insane.

In 1893 patient had a similar attack after a fright, supposed ghost; was unconscious for three hours, lost her speech for two weeks, but there was no paralysis, and she rapidly recovered.

On admission, May, 1895. Right leg weak. Can only say "Yes" and "No"; understands all written commands; cannot write spontaneously. Apparently has attacks of giddiness. Nystagmus. Jaw deviates to R. Left side of mouth acts better than right. Deaf. Slight anaesthesia in right leg. Left knee-jerk more than right.

Notes of an Examination shortly after Admission to Claybury Asylum by Myself.—She is quite unable to comprehend a single word spoken to her, and is apparently stone-deaf. I was unable to convince myself of this, although it is probable, for a tuning-fork applied to the skull on both sides did not attract the slightest attention; but she was then absorbed in watching her observers, therefore it may have been due to inattention. The patient can understand written or printed language, and this is the only means of communicating with her. She appears to understand simple sentences—for example, Question I: "How long have

* This paper was on the list of papers to be read at the Medical Section of the meeting of the British Medical Association at Exeter.

you been married?" was written down, then 1, 2, 3, 4, 5 years underneath; she expressed by gesture and sounds dissatisfaction; then 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11 months was written down, she still expressed dissatisfaction; finally, two years and 1, 2, 3, 4, 5, 6 months was written down; as soon as six was reached she became very pleased and excited and spontaneously said "is that." The time was nearly correct.

Question II. "How many children have you had?" and the numerals 0, 1, 2, 3, 4 were written down, at which again she showed signs of dissatisfaction. I then wrote "miscarriages 2," at which she became very excited, and said "is that." She makes exaggerated attempts by vocal sounds to express simple emotions of pleasure or disgust, the former being much higher in pitch, *ee* equals pleasure with elevation of the head; *ah* uttered very gutturally associated with a lateral movement of the head downwards was an expression of displeasure. She was therefore able to understand the meaning of simple sentences and to exercise reason. There is only partial word-blindness, this manifesting itself by evidence of confusion, and signs of inability to understand the meaning of a sentence when fatigue has been produced by repeated experiments.

Writing from Copy.—She can write, but to a very limited extent; for example, she copied the word *pencil*, she copied her own name, but when asked to copy *chair* she tried to write "Now copy this chair," showing that she did not understand this simple sentence and the logical sequence it bore to the previous sentence.

Spontaneous Writing.—When requested by writing to write her age, she wrote down the numbers 2, 1, 1, 1, 1, 1, as if she were unable to recall the figure 5; asked to write her address and name, she began with a correct H, but was unable to proceed further. At another time she was able to write four letters of her surname correctly. She attempted to write her address, beginning with the correct letter, but became more confused as she proceeded, until the writing was quite incoherent; the same was observed when asked to write her husband's name. She recognizes all ordinary common objects by sight, and shows signs of satisfaction when the right name is written to designate the object, but she soon tires and gets confused when asked to select one from several objects. Not only is all spontaneous speech lost, but she is quite unable to repeat aloud what she has written, to count, to say the alphabet, or to repeat her own name, although once or twice she will get out the first letter or syllable of a word.

Taste and smell were tested, and it was concluded that she possessed these senses; when ammonia was placed near her nostrils, and at the same time the written question, Do you smell this? shown to her, she became very emotional and said "is that." There is some paresis of the lower part of the right side of the face. The grasp was good in both hands, wrist-tap and triceps jerk obtained both sides. She had apparently no loss of sensibility; when pricked or touched she showed signs of feeling on both sides. The right paralysed leg was especially tested and no defect was ascertained. The heart sounds appeared normal, there was no thrill. It was further noticed that when she used her right hand in writing she apparently had some difficulty in judgement of distance, as if there were lack of association of the hand and the eye in this co-ordinate movement. There was noticeable tremor in the left hand of a coarse character, which was not, however, increased in amplitude during effort. She was discharged from the asylum suffering from organic disease of the brain and admitted to Colney Hatch Asylum, June, 1898. The following notes were made:

Heart: First sound somewhat altered, being somewhat muffled and at times reduplicated. No murmur to be heard. Respiratory and abdominal organs healthy.

Mental Condition.—It is difficult to ascertain the patient's mental condition, as she is aphasic. She is word-deaf, being unable to understand spoken language. She is unable to express herself in spoken language, is able to understand written speech, but is unable to express herself in writing. She can point out objects correctly, and can also point out her age when shown figures. When she attempts to answer a question she becomes excited, makes inarticulate noises, and laughs childishly. She is able to look after herself, such as taking her food, washing, dressing her hair, but is unable to walk on account of paralysis of her right leg, which she can only move to a slight extent voluntarily.

In September, 1898, I found her in much the same state as she was when at Claybury, twelve months previously. She recognized me immediately although she had not seen me for twelve months, and expressed great satisfaction. The following note was made by Dr. Cole on June 30th, 1899: Absolutely aphasic and also word-deaf; vision considered impaired, but she recognizes persons fairly easily; can recognize some words, as cat, dog, pig, etc., if written in capitals; she accurately associates the names with pictures of these animals, correcting, sorting out, or selecting for each its proper name. Simple sentences she could not be made to understand. (There is, therefore, a difference in her condition to that exhibited when in Claybury a year before.) Such a sentence as "Does your head ache?" was tried without result. In attempting to read she screws and rubs her eyes, and varies the distance of the paper from

the eyes, indicating either some visual defect or an inability to understand why she cannot grasp the meaning of the symbols. Her power of attention and concentration seems impaired; by her expression of face and the way in which she rubs her forehead, she seems to have sudden sharp pains in the head. Dr. Cole thus interpreted this: more probably it was an attempt by gesture to show her defect of speech through the brain disease, for the next moment she would be laughing.

The notes state that she rubs her right thigh occasionally as if painful; more probably again, this is to show that the paralysis of this limb prevented her walking. She cannot walk unsupported, but is wheeled about the grounds in a chair. There is no squint, no facial weakness; the pupils are of moderate size, equal, react to light and accommodation. She is absolutely deaf and takes no heed of any

sound, except perhaps the loudest noises, as, for example, she apparently heard the fall of a curtain rail from near the ceiling. Frequently she utters extraordinary sounds, and occasionally a few articulate syllables, but these are never intelligible. There is marked rigidity of right leg in position of full extension and some rigidity of left leg. Both kneejerks much exaggerated. Double ankle-clonus, best marked on left side. Plantar extensor reflexes present. If made to walk, walks on tip-toe and crosses one foot over the other (scissors gait); but walking is painful to right leg. Some rigidity of right arm (only voluntarily), no rigidity of left arm. Hand grasps good. Co-ordination of right hand good. Coarse intention tremor of left arm resembling that of disseminated sclerosis; steady with right hand, but spills with left. Supinator longus and triceps jerk present. There seems to be no defect of cutaneous sensation and none to pin pricks. No deformity of spine. Muscular development good. Often calls the attention of the nurses by crying out and pointing, if she sees other patients doing what they should not. *Her habits are clean.* Menstruation continues, but is somewhat infrequent. As to her mental state there is apparently some dementia, but it is difficult to determine its degree. General bodily health good.

I visited Colney Hatch Asylum, December 16th, 1901, and made the following notes: She recognized me when I entered the ward. When at Claybury she could write a few letters; she is now unable to do so. She can read simple sentences, and apparently understands the meaning. Thus: "Are you in Claybury? Yes or No?" Pointed to "No." "Are you in Colney Hatch? Yes or No?" Pointed to "Yes." "Which is Dr. Jones?" There were three doctors present and she recognized correctly

Fig. 1.—Photograph of the patient.

Dr. Jones. "Put your tongue out." Protrudes tongue, slightly deviating to the right. There appears to be a slight paresis of lower part of face on right side. She can imitate, showing her teeth and putting up two or more fingers. "Do you think you could use a typewriter? Yes or No?" Pointed to "No." "Do you hear a noise? Yes or No?" She now became emotional, and was apparently upset. A watch was placed on the side of her head, and shown to her. She shook her head, indicating that she did not hear it.

Physical Examination.—No heart murmur detected. Walks with great difficulty—crossed leg progression. Spastic condition of both legs, more marked in right. Foot drop, marked rigidity, exaggerated reflexes and ankle-clonus on both sides. Coarse tremor of left hand, none in right hand. She feels the prick of a pin or a hot spoon in both legs, but response is somewhat delayed. There is no loss of control over sphincters. The appetite is good. There is no difficulty in *swallowing or masticating her food*; she is also able to cut up her food. The attendant informs me that she gives no trouble, and that the menses are regular. She recognizes her friends immediately, and often by sounds she will indicate to the attendants that other patients are getting into mischief.

April 7th, 1902.—Signs of phthisis pulmonalis detected.
June 3rd, 1902.—Death.

Post-mortem Notes, June 6th, 1902.

The body is emaciated. *Post-mortem* staining on dependent parts. Small bed-sore on sacrum, and upon right great trochanter, and an eschar on right heel.

Skull.—Calvarium of normal thickness. Over the upper and posterior part of right parietal region on the inner surface of the dura mater is a thin, rusty coloured false membrane, the size of a crown piece, otherwise the dura is normal.

There is considerable excess of subdural and subarachnoid fluid, which is quite clear. The veins of the pia mater are engorged. The brain substance is softer than natural, and there are three large areas of old softening in the following situations:

1. Occupying the area of distribution of the anterior cerebral of the left hemisphere, and causing an atrophy of the middle portion of the mesial surface of the hemisphere involving the whole of the paracentral lobule in its mesial aspect, and the greater part of the gyrus fornicatus; in fact, the whole of the mesial surface above the corpus callosum. No doubt this lesion accounted for the paralysis and anaesthesia of the right leg. Following Dejerine, this lesion corresponds to the area irrigated by the middle and posterior branches of the anterior frontal.

If a portion of the superior operculum of the Sylvian fissure be cut away in a normal brain as shown in photograph (Fig. 2), the convolutions forming the island of Reil, as well as the convolutions (gyri transversales) forming the Sylvian surface of the temporal lobe, can be seen, together with the annectants these form with the

inferior parietal lobe and the insula. It has been shown by Campbell that the structure of the posterior third of the first temporal convolution, the transverse gyri of Heschl, and the connexions of the same with the parietal lobe, together with the posterior gyri of the insula, have a totally different histological structure to the anterior

main portion of the insula. This latter is probably olfactory in function, and the hinder portion has a structure like the temporal lobe, and probably therefore has a similar function. In the posterior third of the first temporal convolution and the adjacent portions of the transverse gyri of Heschl within the Sylvian fissure Campbell notes the existence of very large fibres and a cell-structure lamination which differentiates it from the rest of the temporal lobe; he therefore calls this the audito-sensory area.

Now this patient was stone deaf, and the complete examination of the brain by sections through the smaller lesion of the right hemisphere stained by Weigert-Pal method shows that the structures destroyed were as follows:

The Left Hemisphere (vide Fig. 3).—The posterior third of the first temporal and its connexion with the second, as well as a portion of the hinder part of the second, a large portion of the inferior parietal lobe, including the marginal and a portion of the angular. Besides this obvious convolutional destruction there was a very extensive destruction of the convolutions lying within the Sylvian fissure (vide Fig. 2). This destruction was ascertained by separating the hemisphere into two portions in such a way as to separate the temporal lobe by an antero-posterior cut through the lesion. It was then observed that the whole of the posterior portion of the Sylvian surface of the temporal lobe was destroyed, as well as the posterior portion of the insula and the annectants of the temporal lobe with the insula and the inferior parietal lobe.

The Right Hemisphere (vide Fig. 4).—The lesion was rather less extensive, but involved practically the same area; the extent of destruction of the gyri transversales and their annectants with the inferior parietal lobe and the insula was determined by vertical sections through the lesion stained

by Weigert-Pal method. It was found to correspond with the area enclosed in the dark line in Fig. 2; it included the posterior third of the first temporal and its connexions with the posterior portion of the second temporal, a portion of the inferior parietal lobe, including a large portion of the marginal and some of the angular, the transverse gyri of Heschl and their annectants with the inferior parietal lobe. The extent of this lesion is shown in the four upper photographs in Fig. 5. Vertical sections were made through Broca's convolution of the left hemisphere, and stained by Weigert-Pal method, and I could find no evidence of subcortical softening. The sections were shown in the Museum of the British Medical Association meeting at Exeter.

The right hemisphere, after stripping, weighed only 429 grams, and the left hemisphere 328 grams. As the

Fig. 2.—A normal cerebral hemisphere with a portion of the fronto-parietal operculum cut away so as to expose the island of Reil and the gyri transversales with their parietal annectants. The part enclosed in the dark line lying within the Sylvian fissure was completely destroyed on both sides.

Fig. 3.—Left hemisphere stripped, showing cortical lesion. It will be observed that the whole hemisphere is shrunken from above downwards, and that the whole of the angular gyrus is not destroyed.

convolutional pattern is complex and indicative of a good normal female brain before the lesions occurred, it follows that the loss of substance has been profound. The corpus callosum was observed to be very thin and wasted, and almost transparent in a region corresponding to the mesial softening. The splenium, which contains the commissural fibres connecting the two occipital lobes, is the part least affected.

Thorax.—No effusion into pleural cavities, but adhesions at both apices. Both lungs riddled with tubercle with cavitation of the upper lobes, especially on the right side. Right 28½ oz., left 27½ oz. Heart, 7½ oz., contracted. Muscles firm, pale brown. Aortic valves competent. Mitral valve, old sclerotic vegetations on superior surface of orifice. No marked dilatation or signs of failure.

Liver 50 oz., congested, fatty. Kidneys, right 2½ oz., left 3 oz.; capsule adherent, cortex diminished, surface uneven, probably the result of old infarctions. Spleen 5 oz., two opaque thickenings of capsule, result of old infarcts.

Cause of Death.—Pulmonary tuberculosis.

F. W. M.

Commentary.—In all cases of embolic softening it is extremely difficult to decide how far the symptoms manifested during life are due to destruction of the grey matter of the cortex cerebri in particular localities, and how far to recognizable and unrecognizable softening and destruction of the subcortical white matter, which consists not only of fibres proceeding to and from the area of cortical matter destroyed, but likewise of association systems uniting other and even remote apparently undamaged cortical areas. Where there have been a number of lesions produced by embolic occlusion, as in this case, it becomes extremely difficult to make any positive assertions in respect to correlation of function and destruction of definite cortical areas. With this reservation I will attempt, however, to correlate the lesions found *post mortem* with the symptoms manifested during life. The patient's illness may be divided into three stages, one before marriage and two after. At no time was the mitral stenosis, which caused the lesions, recognized by the existence of definite physical signs, although redupli-

cation of the first sound was noted. The symptoms were at first considered to be hysterical.

The notes obtained from the friends at the National Hospital, corroborated by a letter from Dr. Mack, show that at 20 years of age, and before her marriage, the patient (after a fright by a supposed ghost) had an attack of loss of consciousness which lasted three hours; the speech was lost for two weeks and impaired for a month, but there was no paralysis, and she rapidly recovered. There is no mention of her being deaf; presumably she was, because the attack was similar. The practitioner regarded this attack as hysterical in nature, but the loss of consciousness for three hours and the statement of the friends combined with the *post-mortem* findings, allow of a completely different interpretation.

It is probable that an embolus lodged in the posterior division of the left middle cerebral beyond the origin of the ascending parietal branch, and caused softening of the left auditory cortical centre. This would produce word deafness and a temporary sensory aphasia without any paralysis; this rapidly cleared up, and she became a normal individual as regards her speech and mental condition, for she was married in July, 1894. In the following November she had a fright, followed by some family differences; a

fortnight later she had two fits. The first fit, according to the husband's statement, coincided with the development of deafness and aphasia, but ability to understand written questions. It may be considered that this was due to embolic occlusion of a posterior branch of the right middle cerebral artery. The deafness was now absolute and permanent; it may be accounted for by the complete bilateral destruction of the auditory cortical centres. The aphasia also was complete and permanent. The second attack, which occurred, according to the husband, the same day on which he noticed the bed shaking violently, was possibly associated with the blocking of the left anterior cerebral artery. These two attacks are difficult to separate, but it is probable that the shaking of the bed was the result of clonic spasms, indicating softening of the motor area of the leg.

After being in the Beckenham Cottage Hospital at the

Fig. 4.—Right hemisphere stripped. It will be observed that the lesion is mainly confined to the auditory cortical area.

Fig. 5.—Vertical sections through the right hemisphere to show the portion of the temporal lobe and inferior parietal lobe which have been destroyed. The lower three sections are through the uninjured anterior two-thirds of the temporal lobe, and show that in this region the convolutions forming the boundaries of the Sylvian fissure are uninjured. The upper four sections are through the lesion, and the middle two show a complete destruction of the convolutions bounding the end of the Sylvian fissure. The sections have been reduced to one-third of the normal size. The area of destruction is represented in the area enclosed in Fig. 2.

end of January, 1893, we hear of her next at Queen Square Hospital, and the notes of Dr. C. Beevor conclusively prove that she was aphasic and her condition as regards speech defects about the same as when I saw her at Claybury. Her physical condition subsequently deteriorated, either from extension of thrombosis, fresh embolism in subcortical structures, or from contracture and disuse of the limbs.

I think it may be concluded that there was *absolute deafness*, and that this was due to destruction of the cortex on both sides corresponding to the region in which auditory sensory projection fibres terminate; namely, the posterior third of the first temporal, the transverse gyri of Heschl and their annectants with the inferior parietal lobe and insula, and the posterior part of the second temporal.

There certainly was not word-blindness, and the optic memory images were present, for she could associate correct words with pictures; moreover, although she was unable to write, she was able to understand the meaning of simple sentences, even shortly before her death, although she easily became fatigued.

The evidence of slight facial paresis observed cannot be explained by the obvious cortical lesion, and it was probably due to subcortical damage.

It is necessary to mention this, because M. Marie in an important paper contained in *La Semaine Médicale*, May 23rd, 1906, *La Revision de la Question de l'Aphasie*, rightly points out that the area of cortex destroyed is not a true index of the damage to the brain, and by assigning functions to areas of cortical destruction without considering the possibility of the loss of function being due to destruction of subcortical fibres belonging to other regions we have fallen into a great error. Whether in his desire to call attention to this fallacy, which undoubtedly so many people have fallen into, he has overstepped the mark in asserting that the third left frontal convolution does not play any special rôle in the function of language, and that the *sensorial aphasia of Wernicke* can no longer be accepted, subsequent careful observations will show. At any rate, a pronouncement by so distinguished a neurologist has come as an earthquake to our cherished beliefs in cerebral localization; yet he is right in asking all observers to look to the facts and not follow preconceived ideas based upon a scheme. As he states, very few cases are free from such fallacies if we consider them without prejudice.

Dejerine, in the *Presse Médicale*, July, 1906, has criticized in a masterly manner the above destructive pronouncement of Marie in a paper entitled *L'Aphasie sensorielle et l'Aphasie motrice: Localisation et Physiologie pathologique*.

M. Marie lays great stress on the loss of intelligence which lesions of the zone of language occasion, but M. Dejerine points out that a general paralytic can speak and write, but the dementia is much more pronounced than in an aphasic individual. I quite agree with him, and I think this case shows that disturbances of speech and disturbances of intelligence are processes of essentially different nature. M. Marie states: "Si, pour ma part, j'avais à donner une définition de l'aphasie le fait que je m'efforcerais surtout de mettre en lumière serait la diminution de l'intelligence."

If we look at the photograph of this patient, if we consider her acts, her endeavours to make people understand her own wants, and her desire to help the attendants when the other patients were getting into mischief, also the fact that her habits were clean, that she recognized me after a year's interval—everything shows that she reasoned, and that the mental impairment, although undoubtedly great, was more apparent than real. She comprehended her surroundings, and reasoned and acted to the best of her ability in response to the stimuli arriving in her cerebral cortex by the sensory avenues of intelligence still open. Considering the enormous amount of cerebral destruction as shown by the loss of brain weight, her mental condition compared most favourably with the average asylum patients whose brains seldom lose anything approaching the amount of loss by this patient, which must have been quite one-fourth of the total brain weight.

In 1874 Wernicke published his important memoir entitled, *Der aphasische Symptomencomplex*, and in this paper he called attention to, and explained, word-deafness,

and accurately determined the region at fault—namely, the posterior half of the first temporal convolution with perhaps a portion of the hinder extremity of the second. Word-deafness and word-blindness were, however, explained by Bastian as early as 1869, in a paper entitled, *The Various Forms of Loss of Speech in Cerebral Disease*.

Marie states, loc. cit., p. 3: "Rien, absolument rien, au point de vue de la stricte observation clinique ne nous autorise actuellement à considérer la première circonvolution temporale comme étant le centre de l'audition et Wernicke a commis une erreur manifeste quand il a pris la notion de ce centre auditif pour en faire la clef de voûte de sa doctrine de l'aphasie sensorielle."

Bastian, in his Lumleian lectures, *Some Problems in connexion with Aphasia and other Speech Defects*, *Lancet*, 1897, states that he only knows of four cases of double lesion of the superior temporal convolutions. The first case recorded was by Kahler and Pick, the second by Mills, the third by Friedlander and Wernicke, and the fourth, a case of great importance, by Pick.

In respect to defects resulting from destruction of the auditory and visual word centres in each hemisphere, there is only one case on record in which these combined lesions have existed in the two hemispheres; it was published by J. C. Shaw in the *Archives of Medicine*, New York; the notes, however, were from the King's County Lunatic Asylum, and are very meagre.

An extraordinary case has been recorded by Pick, which seems, moreover, quite incapable of being understood except by the supposition of the adequacy of the direct action of the visual word centre upon Broca's convolution even for the production of correct speech: A day labourer, aged 24, was completely word-deaf, and behaved like a deaf person, taking no notice of ordinary sounds near him. It was observed that he only noticed loud calls, clapping, or ringing of bells, and these not always readily. Yet if one shouted at him unexpectedly, he said angrily, "Don't shout at me!" and he often said spontaneously, "I can hear quite well, but I don't understand; I can hear a fly flying past me." His power of recognizing musical airs that he previously knew was quite lost. He spoke fluently, and only occasionally hesitated about the right word. He named objects shown to him correctly, but he was unable to repeat words and phrases.

The case I have reported, in my opinion, supports Bastian's views that the primary revival of spoken words during thought takes place in the great majority of persons by a subconscious process in the auditory centre, for word-deafness can alone explain the loss of speech in the first attack, and instantly that the patient became absolutely deaf from the second lesion, she again became dumb and remained so. The subconscious revival of word images precedes the correlated revival of the complementary kinaesthetic images, and these again successively incite the associated groups of neurons in the bulbar centres. It has been shown by many observers, commencing with Stricker, Galton, Ribot, and others, that different persons think in different ways, according to the predominance of the visualizing, auditory, kinaesthetic (or motor) faculties; a large proportion of individuals are, however, indifferent.

Bastian argues that "although the first stage in the revival of words seems to occur in the auditory centre, the molecular disturbance thus initiated is, however, immediately transmitted in a varying extent in two directions. It is transmitted to the visual word-centre, on the one side, and to the glosso-kinaesthetic on the other; to the latter strongly when the latter is to issue in speech, and to the former strongly when it is to issue in writing."

This patient was unable to revive in thought the words to enable her to write spontaneously, although she was able to copy a little.

The fact that she was unable spontaneously to initiate written speech, although her ability to read and to understand simple sentences (when I saw her) and to name correctly objects by means of printed or written letters and words, showed that her visual word centre was not entirely destroyed. This suggests to my mind the correctness of the hypothesis that the primary revival of the articulation of words in her case took place in the auditory centres, and I would agree with Bastian that in *motors, visuals, and indifferents*, the

primary revival of words takes place in that portion of the brain in which (in a child) speech first develops. In the child, words are first learnt by hearing certain sounds associated with certain objects, and simple thoughts connected therewith are acquired before the child has the power of articulating them. He sees an object before he can name it; he must revive those auditory impressions which he had previously heard associated with it, otherwise how can we explain the fact that a child in full possession of speech even as late as the fifth, sixth, or seventh year, if he becomes deaf will certainly become dumb, unless he is trained by lip reading, that is, unless the primary incitation to articulate speech be transferred from the auditory to the visual word centres? The facts related in this case do not accord with the statement of Bernard that the motor centre of speech may become independent of the sensorial centre which had presided over its education.

There are, however, certain exceptional persons (visualists) who, as it were, read rather than hear their thoughts and in whom (as Ballet says) the visual images of words acquire such an importance that they alone constitute the medium of their internal speech.

The general rule, however, is that auditory images constitute the most potent representations of words (even in the common class of indifferents), whilst visual images form the most potent representations of ordinary external objects.

In exceptional cases it seems that the ordinary functional coupling of the auditory with the glosso-kinaesthetic centre, and of the visual with the cheiro-kinaesthetic centre, is not adhered to. Thus the deaf-mute has to rely upon signs and gestures, or lip-language, and he thinks in the main with revived visual symbols; and it is from the organic seats of these that incitations pass to related parts of the glosso-kinaesthetic centre.

Again, in children who have been born blind, but have nevertheless ultimately learned to write, a direct association must have become established between the auditory and cheiro-kinaesthetic word centres. In the simplest process of thought we have no limitation of activity to any one area, but there are widespread processes of primary activity in all these speech centres and their associated tracts in both hemispheres—an activity which is not limited to concept or percept centres, but spreads to all parts of the cortex of both hemispheres. It is generally accepted that lesions of the glosso-kinaesthetic and cheiro-kinaesthetic centres (even when both are affected) do not produce failure to comprehend the meaning of words written or spoken, although the patients themselves are unable to speak or write (Bastian loc. cit.).

The degree of derangement from such lesions varies in different individuals, as one would expect, from relative potency inherited or acquired of the various centres. A more important and probable explanation is the variability of vascular supply and possibility of collateral circulation being established, and still more the escape or involvement of perforating arteries supplying subcortical structures.

The right facial paresis suggests the possibility of a subcortical lesion and the possibility of a small lesion in the anterior portion of the posterior half of the capsule, and although sections through the brain in the region of Broca's convolution and the zone of the lenticular nucleus showed no lesion, yet it is open to Marie to argue on this account that the aphasia was explicable by a condition of anarthria. I would submit, however, that the few times she uttered "is that" her utterance was normal. This suggests a lack of sensory initiation.

PROFESSOR BUDIN'S SUCCESSOR.—Dr. Paul Bar was appointed on July 4th to succeed the late Professor Budin in the Chair of Obstetrics in the Medical Faculty of Paris. Born in Paris in 1853, he was *externe* of the hospitals in 1875, and gained the coveted post of *interne* in the following year. For a year he was under Tarnier at the Maternité. In 1881 he took his degree, and was awarded a silver medal for his thesis on the pathology of hydramnios. In 1883 he was appointed accoucheur to the hospitals, and in 1887 he won the rank of *professeur agrégé*. He took an active part in the foundation of the Société d'Obstétrique, of which he is now President. Professor Bar collaborated with Tarnier in his treatise on midwifery, and has written much on eclampsia and other obstetrical subjects. He has already established his reputation as a teacher.

THE PREVENTION OF VALVULAR DISEASE.

By RICHARD CATON, M.D., F.R.C.P., J.P.,

CONSULTING PHYSICIAN, LIVERPOOL ROYAL INFIRMARY.

As medicine becomes more scientific in methods of inquiry and less vague in theories of treatment, it naturally follows that definite and practical measures, such as the arrest of a pathological process in its early stage, are recognized as of great value. Much more may be done for the welfare of the patient by checking a disease of structure in its incipient stage than by any curative measures when the morbid change has become fully developed.

Though we have yet something to learn in regard to the diagnosis of valvular disease, the clinician can in the majority of cases not only detect a fully-developed regurgitation or stenosis, but he can also follow the advancing stages of such a valvular defect in the making. I fully admit that experience and close observation are needed for a diagnosis of the latter order, but it is quite within the powers of any well-trained physician.

In the case of the development of that too common ailment mitral regurgitation, the normal first sound at the apex is compounded of two elements, first of the less important muscle-sound, and secondly of the "crack" of the stretched valve cusps made "taut" by the sudden high pressure of the blood stream under the powerful squeeze of the ventricle. This "crack" is much like the sound produced by a sail which has been hanging slack when the wind suddenly fills and tightens it; this second factor, due to sudden valve tension, is much the more important of the twin causes which in conjunction make up the first of the two sounds, the "lubb-dupp" at the apex, with which we are all so familiar. Those of us who have watched with care the progressive changes of sound which lead up to the bruit of valvular disease know that the sharp clear "lubb" is gradually changed into a longer and duller sound, often somewhat muffled in quality. This change is due to a thickening and stiffening of the valve membrane from inflammatory change with effusion under the endothelia, and later to the formation of vegetations on the free surface of the membrane. Necessarily the cusp becomes less pliable and adapts itself less quickly to the movements of the blood-wave. In like manner, if we were to thicken and stiffen the mainsail of a yacht so as to render it less pliable, the sharp clear tone at the instant when a sudden wind-wave rendered it "taut" would not be produced. Then as we watch such a valve we note that its sound becomes more prolonged and assumes what may be termed a "whiffing" character, a sort of tuneless impure whistle, due I imagine to less perfect closure of valve cusps and partial escape of fluid between them.

Next this whiffing sound develops into a genuine regurgitant bruit as the amount of reflux increases. The thrombi and projecting vegetations which form on the edge of the cusp almost inevitably prevent exact closure, and the intraventricular pressure being great the reflux soon raises tension in the auricle and the pulmonary network, and causes augmented pressure in the pulmonary artery and sharper closure of the pulmonary valve.

Long years of experience in hospital and private practice have taught me the vast importance of watching these early stages and directing one's scheme of treatment in accordance with the indications they give.

In acute rheumatism the inflammatory changes which take place (a) in the joint tissues and (b) in the endocardium are like in kind. But the two structures are differently situated as regards functional activity. The joint tissues have secured for them the great advantage of physiological rest—a condition of the greatest aid to natural and artificial means of cure. The sensory nerves, whose function under normal conditions is dormant, wake up during the presence of inflammation to a phase of acute irritability, and absolutely forbid all movement. As it seems to be a law that functional activity and the repair of lesion cannot proceed in any living structure at the same time, the pain thus caused by movement has a great therapeutic function in the rheumatic joint in thus ensuring rest.

The heart is very differently circumstanced. For its complete rest is not possible, and doubtless the necessary