for. Sometimes he barked like a dog, which was said to be merely for the purpose of making us laugh.

At a place near Korusco I saw an imbecile with a curiously distorted head. Almost all the imbeciles I have seen have been ill-developed generally, with small heads, narrow chests and weak legs.

CLINICAL NOTES AND CASES.

Two Cases of Temporary Aphasia from Shock after a Series of Severe Epileptic Fits. By FLETCHER BEACH, M.B., M.R.C.P., Medical Superintendent of the Darenth (late Clapton) Asylum.

(Read before the Medico-Psychological Association, November, 1878.*)*

It will be impossible for me to discuss this evening the different theories that have been brought forward to account for the production of aphasia (including under this term all forms of affections of speech), and I limit myself therefore to a few remarks upon one of them.

It is held by some authors in this country, and more especially by Drs. Bastian and Broadbent, that there is a special "perceptive centre" in relation with each sense, and that these "perceptive centres" are situated in convolutions which receive radiating fibres. Ferrier's researches have located the centres for vision, hearing, smell, taste, and touch in some of the convolutions into which these fibres have been traced, more particularly fibres from the extraventricular portion of the optic thalamus. It has been considered probable that the formation of a complete idea of external objects would be represented structurally by the convergence of commissural fibres from each perceptive centre to some part of the cortex not in direct relation with the basal ganglia. This formation of a complete idea is of course not present in the young infant, but is gradually attained, the perceptive centres also being more highly developed as the child grows up. A part of the intellectual process above mentioned would be the association of a name with the idea, so that ideas may be expressed in language. If a breach were made in the channel of communication between one of the perceptive centres and the "idea centre," or "naming centre," say the "visual

perceptive centre" and the "naming centre," the patient could not name the simplest object at sight, and such a case has really been observed. So if a lesion occurred between the "auditory perceptive centre" and the "naming centre" he would not only fail to understand spoken words, but fail to know what he was saying. A case of this kind has been related by Dr. Broadbent. If again a lesion were to occur between the "naming centre" and the "centre for articulation," which is generally understood to be in the medulla, since in that region are the nuclei of the various nerves supplying the parts employed in articulation, the patient would be unable to express his thoughts in words. If a question were addressed to him, and his "auditory" and "naming centres," and the channel between them were intact, he might be able to name mentally his ideas, but the communication between his "naming centre" and the "centre for articulation" being cut off, he would be unable to communicate them to the outside world.*

Adopting this theory for the present, I will attempt to show how it may be applied to my two cases, the particulars of which I will presently give. In the first of the two the communication between the "auditory" and "idea" centres was intact, since the patient on recovering from her unconscious condition showed by her expression that she understood what was said. In the second this was not at first the case. The communication between the "idea" and "naming centres" and the "centre for articulation" was, I think, temporarily injured. That for articulation certainly was, for although the lips, which are supplied by the facial nerve, moved, no sound could be heard. It may perhaps be said that the lesion was only in the "centre for articulation," but I am inclined to think from my observation of the cases that there was one higher up.

The lesions which ordinarily occur in aphasia are organic, but those to which I now refer being only temporary, are functional in character. "There probably exists in these cases (functional) an altered molecular state in the brain tissue of the affected region, which for a time renders it unfit to discharge its proper functions; although if we could examine the part at the time there might be no change visible to the naked eye, or even by the aid of the microscope."† Many cases of temporary hemiplegia after epilepsy may be

* I do not enter into the question of his ability to *write* his ideas.

† Bastian—"Paralysis from Brain Disease."

placed in this category. "A patient will suffer from unilateral convulsions from time to time, and after one of these attacks more severe than usual he is found to be paralysed on the side which was previously affected with the convulsion. The hemiplegia is of the ordinary kind, except that it is temporary in its duration. It may last only for a few hours, or a few days, and then the patient rapidly gains power in the paralysed limbs."* This we have all of us seen over and over again. Occasionally hemiplegia, too, follows where the convulsive condition has been general, though most marked on the side which subsequently became paralysed. By some the condition has been associated with "spasm of vessels," producing and keeping up anæmia of the brain. We all know the pallor which occurs in many cases before the commencement of an epileptic fit, and I have seen with the ophthalmoscope the arteries of the retina contracting at its commencement. It is difficult, however, to suppose that this spasm of vessels could be continued for many hours. It is more probable that the abnormal discharge of nervous energy causes molecular damage, and a state of nervous exhaustion which is of a recoverable kind; a damage which hinders the proper nutrition of the nerve fibres for a time, and so prevents them from exercising their functions in a proper manner. So no doubt it is in the temporary loss of speech after severe epileptic fits. The abnormal discharge of unstable grey matter which, according to Dr. Hughlings Jackson, is the cause of the convulsions, occasions, when the convulsions are severe and numerous, a condition of shock, of temporary paralysis, not only of the parts concerned in movement of the body, but sometimes also of those concerned in speech. After a time molecular restoration occurs, and the patient slowly recovers.

Case I.—About eight months ago I was called one evening to F. A., a stout, somewhat plethoric girl, aged 15, who was in the "status epilepticus." She had had upwards of 14 fits in a very short time, and when I saw her she was having them about every five minutes. Both sides of the body were affected. It being impossible to give any medicine by the mouth, I at once injected bromide of potassium into the rectum. A fit came on while this was being done, and the injection returned. Four times an injection was returned, but the fifth remained. Two more fits occurred in the night, but no more for some days after. Next morning the girl was unconscious, and remained so for some hours. On the following morning, however, con-

* Opus cit.

sciousness had completely returned, but she was unable to answer questions. Her facial expression showed that she understood what was said, and her lips moved, but no sound escaped. Next day words came slowly in answer to questions.

Case II.—About two months after the occurrence of the preceding case I was called to J. S., aged 15, a stout and well-proportioned boy, who had had 19 severe fits in a comparatively short time. His breathing was affected by their severity. Bromide of potassium in large doses was given in the intervals between the fits, which in the course of the night ceased. The convulsive movements, as in the preceding case, were general. The next morning he was in an unconscious state, but during the day consciousness partially returned. Next day, when spoken to, he only put out his tongue. Gradually, but slowly, speech returned.*

Now in both these cases we are at liberty to suppose that molecular damage of the nervous tissue resulted, so that it could not for a time discharge its functions. This damage was soon repaired, and the parts affected regained their normal condition more slowly in the second, quickly in the first case. The "auditory," "visual," and "idea" centres recovered quickly in the girl, since 24 hours after the fits her facial expression showed that questions addressed to her were understood. There was, however, a breach between the "naming centre" and the "centre for articulation," the latter also being weakened. The patient could not make use of words, and though her lips moved slowly no sound was produced. Finally all the centres regained their previous power. In the second case all the perceptive centres recovered more slowly. The fits were more severe and continued longer, and the exhaustion of nervous tissue was more complete. The "visual" and "idea centres" first recovered, the "auditory centre" being at fault for a longer time. When asked the question "How are you?" he put out his tongue. His "visual" and "idea centres" told him that the movement of my lips was probably due to my asking a question, and as a common request of mine is to ask patients to put out their tongue, there was a probability that that was the question I was asking. Had his "auditory centre" been in a normal condition it would have told him differently. There was also a breach between the "naming centre" and the "centre for articulation," no attempt at speech being at first made. Complete recovery, however, took place. In Dr. Broadbent's case, before referred to, post mortem examination

* I do not now enter into the condition of the limbs, further than to state that no permanent loss of power has resulted in either case.

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showed softening of the brain between what may be supposed to be the "auditory perceptive centre" and the higher one in which the "name centre" is associated with the idea. This patient made use of what was really a jargon, but it was obvious from his gestures that he thought he was giving expression to ideas present in his mind. He did not recognise the fact that his language did not convey these ideas. In my cases, although the lesion was a less serious one, yet during the time of molecular exhaustion and unconsciousness, all the centres were paralysed, and no speech was possible, but as this exhaustion was recovered from, they regained their normal condition.

The theory referred to in this paper, perhaps, requires further proof before it is fully accepted; but as all theories are useful for stringing facts together, I determined to make use of it, in order that I might possibly throw some further light on the causation of temporary aphasia.

Two Cases of Recovery from Insanity, after many years in an Asylum. By GEO. H. SAVAGE, M.D.

The subjoined cases are of most interest from the duration of the symptoms which were finally recovered from.

Such cases open up very difficult questions both on their practical and their scientific sides. To me, now, it seems possible that both cases might have recovered if they had been sent home sooner. But the terrible risk of sending out an intensely suicidal, and an intensely homicidal case, was hardly to be accepted.

Many insane people seem to be specially plastic mentally, and readily suit themselves to their surroundings; they may thus fit themselves only for insane surroundings, and by residence in an asylum be rendered unfit for the outer world; this is to be acknowledged and avoided where possible. In the next place these cases of late recovery make us again consider the relationship between diseased mind and diseased brain. How are we to have a mind depressed and acting feebly and painfully for many years recover completely? Can a diseased process go on in the brain for twenty years, and then be healed?

I, for one, cannot admit the possibility of a functional brain disease apart from organic changes, but as yet we know so little of brain action that there may be many changes that