and Dr. Moutier, is that of an old man aged 64, who died at Bicêtre under their care.

He had been admitted for mitral disease and bronchitis, and there can be no doubt that language was quite unaffected. Moreover, he had always been right-handed. He died on December 11th, 1906; on the 2nd of that month he complained of a little numbness and weakness of the right hand, but there was no hemiplegia nor any Babinski's sign. There had been nothing to attract attention to the brain, which was only being examined in accordance with their rule of making a thorough autopsy of all cases, and this is what they found: In the left hemisphere the foot of the third frontal convolution was very distinctly atrophied in comparison with the foot of the right third frontal. Moreover, a "worm-eaten" patch had destroyed the grey matter of the upper half of the posterior branch of the cap and of the superior third of the foot of the third left frontal.

The published photograph[3] shows better than any description the extent of the lesion, and is almost an exact copy of the lesion shown in the plate published by Tuke and Fraser[2] in the account of their case. It differs from Tuke and Fraser's case in there never having been any disturbance of the faculty of language.

It is difficult to see how it is possible to escape from the amount of evidence brought forward to show that destruction of the third frontal convolution, even on both sides, does not cause aphasia; but almost equally cogent, and even more abundant, is the evidence that aphasia occurs in cases where this convolution is intact. The usual answer in this evidence is that while it may appear intact, it is not so really; but Professor Marie has lately published three cases of this kind in which the convolution was examined microscopically by serial sections without any lesion being discovered. The only rejoinder that the supporter of Broca can make is that the lesion is functional in character, and reveals itself by no changes which can be detected by our present means of research. But those who make this assertion must bear the onus of proof, and it is not easy to produce evidence in its favour. If the convolution of Broca is the seat of the faculty of language its destruction should be invariably followed by loss or impairment of that faculty; further, when there is loss or impairment of the faculty of language this convolution should show always some demonstrable change; if neither of these relations exist, then the only possible verdict must be that the existence of the supposed relation is not proved.

REFERENCES.
[1] BRITISH MEDICAL JOURNAL, 1876, vol. i, p. 433. [2] *Journal of Mental Science*, vol. xviii, p. 46. [3] *L'Aphasie*, p. 397.

TWO CASES OF HEREDITARY CONGENITAL WORD-BLINDNESS.

BY

JAMES HINSHELWOOD, M.A., M.D.,

SURGEON TO GLASGOW EYE INFIRMARY AND LECTURER ON OPHTHALMOLOGY IN THE GLASGOW WESTERN MEDICAL SCHOOL.

AT the Exeter meeting of the British Medical Association in 1907 I read a paper[1] in the Section of Ophthalmology on four cases of congenital word-blindness occurring in the same family. Dr. C. T. Thomas[2] in a paper in the *Ophthalmic Review*, in August, 1905, had already called special attention to the fact that congenital word-blindness may assume a family type and that a hereditary tendency is probable. I recorded the four cases in one family as a confirmation of the correctness of this observation. The two cases I am about to record are a still further confirmation of the hereditary tendency, as they occur in the second generation of the same family which afforded the subjects of my communication to the British Medical Association in 1907.

The four cases previously reported were the youngest members of the family. The two cases in the present paper are the children of the oldest daughter of the same family, and hence are the nephew and niece of the cases previously reported. The mother of these two children never herself experienced any difficulty in learning to read. She has six children and the other four members of the family have learnt to read without any special difficulty.

A., a girl 12 years of age, has been at school for seven years. She has experienced the greatest difficulty in learning to read, and at present can only read very imperfectly, being able to recognize by sight only some of the commonest monosyllabic words. For several years she seemed to make very little progress, but has done much better during the last year. She knows all the letters of the alphabet, but was a very long time in learning them. She can recognize by sight most of the common words of one syllable, such as *the*, *of*, *in*, *are*, *which*, *to*, *with*, and so on, but most of the rarer monosyllabic words she cannot recognize by sight alone, although she recognizes them nearly all at once when spelt out aloud to her. She recognizes by sight scarcely any words of more than one syllable, but many of these she also recognizes, if they are spelt out aloud to her. She has never had the slightest difficulty with arithmetic, and in fact has been rather good at it and above the average. She is at present in the compound rules, and has no difficulty in keeping up with the rest of the class in this subject. She has no difficulty in committing things to memory, and seems a sharp, intelligent girl in every other respect.

D. is a boy aged 10 years, and has been at school for four years. He has made very little progress in learning to read by sight. He does not know by sight all the letters of the alphabet, although he can repeat them correctly in sequence. He can scarcely recognize any words by sight. Out of a whole page of a child's primer he recognized by sight only two words, *to* and *go*. When I spelt the words out aloud to him and appealed to his auditory memory he recognized every word on the page. He can spell nearly all the small words, and can recognize them when they are spelt aloud to him, but he cannot recognize them by the sense of sight alone. Whilst the visual memory for words is so defective the auditory memory seems good. He has therefore experienced no difficulty in committing things to memory which have been read aloud to him, such as pieces of poetry. With regard to arithmetic he has had the same difficulty as with regard to reading. He can repeat the figures in sequence up to "100," and can write them also. But if the figures are written down separately he can only recognize them with certainty up to "20," and very few combinations after that.

In other respects than learning to read and count, he seems quite a smart intelligent boy.

The first case, that of the girl, does not call for any very special remark. It is a typical case of abnormal difficulty in registering in the brain the visual symbols of letters and words. The degree of difficulty in this case, however, has not been nearly so great as is met with in many cases. The difficulty, of course, varies greatly in degree, some cases being comparatively mild, and others where the difficulty is so great as to seem at first insuperable. The girl's case is a mild case, because she has now, after seven years of teaching in the ordinary way, acquired the visual memories of the most common monosyllabic words. I have seen and recorded cases where, after seven years of ordinary training, the child had practically acquired nothing so far as the visual memory of words and letters is concerned. The case of the girl may be regarded as a mild case of congenital word-blindness, and if now taught in the proper way, I am certain will soon be able to read quite well.

The second case, that of the boy, is of a much more severe type. Here the difficulty has been much greater than in the case of the girl, as, after four years' similar training, he has acquired scarcely any visual memories of words or letters, not even recognizing by sight all the letters of the alphabet. An important point in his case is that he has experienced the same difficulty in dealing with figures. This is in contrast to his sister, and also in contrast to the four cases which I reported in 1907, none of whom experienced any difficulty with arithmetic and some of whom were above the average at it. When this difference exists, it is helpful in the diagnosis.

In my book[3] on letter, word, and mind blindness I have shown from the study of cases of acquired word-blindness that the visual memory for words and letters is completely independent of that for figures, and hence in congenital word-blindness it is not surprising that whilst the visual memory for words may be very defective, that for numbers may be perfectly normal.

In my last paper[1] I remarked, in discussing this subject, that too much importance in the diagnosis should not be assigned to the relative difference in acquiring the visual memories of words and figures, as cases had been reported where the difficulty extended to both figures and words. The case now under consideration affords confirmation of this observation. It is a typical case of congenital word-blindness, where the difficulty also extends in equal degree to figures. Nor should the occasional occurrence of this

combination surprise us, since we know that the cerebral areas utilized for the storage of the visual memories of words and figures, though distinct, are close together, and hence may be sometimes simultaneously involved, as in the present case.

In the discussion that followed the reading of my paper on hereditary congenital word-blindness at the Exeter meeting in 1907, Mr. Sydney Stephenson narrated a striking example of six cases of congenital word-blindness affecting three generations of one family, which he subsequently published.[4] Mr. T. Herbert Fisher[5] also published a case of congenital word-blindness, and in his paper called special attention to the fact that the uncle of the child had also experienced the greatest difficulty in learning to read. The evidence is thus gradually increasing as to the hereditary tendency in congenital word-blindness. I have little doubt that in future, if observers in every case would make careful inquiries into the family history both of the present and previous generations, the evidence would rapidly increase as to the frequency of this hereditary tendency.

The fact that congenital word-blindness is sometimes hereditary, as is typically exemplified by these six cases occurring in two generations of the same family, is of some importance from the etiological standpoint. In my paper on congenital word-blindness at the Oxford meeting of the British Medical Association in 1904,[6] I said that in these children their difficulty in learning to read can most readily be explained on the ground of some defect in the special area of the brain, generally the left angular gyrus, where are stored the visual memories of words and letters, and that if there be any abnormality within this area; due either to disease, to injury at birth, or to faulty development, it was easily conceivable how such children should experience abnormal difficulty in learning to read. In these six cases belonging to the same family and other hereditary groups it is evident that the abnormal condition of the visual memory centre is a matter of faulty development, and it is probable that in most cases of congenital word-blindness the condition is the result neither of disease nor injury at birth, but of defective development of this definite cerebral area occurring in the early stages of embryonic growth. This view, that congenital word-blindness is the result of faulty development and not of disease or injury, derives considerable further support from the fact that homonymous hemianopsia, which is so frequently associated with acquired word-blindness, has never been met with in any case of congenital word-blindness. In a paper of mine on word-blindness with right homonymous hemianopsia[6] I discuss the reason for the frequency of this combination, and have in my own experience met with five cases of acquired word-blindness associated with right homonymous hemianopsia. The fact that this association, so frequently observed in acquired word-blindness due to cerebral disease, has never yet been met with in the congenital form is important and significant.

With regard to the future of these two cases of congenital word-blindness, I have no doubt that both children can be taught to read. In the case of the girl, she has already, although very slowly, made definite progress, and this will be much accelerated if she is taught in the proper way. Even in the case of the boy, where little progress has been made after being four years at school, I am certain he can be taught to read if the task be undertaken in the proper way and with sufficient persistence and perseverance. For my views on the proper method of teaching these cases I would refer to my two previous papers on this subject in the BRITISH MEDICAL JOURNAL.[1 6]

Of the many cases of congenital word-blindness which I have seen all have ultimately been taught to read. But it is to be observed that by congenital word-blindness I mean pure cases, where this defect exists alone in an otherwise healthy and normal brain. Cases of inability to learn to read associated with general defective intelligence and defect of other cerebral centres belong to quite another category, and in such cases the prognosis is not so hopeful.

Pure congenital word-blindness, which is a local affection of a limited cerebral area, can always be overcome by perseverance and proper methods of education, and herein lies the great importance of the recognition at an early period of the true character of such cases.

REFERENCES.
[1] Hinshelwood, BRITISH MEDICAL JOURNAL, November 2nd, 1907. [2] Thomas, *Ophthalmic Review*, August, 1907. [3] Hinshelwood, *Letter, Word, and Mind Blindness*, London, H. K. Lewis, 1900. [4] Sydney Stephenson, *Ophthalmoscope*, September, 1907. [5] Fisher, *Ophthalmic Review*, November, 1905. [6] Hinshelwood, BRITISH MEDICAL JOURNAL, November 12th, 1904.

AN EPIDEMIC OF INFANTILE PARALYSIS IN BRISTOL.*

BY GEORGE PARKER, M.D.CAMB.,
PHYSICIAN, BRISTOL GENERAL HOSPITAL.

THE spread of infantile paralysis in an epidemic form during the last few years over a great part of Europe and America has been so extraordinary, and the outlook for the future is so serious, that I need hardly apologize for bringing before you the facts of an outbreak in Bristol from June, 1909, to January, 1910. It is one of the first which have occurred in England. W. W. Treves reported a group of 8 cases in 1908, and in 1910 some 13 were recorded at Maryport. The cases which I have collected by the kind help of friends and colleagues number 37. I have seen nearly all of them myself, and examined their present condition. If the numbers are small compared with those of some foreign epidemics, they are, I think, quite enough to show that we have more than the sporadic form of the disease to deal with. Happily we have not yet reached the condition of the United States, where Flexner estimates that 20,000 cases occurred during the last summer.

I could find hardly any cases here in the first months of 1909. In June 5 appeared, and 27 more by the end of October, leaving 5 only during the next three winter months. The usual time is during the three summer months, and it is curious that our attacks should go on past mid-winter. The summer, too, had been cold and wet with us.

The age of the patients varied from 4 months to 16½ years, giving an average age of 3 years, or if we omit three adolescents the average age of the rest was almost exactly 2 years. There were 24 males and 13 females.

Gowers has remarked that in the epidemic, as distinct from the sporadic, type there are a considerable number of complete recoveries without paralysis, that the mortality is considerable, perhaps 10 or 12 per cent., because fatal bulbar and cerebral attacks are recognized as due to this disease, and that adults suffer as well as children. As our statistics were collected after the outbreak, and as public attention was not directed to poliomyelitis at the time, it is probable that most of these abnormal cases were overlooked. I have not included in my list any cases where paralysis was absent throughout. On the other hand, I cannot point to any absolutely complete recovery, though three or four are practically well. The great majority are more or less crippled for life. Two patients died, a mortality of nearly 6 per cent.

Most of the children had a febrile attack before or immediately after the onset of paralysis. In about a quarter of them pains of a rheumatic character are recorded. In one child the knee was kept flexed for some days as if from joint pain, a symptom which I have seen before. Some had pain down the spine, and in two retraction of the head was noticed, which might lead to the diagnosis of meningitis. Facial paralysis occurred in No. 21. In view of Flexner's suggestion that the entrance of the infection is often through the naso-pharyngeal membrane, we may remark that tonsillitis appeared in two cases, Nos. 36 and 37, and pulmonary troubles in several, such as Nos. 8, 20, and 25, whilst in three there were symptoms of measles—Nos. 15, 24, 36. How far the latter were real attacks of measles, which predisposed the patient to the infection of poliomyelitis, or merely symptoms of the latter disease, it is hard to say.

On the other hand, there were half a dozen children who were suddenly paralysed while apparently in perfect health—for example, 7, 11, 14, 21, 22, 31, 32. In others the history of a previous fall or blow was recorded. The importance of this is problematical. In one, the details of

*Read before the Bristol Medico-Chirurgical Society, January 11th, 1911.