CLINICAL DEMONSTRATION AT THE MAUDSLEY HOSPITAL.

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AMONGST the cases for examination are:—
A man showing the retinal signs of arteriosclerosis.
A young woman whose eyes have suffered rather severely as a consequence of congenital syphilis.
A woman, aged 61, who for about 12 years at least has been the subject of diabetes.
A woman of about 50 with advanced secondary optic atrophy.

Before looking at these cases may we say a few words about them

With regard to the man who is the subject of arteriosclerosis; if we speak to him we find that his brain—like his eyes—is deteriorating, owing to a defective blood-supply. His intellect is slow and uncertain, his memory is bad, his speech is halting and indistinct; if now we examine his retinal vessels, which, being ultimate branches of the internal carotid, are a perfectly fair sample of the other branches of the internal carotid in his brain, we shall, I think, see the reason for his mental deterioration, remembering that the retinal vessels are the only vessels in the whole body which are clearly visible during life, and that—as seen by direct ophthalmoscopy—they are magnified about 15 diameters. It will be seen that in either eye the retinal arteries, instead of being of uniform calibre, are irregular, narrowing down in places, and again becoming of full diameter further along—a very characteristic sign of arteriosclerosis.

If we look along the upper temporal vessels of the left eye we shall see that one of the branches of the artery crosses one of the retinal veins; and here the vein, instead of continuing its course, undergoes a curious sort of sigmoid curve under the artery. This is seen in several other crossings in the eye and is pathognomonic of the disease. The arteries too are of the "copper wire" type—a sign, however, which is no more than an exaggeration of normal appearances and so is less valuable than the two former. In each eye are a few hemorrhages which are situated in the nerve fibre layer of the retina and show up the texture of this layer; they consequently have somewhat the appearance of tongues of flame, and so are spoken of as "flame-shaped hemorrhages."

The next patient is a girl 22 years old. It will be seen that both corneas are somewhat diffusely hazy, and on more careful examination it will appear that this is due to a faint nebulous condition of them and that the tracks of old blood-vessels are present in their substance. If these blood-vessels are traced towards the sclerotic they will, for the most part, be found to disappear from view because here they enter opaque tissue. In contrast with this, the blood-vessels that are formed in connexion with superficial lesions of the cornea, when traced outwards, can be followed over the sclerotic into the conjunctival vessels. Both eyes are affected, as it is invariably the case in the interstitial keratitis of congenital syphilis. In each fundus, scattered around its periphery, are very numerous more or less circular whitish areas, with pigment interlaced over them. These are scars of choroiditis, which was very likely an active disease at the same time that the interstitial keratitis was active. There are a good many fundus lesions, of which the cause is by no means certain, but when one sees the appearances which are present here one can be pretty sure that they are due to congenital syphilis. This patient has not the characteristic facies; she has, however, Hutchinson's teeth, and her hearing is faulty from an involvement of the internal ear.

The next patient is a woman, 60 years old, who for 12 years at least has been the subject of diabetes, and during the last few years her sight has deteriorated. It will be seen that she has some opacities in her lenses; but a woman of 60, whether diabetic or not, is almost certain to have some lenticular opacities. It is a misnomer to speak of these as diabetic cataracts. The proper diabetic cataracts are those which occur in young subjects who, but for the disease, would certainly not have developed cataract. The youngest I have seen them occur in is a young girl of 16, but no doubt they may develop earlier than this. They usually start in the posterior part of the lens and progress rapidly, so that within a few months the whole of each lens is opaque. On examining the patient's retina we find scattered over the central region a number of hemorrhages which for the most part are small and have a blotched sort of appearance. This type of hemorrhage is rather characteristic of diabetes. It is unusual to find flame-shaped hemorrhages. We also see a number of opaque white spots and areas in the central region distributed roughly around the yellow spot. One or two of them have a soapy or waxy appearance, they have hard edges, and there is no evidence of cedema around them. There is no star figure and there are no cotton-wool patches.

It is sometimes said that there is no such thing as diabetic retinitis, that it is no more than renal or arteriosclerotic retinitis occurring in a patient who also has diabetes, and at first sight there are certain points which seem to support this view, for it is unusual to find retinitis in a diabetic without the blood pressure being raised and without albumen in the urine. There are, however, a good many points which make it clear that there is a form of retinitis which is dependent upon diabetes, for whereas in the first place renal retinitis may occur at almost any age (I have twice seen it in a child of 7) I have never seen diabetic retinitis in a patient younger than 30. It is true that as a rule there is albuminuria and the blood pressure is raised, but I have on several occasions seen retinitis in a diabetic in whom albumen was found to be persistently absent in the urine and the blood pressure

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was not raised. Further, a patient with renal retinitis seldom lives as long as two years; a patient with diabetic retinitis may quite well live for ten years or longer. And lastly, in many cases one can identify diabetic retinitis as distinct from renal retinitis from the ophthalmoscopic appearances, the chief points being the blotch hemorrhages, the solid soapy or waxy appearance of the exudate, the absence of a star figure, and the absence of cotton-wool patches.

The fourth patient whom we will consider is a woman of 50 with advanced secondary optic atrophy. By secondary optic atrophy is meant an atrophy which is the result of an antecedent disease in the optic nerve or retina. The most important example is that which follows papilloedema. By primary optic atrophy is meant the degenerative processes which occur, for example, in tabes dorsalis. It is important in some cases to be able to say that this is a secondary or primary atrophy, and it is often possible to do so with assurance. In a good many cases, however, it is not possible from the ophthalmoscopic appearance alone to say which form of atrophy we are concerned with. We may take the following as the chief points of distinction between the two:

In primary atrophy the disc edges are sharp, the disc itself is greyish in colour, it shows a shelving sort of cupping, the lamina cribrosa is exposed, and the vessels are not reduced in size. As syphilis is the commonest cause of this there may incidentally be signs of syphilitic disease of the retina or choroid. In secondary optic atrophy the disc edges are blurred, the disc is an opaque white colour, the physiological cup tends to be filled in, the vessels are reduced in size, there may be a white sheathing along them, more especially along the veins, and there may be evidence of change in the surrounding parts of the retina, but nevertheless a good many cases of optic atrophy are observed where it is not possible to tell from ophthalmoscopic examination alone whether it is a primary or a secondary change; and let me say a word or two about the papilloedema which precedes the atrophy.

In papilloedema the disc edges are blurred, the disc is swollen, and retinal hemorrhages and exudates may be present, but the degree of sharpness or otherwise of the disc edges varies greatly under physiological conditions, and in the early stages of papilloedema it may quite well be impossible to say whether it is indeed early papilloedema or whether the blurring of the disc edges which is present is no more than a physiological variation; and if we are asked to report upon the condition of the optic discs in a patient whom we are told has headache and vomiting and in whom the disc edges are blurred it is particularly important to maintain a critical mind. It is obvious that if we allow a report upon the optic discs to be tinctured by the information that is given that the patient has headache and vomiting it ceases to be of any value. Unless there are quite undeniable evidences of pathological change such as hemorrhages or exudates or measurable swelling, the only possible correct attitude to take up is that we are unable to say whether early papilloedema is present or not, and that we would like to see the patient again in the course of a few days. I have known observers misled to the extent of saying that 6D of swelling of a disc was present because the disc edges were blurred, and it seemed quite certain that the patient had a cerebral tumour, whereas in fact—as was positively proved by the subsequent course—there was no cerebral tumour, and no pathological change in the disc was present.

With regard to the estimation of swelling of the disc, if particular precautions are taken and the patient is thoroughly amenable, it is possible to estimate swelling of the disc certainly within 1D, but I have known observers profess to estimate swelling of the disc, and to let important decisions rest upon the result, when they were indeed doing no more than estimate the patient’s astigmatism. There also is a tendency amongst such observers to act on the principle of “When in doubt say 3D.” I have not often known the estimation of the swelling of the disc prove of any real value.

Correspondence

POST-GRADUATE TEACHING AND THE NEVILLE CHAMBERLAIN COMMITTEE.

To the Editor of the Post-Graduate Medical Journal,

SIR,—Though it has been an open secret for some time the announcement by the Minister of Health of the decision of his Committee on the future of the post-graduate movement must give rise to many serious speculations. I notice that from first to last no mention is made of the Fellowship of Medicine, which has done so much for post-graduate teaching in London, which is known throughout the world, and which has so successfully coördinated and combined the 50 or so London hospitals which were willing and able to welcome qualified men to their classes. I trust some means will be found to combine the Fellowship with any new body that is formed.

I feel sure everyone will regret that the accepted principle of a new hospital erected for the purpose has had to be abandoned, and that the Minister has also abandoned the idea of a central hospital which the Athlone Committee considered essential.

It is proposed to enlarge the West London Hospital by 100 per cent. Even if the Government will advance the money for this enlargement, will they also maintain the hospital, which certainly cannot live on its school? It may be said that this is a matter for the hospital authorities, but it is also the concern of all those interested in the post-graduate movement, for if the beds cannot be kept open the scheme falls to the ground.

How will the scheme affect the other post-graduate college in London? I refer to the North-East London Post-Graduate College connected with the Prince of Wales’s General Hospital. This Hospital has borne the burden and heat of the day for 20 years, and it is not too much to say that had it not been for the self-denying work of this College post-graduate teaching in London would have done much less than it has. Is it to be the fate of this College that it must try to carry on with the whole weight of a state-aided organisation against it? There is no suggestion in the Minister’s statement that its position will be assured. I do hope that in any discussion about post-graduate education the Fellowship will be consulted, both about principle and details.—I am, Sir, yours faithfully,

H. W. CARSON.

Clinical Demonstration at the Maudsley Hospital

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