A Lecture

ON

THE DIAGNOSIS OF GLAUCOMA.

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BY

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THE teaching of post-graduates differs from that of students in one very important particular—namely, that of the angle from which we approach the subject. The teaching of the student is synthetic. An idea is presented to his mind under the cover of the name of a disease—for example, glaucoma. Round that idea we build up a number of items of knowledge having reference to the symptoms, the diagnosis, the pathology, the prognosis, the treatment, etc., of the disease we are studying. Such a line of study is made easy for the student by the fact that when he enters the wards he finds the cases allotted to him already marked with the diagnoses which have been made by his teachers.

The post-graduate practitioner is in a very different position. His patient comes to him with a complaint of one or more signs or symptoms, any or all of which may be, and indeed usually are, common to many diseases. Starting from one or more of the clues thus offered, the medical man has to work his laborious way in a search for the cause of the trouble. Very often he has to do so through a tangle which time and study alone will enable him to unravel.

One of the tragedies of ophthalmic practice lies in the frequency with which we meet men and women blinded by glaucoma, simply owing to the failure of the medical men in charge to make an early diagnosis of the condition and to take unflinching action thereon. It is not that there is any real difficulty in recognizing what is wrong, but rather that the possibility of the existence of the disease is too often forgotten, with the result that the symptoms are ascribed to some more commonly met with condition. It is therefore from this angle that we shall approach our subject to-day. First, we shall take the outstanding signs of glaucoma which first impress themselves on the attention of the patient and of his doctor; then we shall show what steps the practitioner can and should take in order either to make a firm diagnosis of glaucoma, or to exclude the presence of that disease. On another occasion we shall study the means at the disposal of the expert for the assistance of the practitioner who consults him.

HEADACHE.

This may be of various kinds.

(A) *A Passing Morning Headache.*

The patient complains that he awakes in the morning with a dull headache, which passes away after he has been up and about for a while; sometimes he will volunteer the information that it is gone by the time he has finished shaving, or that he has forgotten it after reading the paper on his way down to office. On questioning him he will admit that the sight of that eye is dim whilst the headache is on. Rarely he will have noticed dilatation of the pupil and still more rarely haloes round lights. I am far from wishing to suggest that all morning headaches, or even the majority of those headaches which undoubtedly emanate from eye trouble, are due to glaucoma; for, strange as it may seem, headaches due to an error in refraction are sometimes most complained of in the morning, though, as a rule, they are worse at night and relieved by sleep. We have also to take account of the large numbers of other causes of morning headache which are in no way connected with the eyes.

The points of diagnosis have already been indicated. The patient should be warned to look out for the association of headache, misty vision, dilatation of the pupil, slight circumcorneal congestion and haloes round lights; and an ophthalmoscopic examination should be made to ascertain whether there is cupping of the disc, or abnormal pulsation of the retinal vessels.

The explanation of this early morning glaucoma is of interest. Throughout life the clear fluid which fills the chambers of the eye (both the aqueous and vitreous

chambers) is being continually derived from the blood circulating through the ciliary body. With equal regularity it is steadily oozing away at the angle of the chamber through the pectinate ligament into the canal of Schlemm, and thence into the vessels which lead the blood out from the eye. If the rates of inflow and outflow correspond, the amount of fluid retained within the eye remains constant, and the intraocular pressure continues at the same level; but if there is any obstruction to the outflow of the fluid, whilst the rate of inflow remains the same, the tunic of the eye will become overstretched by this increase in the mass of the fluid it contains, and the intraocular pressure will rise correspondingly. If I may be permitted to use a simile: We take a rubber globe like a football, and to it we attach two pipes; one of these leads fluid into it from a tap, which is kept constantly running at an even rate, the other allows fluid to escape at exactly the same rate. The pressure of the fluid within the ball remains the same, and the tension of the coat of that ball does not alter. If now, we slightly pinch the exit tube, more fluid accumulates within the globe than before; the walls are distended in consequence; the pressure of the fluid within the ball rises; and the coat, as felt from outside, becomes harder. If we increase the rate of outflow, the previous condition of affairs is restored.

Professor Arthur Thomson has shown strong reason to believe that during our waking hours there is constantly at work a mechanism whereby the intraocular fluid is pumped out of the eye. The motive force of this pump is supplied by the movements of the iris, and by the contraction of the ciliary muscle. Every time that the pupil contracts and expands under varying conditions of light, and every time that we bring our ciliary muscle into play, as we adjust the eye for sight at varying distances, fluid is being sucked from within the globe into the canal of Schlemm and is again being driven out of the canal of Schlemm into the efferent vessels. During the hours of sleep this pump action ceases, but a normal eye is capable of providing sufficient outflow even without the aid of this mechanism. It is when the excretory channels are, for one reason or another, becoming less efficient, that danger arises. The cessation of the pump action during the hours of sleep may then determine the onset of a condition in which excretion is insufficient for the needs of the eye. A slight addition to the fluid accumulated within the globe will then give rise to moderate attacks of increased tension during the hours of sleep. The consequence is that the patient awakes with a slight attack of glaucoma, as evinced by the headache, the dimness of vision, etc. When he gets up, and begins to use his eyes, the pump action is restored, excretion is hastened, and the symptoms pass away for the time, only to return with increasing frequency and severity as time goes on. Here we have (1) a ready explanation of these morning headaches, and (2) an obvious direction as to the timing of our miotic drops when we use them.

Diagnosis.

It is only when the headaches are accompanied by the other signs and symptoms of glaucoma that we are justified in making a diagnosis of that disease. In any and every case of the kind one of the first things to do is to have the patient's refraction carefully estimated and any errors corrected. It must never be forgotten that an error in refraction may be a cause of congestion of the eye, and so may be a predisposing factor in the onset of glaucoma.

You will ask me why it is that medical men consider it an axiom that sleep is good for glaucomatous patients, and how I reconcile this with the statements I have just made. The answer is simple. Glaucomatous attacks usually depend on the onset of a state of congestion of the eye; sleep quiets the whole nervous system, inclusive of its vasomotor mechanism, and so gives the eye a chance of recovering its normal condition of vascular equilibrium. On the other hand, if the channels of excretion are gradually becoming too constricted for their work, and if, in consequence, instead of being free enough to allow of the ready escape of the necessary amount of intraocular fluid they are so far constricted that they need the assistance of the pump action above outlined, the patient has reached a condition in which sleep brings a constant and steadily increasing danger. I say steadily increasing because (1) it increases more and more as the constriction

of the channels becomes aggravated with the advance of life, and (2) in any one night it increases in proportion to the length of sleep the patient indulges in. The order of events is here as follows: Cessation of the pump action; overfilling of the eye with fluid; consequent obstacle to the outflow of blood through the overstretched coat of the globe; and thus increase of intraocular pressure produced in a double manner and with the establishment of a vicious circle.

(B) *Headaches after Near Work.*

As a man advances in life small errors in refraction, which have not troubled him hitherto, give rise to symptoms, prominent among which are headache, pains in the eyes, a feeling of ocular fatigue, and increased lacrymation. The onset of presbyopia, too, takes its toll of his energy. If his refraction is carefully corrected, and suitable glasses are prescribed, and if any error in muscle balance is likewise attended to, the symptoms should pass away completely, leaving the patient once again forgetful that he even possesses eyes. If, however, the symptoms do not completely subside, and especially if the presbyopia increases more rapidly than we should normally expect it to do, we must look further afield for the cause of the trouble, and one of the many possibilities we should bear in mind should always be the occurrence of high tension in the eye. Such a suspicion should lead us to examine the fundi with great care. The presence of a glaucomatous cup would at once put us on our guard, but, even apart from this, an increase in the pulsation of the retinal vessels, especially if arterial in character, would have a marked significance for the careful observer. Increased pulsation—especially when arterial—is probably invariably the earliest sign of glaucoma, though it cannot always be elicited, owing to the fact that the increase in tension is often intermittent in character. The moral of all this is: Be suspicious of headaches in elderly patients when these persist in spite of careful correction in errors of refraction and muscle-balance.

(C) *Severe Headaches often attended by Vomiting.*

These are often taken to be due to some form of gastrointestinal disturbance, and are then spoken of as "bilious headaches." At other times they are caused by a tumour of the brain, or by some other form of cerebral disturbance. There are many other possibilities, into which we have no time to enter. The one lesson I desire to drive home to-day is that whenever you see an elderly patient suffering from severe headache, whether accompanied by vomiting or otherwise, you should at once rank glaucoma as one of the possibilities to be considered and to be excluded if possible. Other causes of severe headache come and go, leaving the patient little the worse, except for the memory of his experience. It is far otherwise with glaucoma. The headache and the sickness are mere passing events in comparison with the appalling possibilities that loom before your patient if you leave one of these severe bouts of increased tension untreated.

You must obtain a good view of your patient's eyes and, if necessary, you must instil a 4 per cent. solution of cocaine to enable him to face the light for the purpose.

If the eye is congested, and especially if the circum-corneal zone stands out pink, as contrasted with the rest of the eye, two ideas should flash into your mind, namely, acute iritis and glaucoma. Is the pupil widely and evenly dilated? If so, the probability is that you are dealing with a case of glaucoma. But remember the possibility that a mydriatic may have been used very early for iritis. Search the anterior capsule for evidences of adherent iris pigment; and look also for any characteristic irregularity in the outline of the pupil.

Take the tension of the eye with your hand, especially in comparison with its fellow. The acute glaucomatous eye is stony hard. There are many cases of glaucoma in which the rise in tension is so small that two or more medical men may well argue whether it is present or not. No such doubt exists in these cases. The student can recognize the hard eyeball without fail.

Then look at the cornea! In the glaucomatous eye it is steamy; most unmistakably so, indeed. Its polished lustre is gone; the beautiful mirror of the eye looks as if it had been breathed upon; the details of the iris pattern are lost. At the same time we observe that the anterior chamber is abnormally shallow.

In less severe cases, where the media are still transparent, an ophthalmoscopic examination will probably show cupping of the disc. It will not necessarily do so, for, though rarely, glaucoma may make its first appearance as an acute attack, and then the disc has not had time to be depressed, but arterial pulsation will be well seen if the media are clear enough to permit examination.

It is not that there is the least difficulty in making a diagnosis. Certainly not. The trouble is that the busy practitioner may for a moment allow himself to forget that whenever he meets with severe headache of this type, glaucoma is one of the possibilities that should at once flash through his mind. Forewarned will be forearmed.

Just a word or two more on the differential diagnosis between glaucoma and acute iritis. In the latter the pupil, unless it has been artificially dilated, tends to be severely contracted; at an early stage it fills up with plastic exudate. If a mydriatic has been used evidences of irregularity of outline of the pupil, or at least of pigmentary deposit on the anterior capsule, will rarely be lacking. The cornea will be clear, while the pattern of the iris will be muddy; engorged vessels will be seen to stand out on the surface of the iris; the anterior chamber will be of normal depth and not shallow, as it is in acute glaucoma.

There remain the cases in which glaucoma is secondary to iridocyclitis. The wise practitioner will hasten to share the responsibility of such with his specialist colleague at the earliest possible moment.

Loss of Sight.

This, like the headaches, may vary greatly in different patients. We shall consider the typical varieties in turn.

(A) *Gradual Loss of Sight without any Apparent Cause.*

Let me pause here to explain that when a patient comes to you complaining that his "sight is not so good as it used to be," you should not be content merely to set a Snellen type up before him and to ask him to read it down to the 6 m. line. Many a surgeon, under these conditions, will endeavour to reassure his patient, and will then send him away. Those who do so are taking grave risks, for the patient may be right and the surgeon may be wrong, despite the test that has been applied. Let me elaborate the subject. Sight does not merely consist of central visual acuity as measured by a standard card. On the contrary, there are a number of elements to be considered:

(i) *The rapid increase in presbyopia* met with in glaucoma we have already referred to; this is doubtless due to the numbing pressure exerted on the third nerve endings in the ciliary muscle, which gradually robs that structure of its contractile power.

(ii) *The visual field may gradually shrink in area*, thus robbing the patient of one of the safeguards of his daily life, whenever he is on the move amongst other people or even amongst inanimate objects. He does not realize what his trouble is, so naturally he cannot explain it to you, but he is conscious that he is more apt to run into people or things than he used to be. Using even a rough test you may learn that one or both of his fields is contracted. No apparatus is needed. You bid him stand opposite to you, and fix your right eye with his left or vice versa; at the same time you both shut the other eye, then, moving each hand in turn midway between him and you, you roughly map out his field, making your own the standard of comparison; any great contraction of the visual area will be thus easily revealed.

(iii) *The light sense may be greatly reduced.* This is a characteristic of glaucoma, though but little attention is usually paid to it. From the patient's point of view it is a matter of great importance. Print or writing no longer stands out vividly like it used to do, even in a good light, and he finds great difficulty in reading when the illumination is of poor quality. When he passes from bright light into darkness, or comparative darkness, he has great difficulty in seeing until his eyes have become dark adapted. On the other hand, when he passes from a poor light into a bright one, he is dazzled, and must wait until he can get accustomed to the glare.

(iv) *Reading test.* In spite of the fact that the patient can make out all the letters on the 6 m. line of a test

card, he may be conscious, perhaps only dimly so, that he does it more slowly, and with more difficulty, than he used to do formerly; or perhaps he may in past time have had vision well above the normal, and be dimly conscious of the change in his visual power, though he would find it difficult to express the change to a sceptical surgeon.

To sum the matter up: When a patient, who you have reason to believe is honest, thinks that his sight is failing, it is unwise to dismiss him as a *malade imaginaire*. Remember that simple glaucoma may be far advanced, with nothing to indicate its presence but what I have ventured to call "the triad of glaucoma signs"—(a) contraction of the visual field; (b) cupping of the disc, and (c) failure of central visual acuity. Even then the last named defect may be but poorly marked.

Differential Diagnosis.

We shall now shift our ground and assume that the gradual loss of sight has not only been complained of by the patient, but has also been recognized as a clinical fact by the doctor. What dangers still attend us? In other words, with what other conditions are we likely to confuse a chronic glaucoma? I speak from frequently repeated practical experience, when I answer: (1) Cataract, (2) refraction errors, (3) brain trouble, and (4) optic atrophy. We shall consider these in turn.

1. Cataract.

Not a great while ago I saw a patient who had been sent to see a specialist by her doctor. She was told she had cataract, and must wait until she was worse before she could be made better. Six months later the patient made the striking and alarming observation that her "sight was at some times much worse than at others"; her doctor found she had cupped discs; the specialist confirmed it, and I had no difficulty in agreeing. Such a mistake should not occur. A cataract can always be seen, and that quite easily, by a method which I do not think you will find in the textbooks. Put a +10 lens behind the eyehole of your ophthalmoscope, and examine the eye (preferably with the pupil dilated) at a distance of five or six inches, moving backwards and forwards until sharp focus is obtained; every detail of the lens opacity will stand out clear to your view against the bright fundus reflex. Your diagnosis is a positive one, and is very easily made. As time goes on you will be able to follow the increase in the strokes or dots that make up the opacity. On the other hand, the cupping of the disc and, what is often more marked still, its pallor will put you on your guard against glaucoma.

2. Refraction Errors.

When a man finds that his sight is steadily failing, and that the careful correction of any errors present does not help him to see again, you may assume that something is wrong either with his media, or with his retina and optic nerve. Once again the diagnosis is not difficult in most cases; what is important is that the possibility of the presence of glaucoma should be borne in mind, and that the examination of the patient should be careful and on routine lines. There is a great deal of rubbish being sold the public to-day—probably more than ever in history—and medical advice must sometimes be included in the category. It is not that men—specialists as well as practitioners—cannot do good work if they choose; what is wrong is that the hurry and bustle of an aeroplane age lead many to give too little time to their cases, with the result that they do bad work. To put it on the lowest of all grounds: It does not pay. Make up your mind that you will not do it! I knew a chemist who advertised, "I cork my reputation in every bottle of medicine I send out." Try that in your practice.

3 and 4. Brain Tumours and Optic Neuritis.

What has just been said applies here again. Any man can use an electric ophthalmoscope; it requires no learning; any intelligent man can see an optic neuritis and distinguish it from a glaucoma.

5. Optic Atrophy.

Now, here we come to the one real difficulty—namely, the differential diagnosis between the various forms of optic atrophy, inclusive of that due to chronic glaucoma. If the discs are pale and the cupping is not pronounced, do

not hesitate to call in expert aid. You will be wise in your own interests and kind to your patients if you do so as early as possible.

A word may be said about "tobacco amblyopia," because it is so very susceptible to satisfactory treatment. A rough test will suffice in most cases. Take a clean sheet of white paper; on it make a number of dots with black ink, and then a number of similar dots with red ink; give your patient a pencil, and bid him rapidly tick off the red dots; if he can do so without hesitation, it is most unlikely that he has tobacco trouble. If he cannot do so, and if he is an excessive smoker and has been gradually losing his sight without a recognizable cause, except possibly pallor of the outer quadrant of his disc, you may win much kudos by treating him firmly and promptly for tobacco poisoning.

(B) Gradual Loss of Sight, with Intermittent Headaches.

In all such cases be on the look-out for glaucoma as a possible causative factor. Do not be content to treat your patient for biliousness, or for a nerve breakdown—that blessed modern refuge of the diagnostically destitute—until you have at least thought of glaucoma and cerebral tumour. With regard to the former, as it is our subject for to-day, make a point of seeing your patient in an attack, and look for the dilated pupil, the rise in tension, the shallow chamber, and the evidences of ocular congestion that attend rises in intraocular pressure. In the intervals examine his discs and his visual fields. If you are still in doubt, get an expert opinion.

(C) Marked Losses of Sight, clearly following Severe Attacks of Headache.

We have already discussed this subject under the heading of "headaches," and need only now mention the possibility of the eye condition being altogether missed. If the surgeon bears the possibility of its occurrence in mind, he will not go wrong.

ENTOPTIC PHENOMENA.

Photopsiae, or subjective sensations of light, are a common and early symptom of congestive glaucoma. They may take many forms, one or more of which may be described by the same patient. Thus we may hear of flashes of light, compared to summer lightning, or in the tropics to dancing fireflies; of a ball of fire that rolls across the field of vision from side to side, of sudden flash-like spots of light, or of a continuous luminous glow, which may last for seconds, minutes, or even longer. The patient may volunteer the information that he sees these phenomena most when he is tired, and that they are especially common when he is getting into bed, or has just turned out the light, or soon after he has lain down to sleep. It is to be remembered that photopsiae are seen under many conditions other than glaucoma; in fact, they are likely to appear whenever the retina is irritated, dragged upon, or in any way interfered with. Nor must we forget that similar phenomena, but of central origin, are met with in neurasthenic patients under many conditions. The moral is that such phenomena always indicate the necessity for a careful ophthalmoscopic examination, as well as for a thorough investigation of the patient's refraction, and of the state of his general health.

RAINBOWS ROUND LIGHTS.

These are seen when anything occurs to interfere with the natural transparency of the cornea. They are best observed when the patient looks from a little distance at a bright light in the dark. At least two colours are discernible—an inner blue or violet and an outer red; a yellowish band is generally seen between them. The diameter of the halo is from 7 to 8 degrees. Sometimes the patient has to be warned to look for them before he can observe them; in other cases they are so brilliant that every tiny flickering flame in the firelight is iridescent with colours. Some patients speak of them with amused interest, whilst others are inspired by them with deep alarm. Any one of you can observe the phenomenon for yourselves by breathing on your glasses and holding them up to a bright flame or electric light. Anything which interferes with the transparency of the cornea may be productive of haloes, and these may therefore be seen in other conditions beside glaucoma. They are invariably

to be found after treating the iris with silver nitrate. They are also, though not commonly, seen in the early stages of cataract. It must not be forgotten that there are apparently healthy eyes which habitually see haloes round lights in the dark; this phenomenon is probably connected with a faulty state of the patient's refraction. Transient haloes round lights are often complained of by those who suffer from conjunctivitis associated with mucous discharge. Wiping or washing away the offending flake of mucus from in front of the cornea will abolish the symptom.

CONCLUSION.

It is not pretended that this lecture is exhaustive; it merely touches the fringe of a large subject, but it is an effort to help you, by drawing your attention to matters which may too easily be relegated to the background of your mind, with harmful results to your patient's sight and with damage to your own reputation.

NEUROLOGICAL JOTTINGS.*

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I WISH in these notes to direct attention to some points of interest that have come under my notice in the course of neurological work.

Complications of Epilepsy.

When I was seeing, through a long course of years, many epileptics every week at Queen Square, I was not infrequently surprised by curious developments. I remember, for example, one man, treated for many years before I saw him as a chronic epileptic. He had been attending the hospital for seventeen years, and there was nothing in his history to suggest that his case was anything more than one of chronic epilepsy. But one day he appeared at the out-patient room complaining of severe headache, and ophthalmoscopic examination revealed the presence of marked optic neuritis. He was admitted to the hospital, and died in a few weeks of a large tumour of the right hemisphere. Another case which surprised and interested me was that of a healthy looking young man, a railway signalman, who came as an out-patient one afternoon with the history that he had had an attack characterized by loss of consciousness, convulsions, and tongue biting. There was no family history of any such illness, and his personal history was uneventful. He had never had syphilis, and he had no optic neuritis. There was no doubt that the attack was of the nature of epilepsy, and naturally he could not remain at his work as a signalman. He became a barrier ticket collector, and for over four years attended the hospital regularly, and was treated with bromide. At first the attacks occurred about once a month, but they gradually disappeared under treatment. He continued to attend, and one day complained of severe headache which he said he had had for two or three weeks. He had not been subject to headaches before. I examined his eyes then (they had been carefully examined from time to time before, but nothing abnormal discovered) and I found a flaring optic neuritis in each eye. He was admitted, and a decompression operation done to relieve his headaches. He had a large growth in the left hemisphere, from which he died in a few weeks.

I could record a good many similar cases, and I feel sure my colleagues at Queen Square have had similar experiences. I suspect they are cases characteristic of a large group, and the existence of such cases shows how necessary it is not to be content with the diagnosis of "ordinary epilepsy." I remember Dr. Hughlings Jackson, remarking on one occasion that there is no such thing as a case of "ordinary epilepsy," and that every case of convulsion or so-called fainting ought to be investigated most fully and carefully, and kept under observation. These cases would seem to "point the moral," but it is obvious that diagnosis of the true cause of the convulsion is in the first instance impossible.

*Part of this paper constituted the Presidential address to the Harveian Society.

Variability in Response to Treatment in Epilepsy.

I have frequently been struck with the variability in response to treatment of some cases of recurrent loss of consciousness with or without convulsion. One case will not be influenced by bromide until he is getting up to something like 30 grains thrice daily. It may then be found that such a patient not only remains free from attacks with such treatment, but improves greatly in general health. I have a patient I see now about twice a year who for the last seven years has taken 35 grains twice a day, and has remained well, and able to carry out responsible duties in a City office. I have another patient, a lady, who for five years has remained well although taking 30 grains thrice daily.

On the other hand, one has seen patients who, with large doses of bromide, become progressively worse, but who improved on different treatment. One little girl was brought to me at the hospital some years ago who suffered from frequent attacks, several a day, of loss of consciousness without convulsion. She was only 6, and I put her on 10 grains of bromide thrice daily. In a month I saw her again. The attacks had become more frequent. I stopped the bromide for a month and saw her again. The attacks were not so frequent as while she was taking the bromide, but as frequent as when I saw her first. I then put her on 5 minims of tincture of belladonna thrice daily and saw her again in a month. There had been no attacks during that time and she remained well. Of course, I cannot explain this. I only state the facts. I understand that a similar result not infrequently follows the administration of *Adonis vernalis*.

Another interesting point I have observed about so-called "nocturnal" epilepsy. I may say, in passing, that this is an unfortunate term and really incorrect. It would seem to indicate that in these cases the fits occur only at night, but this is not the case. It is really sleep which determines the onset of an attack, for in many of these cases of so-called nocturnal epilepsy inquiry will elicit the fact that a fit occasionally occurs during the day, but only if the patient has happened to go to sleep during the day. The point, however, which I wished to emphasize is that while bromide alone is very useful in these cases, its effect seems to be reinforced by the addition of tincture of digitalis, and 30 grains of bromide with 5 minims of tincture of digitalis taken nightly are, as a rule, effective in controlling the attacks.

Dislocation in Epilepsy.

There is one point about epilepsy which is worth remembering. As is well known, after a Jacksonian fit affecting one side and starting in the arm there is frequently temporary weakness of the affected side, usually more marked in the limb in which the convulsions started—that is to say, in the arm in such a case as I refer to. But it must also be remembered that dislocation at the shoulder-joint not infrequently occurs in the course of a convulsion. I remember one case in which one of these conditions was remembered by the observer but the other forgotten. A junior colleague of mine, in my absence on one occasion, saw my out-patients. One of them was a young man subject to occasional fits. He came up on this occasion saying that he had had a fit the previous week and found that after it he could not use his arm. There was a note made to the effect that weakness of the right arm was present, and that although the right leg was not complained of there seemed to be an exaggerated knee-jerk on that side. Obviously my colleague, who had not seen the patient before, thought he was dealing with a Jacksonian fit with weakness of the affected side following the fits. But when the patient came up the following week it was found that he had only a dislocation at the shoulder-joint, and when this was reduced the weakness cleared up.

Recurrent Attacks of Herpes.

I have been considerably interested in herpes, and one or two jottings on this disease are, I think, of interest. I rather think we were all taught that a second attack of herpes is practically unknown. I think I am right in saying that that great observer, Jonathan Hutchinson, was in the habit of telling us that second attacks of herpes did not occur. I have seen three cases which certainly controvert this. One is the case of a man whom I saw at the Moorfields Eye Hospital for some eye condition unrelated