# Section of Medicine.

President—Dr. HUGH THURSFIELD.

## DISCUSSION ON MIGRAINE.

### Dr. C. P. SYMONDS.

THE subject we are to discuss is one about which much has been written and said in recent years. It held, for instance, the place of honour at the Annual Meeting of the Neurological Society of France, in 1925 [18] and was debated at the last Annual Meeting of the British Medical Association [2]. Such popularity should mean that there have been recent important additions to our knowledge. But from a perusal of the literature I cannot find that this is the case. There has been a good deal of debate and speculation but little methodical investigation.

I have no original observations to put before you this evening. The clinical features of migraine are so well described by Liveing [10] in his book published in 1873 that it is difficult to find any aspect which has not been accurately recorded in his pages.

I propose first to plead for a clearer definition of the subject, then to hold up to critical review the main facts and theories at our disposal, and finally to suggest certain lines along which further research should proceed.

The lack of progress in our knowledge of migraine is partly due to faults in our technique of observation. We have been using too low an objective and trying to take too much into the field. There is a tendency to include under the term "migraine" any obscure form of recurrent headache. This, I think, is a mistake. We know very little yet about the mechanism and ætiology of headaches in general. It is true that in recent years there have been notable advances in our knowledge of intracranial physiology, particularly that of the sources and flow of the cerebro-spinal fluid, and the application of this knowledge has provided a basis for clearer thought and fresh speculation. Through it we have been able to remove from their former obscurity the headaches which, without any objective sign of organic disease, may follow even minor injuries of the head. In another quarter the rhinologists, particularly Sluder, have defined a group of headaches which they believe to be due to disordered function of the nasal sinuses. Both these forms of headache are paroxysmal and recurrent, and may be associated with nausea, and might be—probably have been—at times regarded as migraine. I believe that in this direction there is a large territory still unexplored and that many kinds of headache still await proper classification. Their common feature is the recurrence of attacks over a long period without any evidence of organic disease. To include all these under the head of migraine may be convenient at times. It gives the physician a convenient "label" for his patient. But for the purposes of scientific investigation we should begin by reserving the term "migraine" for a group of cases which in

their clinical characteristics are so well defined that we should have no difficulty in separating them from the main body of obscure recurrent headaches.

I propose, therefore, to define migraine as a liability dating from early life to recurrent attacks of headache, which are at one time or another associated with a characteristic visual or sensory disturbance and are frequently accompanied by nausea or vomiting. This definition excludes a number of cases of recurrent sick headache, which we should all probably regard as incomplete migraine, but if we adhere to it we shall be certain that we are all talking about the same thing. It will be one thing to say that eye-strain or the ingestion of hot buttered toast may cause sick headaches, it will be quite a different thing to say that they cause migraine. The narrower definition will also help us to focus our attention upon a few outstanding clinical facts instead of diffusing it over a large field whose details are obscure and whose boundaries are uncertain.

## ÆTIOLOGY.

In the ætiology of migraine, as thus defined, there is one outstanding factor generally recognized as of first importance—heredity. It is the rule rather than the exception to obtain a history of direct inheritance, and a Danish writer quoted by Christiansen [18] has recently offered proof that migraine is a dominant hereditary factor in the Mendelian sense. But the attacks are occasional. Given the inherited liability, what are the precipitating causes? Most writers from the time of Liveing onwards have put first in this category mental stress and fatigue. With this I emphatically agree. To the question, "Have you found any particular circumstances likely to bring on an attack?" the commonest answer is "worry" or "excitement." As to the other occasional factors, there is much diversity of opinion. Much has been written and said about the importance of diet. Fothergill [10], one of the earliest writers upon the subject and a sufferer himself, was convinced from personal experience that fats were responsible, and of butter he says, "nothing more speedily and effectually gives the sick headache." This observation has been often repeated and has been brought again to notice in some recent correspondence in the *British Medical Journal*. Marmaduke Fawkes [6], in particular, has drawn attention to the appearance of acetone in the breath and in the urine when an attack is impending, suggesting that there is some defect in fat metabolism as the cause. The withdrawal of fats from the diet and their replacement with glucose together with large doses of alkalis, he states, is often successful in averting the crisis. Allison [1] on the other hand, claims that an excessive sugar intake is the important cause and that elimination of sugar from the diet is frequently successful in prevention. Curtis-Brown [4] considers that the sole cause of migraine is an inherited or acquired defect in protein metabolism. This defect is, in a sense, selective. That is, in one patient it may be beef protein and in another mutton protein, which is imperfectly metabolized and leads to the formation of toxic by-products. The attacks can be prevented by putting the patient first on a protein-free diet and then building it up gradually, eliminating any particular food substance which causes an attack. In criticism of these observations it must be remarked that none of these writers presents any detailed instance of an attack of migraine, coming within our clinical definition, which has been investigated and treated on the lines suggested by them. A few biochemical investigations have been published upon these lines, but with no very conclusive results. Pagniez and Nast [13], however, have recorded a case described as a typical migraine with visual disturbance, in which the attacks were provoked, as a rule, by the ingestion of chocolate. This was followed by a hæmoclasic crisis and after a delay of some hours the attack developed. The liability to attacks could be notably diminished by giving the patient

a dose of peptone by the mouth an hour before meals. This, as a single observation, is suggestive. It has also been held that the attacks are due to acidosis, and may be averted by giving large doses of alkali. On the other hand, Weismann-Netter [16] has arrived, by means of biochemical investigations, at an opposite conclusion. Making serial observations upon the pH and alkaline reserve in two cases, he found that during the free interval the figures for these were normal, but that forty-eight hours before the attack in each case a marked alkalosis developed, the pH, for instance, rising from 7·36 to 7·5, and the alkaline reserve from 59·1 to 73. Paullian [14] has recorded an excess of cholesterin in the blood preceding the attacks. In criticism of these and other such observations which I have been able to discover, it must be said, in the first place, that the clinical data are meagre or unsatisfactory, and secondly, that the biochemical observations have been few in number. The mere occurrence of gastro-intestinal disturbance during the early stages of the attack cannot be taken as evidence of a gastro-intestinal origin.

Of eye-strain I would say that in the particular group of cases we are discussing it is rarely an important cause of attacks. I believe there are recurrent headaches due to errors of refraction and muscle balance, which in many cases can be cured by proper correction, but these should not be included under the head of migraine.

## Symptoms.

I have made it a part of my definition that at one time or another the attacks shall have been associated with certain characteristic visual or sensory disturbances. These phenomena are worth studying carefully not only for their diagnostic value but for the light or darkness they may throw upon the vexed question of pathology.

The true fortification spectrum leading on to a homonymous defect in the visual fields is comparatively rare. Much more commonly we meet with a simple defect which may or may not be preceded by vague flashings. This defect may vary from a mere dimness or mistiness of vision to a complete loss. It is usually restricted to a part of the visual field which can be accurately described by the sufferer in answer to leading questions. Usually the defect is a vertical homonymous hemianopia. In the same patient it may be right- or left-sided in different attacks. Sometimes it is definitely quadrantic. It may in some attacks be bilateral, resulting in complete temporary blindness.

There is, however, another less common variety in which the visual fields are divided transversely. Such transverse hemianopia may occur on some occasions in a case in which the more common vertical defect is the rule.

As yet another variant we meet with transient central scotoma, usually bilateral, sometimes affecting one eye only. Finally we may encounter monocular blindness. It is characteristic of all these disturbances of vision that both onset and recovery are rapid and that recovery is complete.

The sensory phenomena are equally distinctive. A sensation of numbness or tingling, beginning in one or sometimes both hands, or in the tongue or lips, spreads slowly from hand to mouth or vice versa. The sensation is similar to that observed in the sensory form of Jacksonian epilepsy, but there are several distinguishing features. The spread in epilepsy takes a matter of seconds or, at most, minutes. In migraine it continues from a time varying from ten minutes to half an hour. The tingling of tongue and lips in migraine is always bilateral, in epilepsy never. Nor are both hands ever affected in an epileptic attack. If the right hand is affected in one of these attacks of migraine there is frequently some disturbance of speech of an aphasic nature. The lower limb is rarely involved.

The sensory and visual disturbances may appear in combination, the one

preceding the other: more usually they occur singly. As a rule the visual or sensory disturbance precedes the headache but may accompany it. It is often associated with only a small proportion of the headaches in an affected person. It may occur without the headaches as an isolated phenomenon.

The headache is of a throbbing, bursting or splitting character. It is commonly unilateral and frequently on the side opposite to a homonymous hemianopia. It is almost as frequently bilateral. If severe it is commonly associated with nausea and vomiting. Both nausea and vomiting, however, may occur in the presence of slight headache, and, in children especially, vomiting may be the predominant feature of the attack. Many of the attacks known as cyclical vomiting in children appear to be predecessors of true migraine. It is worthy of remark that the attacks usually commence in the early morning and the patient often wakes with the knowledge that his attack has begun.

Some persons have a preliminary warning in the form of a peculiar sense of lassitude, or, less often, of extreme well-being the day before an attack is due. Such are the main facts familiar to us all which I have thought it well to recapitulate before embarking upon the difficult problem of

## PATHOLOGY.

The headache is of a kind frequently met with in conditions of increased intracranial pressure, but I would submit that our knowledge of the mechanism of headaches in general is not yet so exact that we may argue from this that increased intracranial pressure is the cause of the headache in migraine. We have therefore to study the other phenomena and it will be profitable first to inquire whether from the data at our disposal we can localize the seat of the disturbance to any part or any level of the brain or brain-stem.

We may take as our starting point the common vertical homonymous hemianopia. This might conceivably be due to a disturbance of function anywhere between the optic chiasma and the occipital cortex. There is one point, however, in favour of a cortical situation—the hemianopia of migraine is frequently preceded by flashes of coloured light. This symptom is not met with in organic lesions of the optic chiasma, tract or radiation, but does sometimes occur with lesions of the occipital cortex. From this point we turn naturally to the sensory phenomena. The right-sided attacks with aphasia are again suggestive of a postcentral cortical situation. There are, however, several difficulties to be met. If the disturbance is cortical, how is it that the tingling in lips and tongue is always bilateral, and that in the hand sometimes so? If we are going to adhere to our cortical localization we must assume that there are two separate areas of disturbance on the two sides of the brain which are, in the instance of tongue and lips, remarkably symmetrical and simultaneous.

Turning now to the other visual phenomena it is conceivable that the transverse hemianopia might be due to symmetrical bilateral disturbance situated above or below the calcarine fissures. Since the macula has cortical representation it is also conceivable, though less likely, that the bilateral central scotomata might be due to symmetrical disturbance at the occipital poles. But what are we to make of the unilateral scotoma or monocular blindness which is said to occur? The imagination cannot be stretched to include this phenomenon within the hypothesis of cortical localization. We should, therefore, be critical of such instances and inquire whether they are indeed within the limits of our definition of true cases of migraine.

I have notes of one such case only, which, though I made the clinical diagnosis of

migraine, ought not strictly to be included, as up to date the visual phenomena have not been accompanied by headache.

The patient, aged 22, is a sister of one medical man and the secretary of another. About three years ago she first noticed occasional attacks in which a quivering luminosity appeared on one side of objects looked at; sometimes to the right, sometimes to the left. The attacks would last about half an hour, usually occurred in the early morning, and were not attended by any other disagreeable sensation.

As separate phenomena she has had two other kinds of attack. In one variety everything she looked at had a black spot in it, though she could see everything all the way round; in the other kind little lighted matches would float down in front of her eyes. Both forms of attack lasted usually half an hour and were apt to occur in the early morning.

These attacks had appeared to her of so trivial a nature that she did not think of seeking medical advice until she had the following experience: About 10 a.m. one morning at her work she noticed that, as it were, something had been clamped over the left eye. She shut the right eye and found she could not distinguish between light and darkness. Then after ten minutes the blindness went half way up like a blind being drawn up. For a minute it stuck at the horizontal level, then rolled right up. The whole attack lasted twenty minutes.

She was examined by an oculist a day or two later, who found nothing amiss apart from a slight error of refraction. There were no signs of any organic nervous disease. None of her attacks were associated with headache or nausea.

As I have said, the absence of headache in this case precludes its acceptance as a definite instance of migraine, although the story of the visual phenomena is suggestive. Writers on the subject of migraine, however, commonly refer to the recurrence of monocular blindness in true migraine. I have not met with a well documented instance in the literature. Perhaps some of you can make good this defect from reading or personal experience. The point seems to be one of importance, for the occurrence of monocular blindness would exclude any conception of migraine as a purely cortical disturbance.

We may conclude that with this possible exception the *situation* of the disturbance in migraine lies in the cerebral cortex, particularly in those parts in which vision and sensation are represented, and that this disturbance is often bilaterally symmetrical.

Having arrived so far we have to inquire what is the *nature* of the disturbance. Here we are on much more difficult ground, for the cortex is beyond our ken. We have, therefore, to depend largely upon analogies from similar disturbances in other parts. The theory of vascular spasm is an old one and, I would submit, the most probable. In the first place it offers an adequate explanation of the order in which the visual phenomena appear. We are familiar with the principle that minor degrees of anoxæmia lead first to exaltation and subsequently to depression of nervous function (e.g., cerebral anoxæmia of aviators); in the second place it offers the most intelligible explanation of the frequent symmetry of the disturbance on the two sides, for which we have a striking analogy in Raynaud's disease. In this latter affection, which we know from observation to be due to transient vascular spasm, it is common to find simultaneous involvement of corresponding digits in the limbs of the two sides. In this connexion it is worth dwelling for a moment on the fact that migraine and Raynaud's disease, although they must be regarded as two separate affections, are not infrequently encountered in the same individual. I have selected the following as a striking example:—

A married woman, aged 31, stated that for several months she had been subject to attacks in which her fingers would go white and cold and remain so for about an hour. Usually two fingers were involved and both hands were symmetrically and simultaneously affected. The attacks were especially brought on by cold.

Two months before I saw her, she had her first attack of migraine, after bathing in the sea. She first noticed a haze in the left half of her visual fields followed by the appearance of serpentine figures, left hemianopia, and numbness of the lips and hands. These sensations from start to finish lasted three-quarters of an hour and were followed by a very severe bi-frontal headache, which continued for four hours and culminated in vomiting, after which she felt perfectly well again, being able to go to a concert two hours later.

Subsequently she had experienced three further attacks. Two were repetitions of the first. In the third the "top half of things went dim," she experienced nausea, numbness of the left arm, of both lips and both sides of the tongue, and also in the left leg. Her "inside seemed to quiver" and she had much flatulence. This was followed by a terrific headache. Next morning it was gone but she felt "woolly" all day.

There were no signs of any organic disease nor have any developed since, though she has had further attacks of migraine. There was no family history in this case, but she herself had been subject to severe bilious attacks in her childhood.

Instances have also been recorded by Osler and others of transient disturbance of vision and sensation occurring at rare intervals in the course of long-standing cases of Raynaud's disease.

Proceeding with the hypothesis of vascular spasm, can it explain the headache? The suggestion originally made by Du Bois Reymond [5], himself a sufferer, was that the arteries during an attack of migraine are in a state of tetanic contraction which is itself painful. "The latter view," he says, "takes for granted that the tonic contraction of the smooth muscular fibres is not less painful than that of the cross-striped fibre, as in cramp of the legs, tetanus from electrical tetanization and the like. The pains of labour and of colic justify us in making this concession."

I find some difficulties in the way of accepting this explanation. If tonic spasm in the arteries is at the same time the cause of the visual and sensory disturbance and of the headache, how is it that the visual or sensory disturbance as a rule precedes the headache, and that either may occur, in fully developed form, independently?

It would be more satisfactory to suppose that we have an analogy with the blue form of Raynaud's disease, and that as in that disease local asphyxia may lead to local œdema. This would occur more readily in the brain owing to its loose texture than in the fingers. This hypothesis would better account for the common sequence of events, and would, I think, explain the headache. We know from our experience of cerebral contusions that a local swelling upon the surface of the brain may be quite small and yet, provided that it is of sudden development, give rise to very severe headache. A small cerebral contusion may result in severe pains; a meningeal tumour ten times the size may never have caused a headache, presumably because the slow development of the tumour allows room for moulding and displacement, so that the tension upon the meninges is evenly distributed throughout the whole envelope and does not reach the pain threshold at any one spot.

I have one further observation to put forward in favour of this hypothesis, and that is the occurrence in some cases of true migraine, during an attack, of subarachnoid hæmorrhage. I have met with two examples of this condition, one of which I have published [15]. Goldflam [9] has particularly drawn attention to this point and has recorded five cases of the association. It is at any rate conceivable that local asphyxia and œdema might lead to local hæmorrhage and rupture into the subarachnoid space.

Finally, it is perhaps worth observing that the theory of vascular spasm would explain the occasional occurrence of monocular blindness, the spasm being presumed in such instances to affect the ophthalmic or retinal vessels. There are on record observations with the ophthalmoscope of such spasm during an attack. Higgens, quoted by Ormond [12], stated—

"that he once had a patient whom he examined on several occasions during temporary attacks of monocular blindness associated with migraine, in which he could see the retinal arteries reduced to mere threads while the veins were full and distended; by watching the fundus he could tell when vision was returning by noticing the enlargement of the arteries."

Unfortunately the data upon which the case was diagnosed as migraine are not recorded.

If the attacks are due to paroxysmal constriction of the vessels, through what mechanism is this produced? The prevailing view upon the Continent is that it is due to over-action of the cervical sympathetic. But if we accept the work of Howard Florey, published in a recent number of *Brain* [7], this cannot be true. From direct microscopic examination of the living cortex of the cat, rabbit and monkey he satisfied himself that neither stimulation of the cervical sympathetic trunk, nor of the vasoconstrictor centre in the medulla, produced any effect in the cortical vessels. The same negative results were obtained from the local application of adrenalin. Florey found, however, that local constriction of arteries and arterioles could be produced readily by the application of mechanical, electrical or thermal stimuli. Experimenting with a few chemical substances he found that a 5 per cent. solution of barium chloride caused marked constriction, and a solution made by shaking amyl nitrite with water caused marked dilatation. The other substances tested were inactive.

It would appear, then, that if vascular constriction is the cause of migraine, it must result from a purely local disturbance of function in the vessel walls. Possibly the same is true of Raynaud's disease [3] [17], for which a nervous origin has not been proved. Such local constriction might be due to some chemical substance in the circulation, or to functional defect in the muscle itself—for which we have as an analogy in the case of striped muscle, the myotonias. In any case we must assume an inherited or acquired local susceptibility.

I fear that in this part of my remarks speculation has outrun the facts, but I have dwelt upon it at some length because I feel there is enough to suggest the lines along which further investigations should be made.

## Treatment.

Of the treatment of migraine there is little new to be said. Having admitted the importance of the hereditary factor in the majority of cases, we cannot speak in any strict sense of a "cure," and the liability to attacks continues, though frequently diminishing with advancing years. In almost every case, however, efficient treatment will succeed in diminishing the frequency—and often the severity—of the attacks. Of these measures regime would appear to be of the first importance. We cannot forbid worry, but we can often prescribe conditions of work, mental and physical, recreation and diet, which are of the greatest value.

Of drugs, luminal in some cases, and in others liquor trinitrini, given regularly as preventive measures, are of undoubted value. Individual patients vary in the degree of benefit which they may obtain from these two drugs, so that it is worth trying them alternately.

There are patients who claim to have discovered for themselves special precipitating causes, dietetic or otherwise.

Once the attack has begun the patient as a rule knows best from experience how to care for himself, complete mental and physical rest being the chief ingredients of his prescription. Occasionally he may be able to avert an attack by some method of his own. The most commonly successful in slight cases is a dose of aspirin and

complete rest for an hour or two. If with this, sleep is obtained, the development of the attack is often checked. Sometimes, especially if the attack begins at the end of a period of fasting, a good meal with a glass of wine will cut it short. A medical friend who has a family history of migraine and suffers from definite attacks with visual disturbance tells me he can often avert them by hanging his head downwards for a few minutes. I have advised this measure in other cases without benefit.

Of refractive errors I have already stated my opinion that they may cause recurrent headaches on their own account, but are not an important cause of migraine. The question will doubtless be debated with acrimony. I will conclude my remarks upon this point by citing in my support Mr. Foster Moore [11] who in the most recent edition of his book "Medical Ophthalmology," says: "Whilst nothing but good will come of the correction of any real error of refraction that may exist, there is, I believe, no direct causal relationship between any ocular abnormality, refractive or otherwise, and migraine."

## SUMMARY.

In summary of these remarks I would say that the evidence which I have reviewed suggests that migraine is due to a susceptibility, inherited or acquired, of the cortical vessels to spasm of their walls. There is nothing to show by what means this spasm is provoked, but mental stress in some way, at present obscure, plays an important part. Possibly the reaction is of a chemical nature and is due to the action upon the vessel walls of toxins derived from neural metabolism.

Other toxins derived from faulty metabolism of fats, sugars or proteins may also be responsible.

There is some evidence to show that the attack may be associated with an alkalosis or with an excess of cholesterin in the blood.

There would seem to be at least two lines of investigation worth pursuing. On the experimental side, further observations should be made along the path opened up by Florey—the effect of various chemical substances, e.g., cholesterin, acetone derivatives, acids and alkalis, upon the cortical vessels, either when applied directly or introduced into the blood-stream. On the clinical side, a number of cases of true migraine, coming within the definition of this paper, should be investigated by the clinician and biochemist in collaboration during the free period, before, during and after the attack. From a comparison of the results obtained by two or three teams working upon these lines we might hope to gain some positive knowledge of the part played by diet and faulty metabolism in the causation of the attacks, and so set this part of our treatment upon a firmer basis.

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regretted that a medical friend (aged 50) of his could not personally be present, but he had written an account of his symptoms, which he now proposed to read, with the addition to the account of what he himself had observed in the bad attack recorded, the patient's record of which is, perhaps naturally, inadequate.

My first experience of migraine was at the age of 18 when a student. The attack occurred at about noon. It was of the same character as most of my attacks. A central clear space of an elliptical shape surrounded by an irregular luminous, in fact dazzling border beyond which was a misty area. On closing the eyes the effect was changed to a central dark spot with a very dazzling frame, outside of which was an area of rapidly revolving bodies. The sensation gradually increased in intensity to a maximum and then gradually passed completely away. These attacks usually lasted for about 10 to 20 minutes. Sometimes the right eye only was affected, sometimes both. As a rule a headache followed in about half an hour or so, but I soon found that I could prevent this by taking a phenacetin tablet directly I got the first symptoms. Not being a "sick" subject I never had any vomiting or even feeling of sickness. I have often noticed my right pupil larger than the left, and I believe, though I cannot be certain, that this was generally more marked during an attack. In my own mind I am quite sure that the attacks are most commonly the sequel of slight digestive disturbance sometimes associated with nervous exhaustion following continuous hard work especially if the latter is accompanied by mental worry. My two worst attacks (of a different character) followed a time of marked mental and physical exhaustion after very hard work. The attacks most frequently occurred in the morning between 9 and 11 but occasionally at later hours in the day. From my personal experience I generally treated my patients with an initial dose of aperient of a saline nature followed by phenacetin or potassium bromide and have usually found this treatment satisfactory in ordinary attacks.

In 1920 I had an attack of a different type. When dressing in the morning I suddenly noticed that I had slight diplopia. On looking in the glass I found that internal squint was present. Thinking that this was only an ordinary attack of migraine I expected the usual visual effect. Instead of that the diplopia continued and later the vision became so obscured that I had to rest. There was also some mental confusion as, although I thought I was perfectly clear and indeed said so, yet I learnt afterward that I made several silly remarks and feeling rather sleepy I agreed to go back to bed. Drowsiness supervened and although I awoke when anyone came into the room yet I found the diplopia still present. Headache was present to a large degree, but I was perfectly certain that the whole thing was of the migrainous type and that it would pass off in a short time and assured my friends of this. Early in the afternoon this had occurred and I found that I was practically all right again, but being a little afraid that the diplopia might return I kept quiet for the rest of the day. The next day I was up and about and indeed surprised some of my friends who had been told that I had had a "stroke."

[*Note by Dr. Thursfield.*—This does not fully describe the real sequence of events, as ascertained at the time. The attack began in the usual way with a diplopia, but instead of passing off in a short time this was succeeded by complete blindness, some mental confusion, a partial right hemiplegia, especially noticeable in the right side of the face. He was carried to bed, and when seen by me three hours later still had an obvious weakness of the right facial muscles; and also some noticeable mental confusion, and inability to express himself properly. Of this he was aware himself, and explained that "he couldn't remember" the name or the word that he wanted. He also had a slight definite weakness of the right external rectus muscle. A night's rest

completely restored him, and he had perfectly recovered from all his symptoms next morning.]

About two years later I had another attack of a different kind. While giving an anæsthetic I suddenly became aware that I seemed to have some loss of power in my right arm and had to exercise considerable mental power to lift it up and move it about. It felt numbed and unnatural. At the same time I found that when I attempted to speak I was inclined to mumble and had difficulty in articulating clearly. There was also a tendency to laugh, due, apparently, to a twitching of the corners of my mouth, especially the left. I said to the operator "I have a slight attack of migraine, so do not worry if I seem funny as it will soon pass off." I was able to continue anæsthetics of two or three more cases and then went on my rounds. I had, however, to explain to several people what was the matter as the tendency of my mouth to twitch and also to laugh continued for some time. During the afternoon it gradually wore off. There was a slight drowsy tendency, but not enough to interfere with my work which I succeeded in getting through.

As far as I can remember the numbed sensation also was present in my right leg, but to a less extent, causing me to be extra careful in moving about. Mentally I was quite clear and perfectly certain that it was another attack of the same kind as before. The attack interested me in that it seemed to be of the nature of a functional hemiplegia. It gradually increased to a certain severity and then passed off in a similar way to all my other attacks.

## Dr. A. F. Hurst.

I agree with Dr. Symonds that migraine depends primarily upon a constitutional and often inherited irritability of some part of the central nervous system. But I believe that the most common and important factor which increases this irritability is eye-strain. In my experience of a large number of both typical and atypical cases improvement, often amounting to the complete cessation of attacks, almost invariably follows the accurate correction of errors of refraction and of disturbances in the balance of the external ocular muscles. Unfortunately very few oculists take sufficient trouble in cases of the kind, but the few who have been convinced of the need of extreme accuracy in order to relieve eye-strain, have been rewarded by obtaining most satisfactory results. In recent years I have found luminal a very useful drug in reducing the excitability of the migraine "centre," but it is not essential, and it can generally be discontinued as soon as the ocular errors have been sufficiently corrected. There is no doubt also that fatigue and toxæmias of every kind increase the liability to attacks, as exhaustion of the nervous system is accompanied by increased irritability.

My colleagues and I have investigated a considerable number of cases by biochemical and radiological methods, but we have never found any constant abnormality. Difficulty in the digestion and metabolism of meat, fat and carbohydrates, hepatic and renal insufficiency, dilatation of the stomach, intestinal stasis, and toxic idiopathies, have all been regarded at different times as the cause of migraine, but in no case were any of these abnormalities discovered. There is no doubt that many women are specially liable to attacks during menstruation, as John Fordyce pointed out 150 years ago, and attacks may cease temporarily during pregnancy and permanently at the menopause. This must clearly be due to some endocrine activity which renders the storm-centre particularly sensitive during menstruation, but with this exception there is no evidence that migraine is in any way associated with disorders of internal secretion.

Radiological investigations have shown that during an attack gastric peristalsis

ceases and a condition of atony occurs, but this is part of the migraine and not a cause. The starvation which accompanies an attack and the loss of fluid following severe bouts of vomiting also give rise to various biochemical changes, which have been wrongly regarded as evidence of a toxic origin.

Finally, I should like to urge those whose experience has led them to regard eye-strain as of minor importance to choose another oculist, or, if necessary, a series of oculists, to examine their migrainous patients. It is necessary to add that the patients must conscientiously wear the spectacles they are given all the time and must not let vanity interfere with their cure.

I have known an attack of typical migraine with hemianopia, fortification phenomena, hemicrania and vomiting occur after an interval of four years in a young woman whose very frequent and severe attacks had been completely arrested by proper spectacles after prolonged drug treatment and rest had failed. She had given up using them for over a year, when a period of physical fatigue and worry brought on the relapse. She at once returned to the discarded spectacles, and no further attacks have occurred in the four years which have since elapsed.

## Dr. E. Arnold Carmichael.

What is the opinion of Dr. Symonds as to the hypothesis which bases the symptomatology of migraine upon a transient unilateral or bilateral hydrocephalus? There are several points in favour of such a hypothesis.

(1) In cases of cerebral tumour, hydrocephalus of the opposite cerebral hemisphere frequently occurs with a resulting large pupil upon the same side. I believe, in persons suffering from hemicranial headaches of migraine, that the ipsilateral eye is dilated. This is a point suggestive of a unilateral hydrocephalus being the underlying cause.

(2) The symptomatology of migraine may be likened to a "slow" epileptiform seizure—the lights before the eyes being positive phenomena and the partial paresis a negative phenomenon. In hydrocephalus, headaches, vomiting and fits followed by varying degrees of transient paresis occur.

(3) The fact that the calcarine fissure causes an elevation on the inner surface of the posterior horn of the ventricle renders this region liable to the earliest effect of hydrocephalus. This would be consistent with the hemianopic visual disturbances met with in migraine.

(4) Spitzner has published the records of several cases of migraine where at post-mortem a degree of hydrocephalus was found.

## Dr. J. Kingston Barton

said he was surprised by Dr. Hurst's statement that all, if not nearly all, cases of migraine were cured if only the patients succeeded in finding an ophthalmic surgeon who was competent enough to find out the error of refraction which was the real underlying cause of all these cases. In his (Dr. Barton's) experience a certain number of people with most troublesome head and eye symptoms had been cured by proper spectacles being prescribed. But in most of these eye cases the patients rarely exhibited many of the stigmata or the syndrome signs that made up true migraine. Dr. Symonds had not mentioned one very important symptom which he (Dr. Barton) believed to be a crucial test of a case being true migraine, namely, the copious polyuria

that the patient developed during the height of the attack. So much so that during the period of headache, the sickness and polyuria, the throbbing heart, and the many contracted small blood-vessels (similar to those which Dr. Symonds held might be present within the cranium), almost supplied a small clinical picture of a case of contracted cirrhotic kidney. This would point to some substance requiring elimination being present in the blood, and making the heart and blood-vessels behave in a way similar to what happened in a case of actual ablation of kidney substance. If it were admitted that vascular spasm within the brain was probably the anatomical feature of migraine then one would look for something that acted through the medulla of the adrenal organs and the vasomotor system generally. Dr. Hurst's contribution to the subject of the motor paralysis of the walls of stomach was a point worth following up. It should be remembered how frequently those subject to migraine suffered with that positive exhibition of vasomotor disturbance, namely, "dead man's fingers."

It was most important that the modern medical units in hospital medical schools should use all their resources to investigate the many sidelights brought to bear on recognized cases. Young house surgeons or physicians, or probationer nurses, were the most likely persons to come across such patients on the spot.

A well-known symptom in migraine was the extraordinary feeling of *bien être* or well-being just before an attack was coming on. That was the time for biochemical investigations into the blood uric acid, the blood reaction itself, blood urea, blood cholesterol, creatinin, blood-pressures, the urine excreted hour by hour, also for ophthalmoscopic examinations, examinations of heart sounds, etc. The interval of *bien être* was generally well marked towards bed-time; the storm usually broke in the early morning.

It had been stated that many of these sufferers ceased to have attacks after 50 years of age. In his (Dr. Barton's) experience most women lost their migraine after the onset of the climacteric, but when they continued to suffer from bad sick headaches after 55 he invariably found that they had not only inherited the gouty phenomena but had continued to eat and drink much too freely, and were only cured by strict attention to diet and a yearly course of treatment at Aix-les-Bains, or Carlsbad. Men seemed to suffer less as they grew older but only because they had learned to avoid excess of meat and to be very chary of taking any alcohol. In fact, large numbers of sufferers cured themselves by a severe limitation of meat diet in daily life (especially in the case of women), and practically complete abstention from alcohol. He thought the older authors were right when they put migraine, asthma, skin affections, etc., in a group under inherited gout.

## Mr. Herbert W. Nott

briefly discussed the results obtained by permanganate treatment. It had been stated in the course of the discussion that the most probable cause of migraine, and other allied conditions, was a floating toxin in the blood, the ill-effects of which might often be controlled by means of a treatment which was found to exercise a rectifying effect upon metabolism, and in cases in which other remedies had failed.

## Dr. J. A. Ryle

said there was one pertinent question which could be put in regard to migraine, in common with the other intermittent, "explosive," disorders (namely, asthma, epilepsy, and gout), and this was: "Why were they all incurable?" For it must be

admitted that, however much we might be able to alleviate the symptoms or diminish the frequency of the attacks in these disorders, no cure in the strict sense could be achieved. Surely the answer to this question was that the disorders depended upon an inherited predisposition and that this predisposition, being, in the biological sense, a true variation and as such transmissible, was from the physician's point of view the ineradicable factor. Dr. Symonds had referred to the frequency with which migrainous patients gave strong family histories. It was not improbable that in *every* case the hereditary factor was present. "Skipped generations" could be reasonably accounted for by students of heredity and were, perhaps, more common in conditions in which transmission took place more evidently through one sex, as was the case with migraine. The discontent of the laity with the achievements of the medical profession in regard to these disorders was really unjust, for in effect they were expecting to be relieved of something which was a part of the general constitution with which they had been endowed by their ancestors.

Dr. Ryle also reminded the meeting of an observation of Lauder Brunton, supporting the view that arterial spasm was a factor in causing the pain of migraine. Sir Lauder Brunton was able to demonstrate in his own case that his temporal arteries were hard and contracted during the period of the headaches, although soft and relaxed at other times.

## Dr. Agnes Savill

said she believed migraine was often primarily of toxic origin, the error of refraction so frequently present being only a contributory or exciting cause. She referred to a family in which several members developed migraine when their vitality was low; the attacks came on, with the regularity of a clock, day after day at the same hour. In one case the migraine was arrested by a course of Carlsbad salts; in another sod. bicarb. with sod. salicyl. always cured the condition. Refraction errors were present, but though the migraine had been arrested by the methods mentioned, the glasses had not been altered.

## Mr. Frank Coke

mentioned a case in which violent attacks of migraine had been associated with asthma due to feather sensitiveness, and another in which a sufferer from migraine, sensitive to pollen, was free from migraine during the time that he was suffering from hay fever.

## Dr. W. R. Reynell.

The problem of migraine is merely a part of the larger problem of the paroxysmal neuroses, the epilepsies, asthma and the rest. It is almost certain that in all of these there is some inherited constitutional defect. The analogy between epilepsy and migraine is striking—the relatively large heredity and constitutional factors in both with similar groups of exciting causes—psychogenic, metabolic, infective and toxic. In both the symptoms are due to disturbances in the central nervous system, and in both the typical attack may be replaced by what is called a "psychic equivalent," in which the symptoms are those of a paroxysmal psychosis. Migrainous patients often give a history of night terrors or cyclical vomiting in childhood and

in cases of epilepsy there may be a history of enuresis or somnambulism in early life. When the problem of epilepsy is solved we shall know more about migraine.

## Dr. F. W. Collingwood.

I have been a subject of migraine for the whole of my life, and with two dioptres of hypermetropia. I have never had the slightest trouble after using my eyes in reading for prolonged periods. But the occasions on which the worst attacks have ensued have been after debates in which controversial questions have arisen, especially those of a political type.

It appears that in my case the state is hereditary, several of my ancestors and relatives having suffered from migraine.