

UNIVERSITY OF GLASGOW

SCHOOL OF MEDICINE

THESIS ENTITLED

A CRITICAL STUDY OF MIGRAINE WITH SPECIAL REFERENCE
TO EYE STRAIN AS A CAUSAL FACTOR.

Presented to the UNIVERSITY OF GLASGOW for the Degree
of M.D.

By Leslie Gemmill Scoular, M.B. Ch.B. Glas. D.O.M.S.

54 Welbeck Street, W.1.

ProQuest Number: 13905511

All rights reserved

INFORMATION TO ALL USERS

The quality of this reproduction is dependent upon the quality of the copy submitted.

In the unlikely event that the author did not send a complete manuscript and there are missing pages, these will be noted. Also, if material had to be removed, a note will indicate the deletion.



ProQuest 13905511

Published by ProQuest LLC (2019). Copyright of the Dissertation is held by the Author.

All rights reserved.

This work is protected against unauthorized copying under Title 17, United States Code
Microform Edition © ProQuest LLC.

ProQuest LLC.
789 East Eisenhower Parkway
P.O. Box 1346
Ann Arbor, MI 48106 – 1346

A CRITICAL STUDY OF MIGRAINE WITH SPECIAL REFERENCE
TO EYE STRAIN AS A CAUSAL FACTOR.

INTRODUCTION.

Of all the symptoms complained of by those who consult an ophthalmic surgeon, headache is by far the commonest. On questioning patients closely about this I have been struck by the frequency with which it is accompanied by nausea, and often actual vomiting. In very many cases this is attributed to biliousness, and the patient will volunteer the remark that the liver is easily upset. On probing the matter further one soon comes to the conclusion that many of these people who complain of bilious headaches and who lay the blame on their stomach or liver are really suffering from migraine. In a great many instances this diagnosis has never been made, and often they have been told by their own doctor that they have a sluggish liver, a chronic appendix, or a dropped colon. Many have actually had their appendices removed and others their colons stitched up with little or no amelioration of their symptoms.

There are two chief reasons why a diagnosis of migraine is not made as often as it should be. The first is that most doctors will only diagnose this condition when a patient presents an absolutely typical picture of the disease; that is, in short, severe headache confined to one side of the head, accompanied by fortification spectre and followed by sickness. They fail to recognise and appreciate the fact that though this is the fully developed variety, there are very many lesser grades and abortive forms of the same symptom complex.

The other reason is that patients often unwittingly mislead their medical advisers by rather stressing the attacks of sickness, and if the headache is mentioned at all it is attributed to the stomach upset, or there may actually be no accompanying headache. Again, should spots be seen before the eyes they are immediately put down to the so-called bilious attack, as the lay mind invariably associates the one with the other; and should there be any transient obscuration of vision this is often never mentioned, as the patient has come to learn from past experience that it is of

little or no significance. In this way the proper diagnosis is very often missed and the patient may be subjected to unnecessary mutilation or to a course of useless treatment.

I have now dealt with a large number of these patients, many of whom have been completely cured, whilst in others the attacks have become much less frequent and diminished in severity, and in practically all there has been some very definite alteration in the chain of symptoms. The treatment has consisted solely of the very accurate correction of refractive errors and any muscle imbalance which might be present. The results obtained in my series I find compare very favourably with those got from drug therapy, and so it has seemed to me well worth while to write a thesis on this particular method of helping these patients. I feel the more encouraged to do this as time and again one comes across the statement that the reflex factor is of very little importance, and this assertion is especially common in the more recent text-books. Several writers also mention the fact that no large series of cases has ever been published offering good evidence that reflex

action due to local irritation could cause this type of headache.

During the past year I have examined some hundred cases of migraine, collected from hospital and private practice, and the fact that I have seen this relatively large number in such a short space of time demonstrates, if such a thing is necessary, how common this very distressing malady is. Out of this number I propose to deal with the first fifty as it is only with them that a sufficient length of time has elapsed from which one can judge the results of the treatment. As I said before, this has consisted solely in the correction of refractive errors, together with any heterophoria should such be found. At the beginning of the examination each patient was interrogated in the same way and the answers written down on a form which I have had specially printed for the purpose. By this means I have been able to cover all the ground relative to the disorder in a systematic way, and so glean much valuable information with which I will deal in the body of the thesis under various heads.

HISTORY.

In the first century, Aretæus of Cappadocia described this disease which he called "Heterocrania", and to him is the credit of sifting out this particular type of headache. Fifty years later Galen described the same condition and called it "Hemicrania". This, in time, became abbreviated through the form "emigrania" to "migrana", and from this the French term "Migraine" was derived. The earliest writers believed the condition was due to various fluids or vapours in the body, which, flying to the head, caused the headache. In 1618 Lepois, who was himself a sufferer, described in an autobiography his symptoms in great detail. He attributed the disorder to effusions brought on by west winds and atmospheric changes. In the eighteenth century Anhalt and Wepfer put forward a theory that the pain was due to distension of the cerebral vessels. In 1858 Bernard published a paper on the result of stimulation of the cervical sympathetic, and demonstrated the production of angiospasm. Several years later, Liveing wrote a large text-book on the subject in which he ascribed the symptoms to a "nerve storm".

He believed that migraine was in some way related to epilepsy, and that one might pass into the other. Since the first half of the last century French authors in particular have classified migraine with asthma and eczema. Through this association Strumpell was the first to suggest the allergic basis of some cases of migraine, and regarded it as an exudative process comparable to urticaria and angioneurotic oedema.

DEFINITION.

The following is, I think, about the best and most comprehensive definition, wherein migraine is described as a periodic, incapacitating headache, culminating in nausea or vomiting, often preceded by visual disturbances, and usually followed by sleep, all occurring against a background of relatively perfect health.

THE RELATION OF MIGRAINE TO OTHER DISORDERS.

Many writers from Living onward have associated migraine with the convulsive state, and have gone so far as to regard it as a sensory form of the latter. Gowers was certainly of this

opinion, and believed that either condition might replace the other. On the other hand, Balyeat was unable to associate the two conditions, and in this he is supported by Bray. Both, they maintain, and I think rightly, are relatively common and it is not surprising that they might co-exist in the same individual. Paskind, as the result of studying the records of a very large number of patients, came to the same conclusion, namely, that there was no special relationship between migraine and epilepsy. There seems no doubt that in certain cases there is an association between migraine and some allergic conditions, and some maintain that it is interchangeable in the linkage with asthma, hay fever, and urticaria. This association I propose to deal with more fully later on when I come to discuss the possible aetiology of the symptom complex.

HEREDITY.

Timme, in the Oxford System of Medicine, states that in fifty per cent of his cases there was a ~~history~~^{any history} of heredity. In my opinion, this is much too low a figure. From the fifty patients I examined a history of migraine in near relatives was elicited in forty-one of them, giving a percentage of 82. Four others were uncertain on this point and so were not included in the figures. This tallies very closely with the percentage found by Moebius who traced an hereditary tendency in some ninety per cent of his patients. Ten of my cases stated that their fathers suffered from migraine, some including their grandfathers as well, whilst in twenty-one the complaint was on the mother's side. In three cases both father and mother were affected, and in five others there was a collateral history. In a further two migraine was found in their children. In four the family history was somewhat indefinite, and in the remaining five no hereditary tendency could be traced. With reference to the occurrence of allergic disease among these fifty, it was only from nine that a history of asthma, hay fever or urticaria

or "headache"?

could be traced in their families. In his book on Migraine Balyeat maintains that heredity is the most important single predisposing factor, and with this I am inclined to agree.

TYPE OF PATIENT.

It is often said that the mentality of the migraine patient is above the average, that they are dynamic characters and leaders in their own line of endeavour. There may be a certain element of truth in this statement, but it does not apply to all who suffer from the malady. For the most part the patients whom I saw in private had an intelligence above the average, but many of those seen in hospital practice were quite dull and it was only with great difficulty that I succeeded in getting from them an accurate description of their symptoms. In the same way they are said to be people of mercurial and sensitive disposition. With this statement too, I would agree only in part, as several of my patients were distinctly bovine in nature.

true!

A very interesting article appeared some time ago by E.J. Stieglitz. In this he reported

on the physique of one hundred consecutive cases of migraine. As a result of his enquiry he formed the opinion that these patients suffer from an unusual degree of vasomotor instability, that they have a lowered resistance to fatigue, and many are chronically tired. In my opinion it is true that a few of these patients are always below par, but whether this is post hoc or propter hoc it is very difficult to say. The majority, however, enjoy perfect health and are full of vitality between the attacks. One observation made by this writer was of particular interest to me. He noted that his patients had unusually large pupils in proportion to the exposure to light, and that this dilatation was increased still further with fatigue and was especially marked just prior to an attack of migraine. In 62 per cent of his patients habitual dilatation was noted. This observation I have not been able to confirm nor yet refute from my series of cases. Though no actual measurements were taken the impression made on me was that the pupils varied within normal limits. Stieglitz found the blood pressure below normal in the majority of his patients; a disturbance of the peripheral circula-

tion, as manifested by pallor and cold moist extremities, and a high incidence of paroxysmal tachycardia; all suggestive of some ill-defined defect in the vasomotor system. He believed that the fatiguing effect of glare on the poorly protected retinae contributed to the maintenance of this chronic state of tiredness.

POSITION AND OCCUPATION.

Some observers state that migraine is commoner among the upper classes, but of the truth of this I am not at all convinced. I think it is certainly found more frequently in those with sedentary occupations who work at high pressure and who are subjected to prolonged eye-strain. Macdonald Critchley recently pointed out that loss of acute sight frequently brings a considerable measure of relief in the number and intensity of the attacks, and drew attention to the fact that it is very rare to find migraine amongst the blind.

AGE.

Timme states that practically all types of migraine disappear between the ages of 30 and 50. With this it is quite impossible to agree as at least 50 per cent of my own cases were over 45 when they consulted me. According to another observer, 10 per cent obtain relief naturally before they reach the age of 40; 55 per cent between 40 and 50; 25 per cent between 50 and 60; and the remaining 10 per cent continue to have their headaches after 60. Among my own patients I have been surprised to find how many gave a history of attacks in childhood; this was followed by a long period of complete freedom until the age of 45 or thereabouts, when the symptoms again made their appearance. There is another group, however, in whom the symptoms do not become manifest until they are well over the age of 50.

In very young children headache is not complained of, but there may be a rise in temperature followed by severe vomiting. It is most probable that the cyclical vomiting of children is the same syndrome as migraine. These patients usually suffer from typical migraine after 10 years of age.

In this connection I have been particularly interested to note how seldom children with obvious refractive errors complain of headache.

Of the patients reported on in this series twenty-two had attacks of migraine before the age of 10 years; in sixteen the attacks commenced between 10 and 20 years; in eight they appeared in the third decade; two were first affected in the fourth decade; and two more in the fifth decade. Thus in 76 per cent of the cases the symptoms had made their appearance before the age of twenty years.

SEX INCIDENCE.

Most writers maintain that migraine is more frequently found in women than in men, but there is a very wide variation in the proportion as found by different observers. Fleteau, in 1912, gave the figures as 1.7 to 1.; Reilly, in 1926, 4 to 1.; Laing, in 1927, 4.1 to 1.; and Smith, in 1922, as 2.7 to 1. Allan, writing in 1932, gave it as his opinion that men and women were about equally affected, and that the proportion of women sufferers has been greatly exaggerated. He be-

lieves that in women the symptoms are more severe, and so a larger number of them seek relief.

In my own series the proportion of women to men was 5.2 to 1.; this, however, cannot altogether be taken as a true indication of the sex incidence. It must be remembered that quite a number of my cases were drawn from hospital practice, and it is well known that women always outnumber men in any out-patient department. The explanation is probably that they have more free time in which they can have their ailments attended to.

PERIODICITY.

In a number of patients the attacks occur at regular intervals, appearing every week, fortnight or still further apart. Twenty-nine of my patients gave a definite history of periodicity, but seven of these were liable to have intermediate attacks. In women the symptoms often make their appearance about the time of the monthly period, usually at the beginning, but in a few it is not until the end that the headache develops.

With regard to frequency, eight of the number had attacks at least once a week or more;

twenty-six had them at regular intervals of from one to four weeks; whilst the remaining fourteen had periods of freedom lasting for a month or more.

DURATION.

It is of little or no value to give an average time for the duration of an attack of migraine. Even in the same patient they vary tremendously and depend on many different factors. Twenty-five of my patients counted the length of attack in hours, the commonest being from 6 to 12 hours, whilst in a similar number it lasted for one or more days. The longest time given for any one attack was 5 days, but several suffered from a chronic headache with periods of exacerbation corresponding to the times at which the typical symptoms would have been expected. This state is comparable to the status epilepticus. I have been surprised to find what a difference there is in the recovery which patients make from an attack. A few feel perfectly well immediately after the headache has left them, but with the great majority there is a feeling of utter exhaustion and limpness. This is usually recovered from following a good night's

sleep, but in others it is several days before they feel completely fit.

PRODROMAL PERIOD.

Prior to the actual development of the headache patients often have vague symptoms from which they know that an attack is impending. For example, several of those interrogated complained of feeling extremely tired and sleepy, and one had a fit of yawning. Others felt unusually fit and had a particularly good sleep just before a headache developed. George Elliot, who suffered from this complaint is quoted as saying "I am dangerously well today". Several had an exceptionally large appetite the day before, and there was one woman who always had a strong craving for pickles. In the majority of my patients the headache came on first thing in the morning, and in fact many awakened with one. In others again, it did not develop until some time after rising. In the literature several cases are reported who dreamt of fire and flashing lights before waking with a headache, and the presumption is that fortification signs actually occurred during sleep.

HEADACHE.

It is from this symptom that the disease derives its name, and though the pain, strictly speaking, should be confined to one or other side, in actual practice it is often found to be generalised throughout. In many it starts on one side, but so quickly spreads that its site of origin is soon forgotten. In 84 per cent I found it was a true hemicrania, at least to begin with. The pain may start in any region though it is common to find it above one or other eye. It was variously described as a boring pain, or like a tight band around the head, but in most there was a throbbing element in it. All could readily distinguish it from the ordinary type of headache, so there is obviously something very characteristic about it. In the remaining 16 per cent the headache was generalised from the start, and it is important to remember this fact, otherwise the correct diagnosis might not be made.

I was surprised to find how many suffered from eyeache during an attack. In all 72 per cent made this complaint, and though in the majority the pain was felt in both eyes, in some it was confined to one eye only. It was described as pain on moving the eyes or referred to the back of the orbit.

In some the headache terminated quite suddenly, but in others it was only got rid of after a sound sleep.

MECHANISM OF THE HEADACHE.

Anything which increases the vascular distension will aggravate the headache, and correspondingly, anything which reduces distension will relieve it. Thus, where the pain is felt on both sides of the head, pressure on one common carotid will relieve it on that side, but increase it on the opposite. From this one is fairly safe in deducing that the pain is at least associated with vascular distension.

As a result of observations in long operations on the human brain it is found that the most sensitive areas are close to the vessels on the surface. The dura is less sensitive, and the radiations and grey matter are not sensitive to ordinary stimuli. The peripheral nerves involved are the intracranial branches of the 5th, and it is stated that no headache occurs on that side if the gasserian ganglion has been removed. To account for the long duration of some headaches it has been

suggested that following the dilatation of the vessels œdema occurs around them, and this, by causing localised pressure on the vessels, may keep up the pain. It is possible to conceive of this vasomotor disturbance acting in several different ways. It may, for instance, consist of a localised vasodilatation with a possible accompanying œdema, or, on the other hand, it may be a spasm of the vessels with an ischæmia of the surrounding tissues. Again, it may be a combination of the two, namely, a localised spasm associated with the aura, followed by dilatation accompanied by pain.

Various authors (Hare, Cushing, etc.) have contended that the pain of migraine arises not from cerebral, but from dural arteries. According to this theory the pain is due to perivascular œdema of the dural vessels with pressure on sensory nerves. This would account for relief of pain on ligation of the middle meningeal arteries.

Working with ergotamin tartrate, Pool made the most important observation that in cats the dural vessels respond to the injection of this drug by constriction, an action dissimilar to that

of pial or cerebral vessels. At the same time, it is possible that ergotamin acts directly as a sedative on the sensory nerves which supply intracranial tissues, particularly those nerves which accompany arteries or supply the dura.

VEGETATIVE NERVOUS SYSTEM.

In many cases, when the headache has reached its height, it is accompanied by nausea or actual vomiting. This may relieve the pain, but more often it continues unabated. The vomit at first usually consists of undigested food, but later on it is only bile-stained mucus which is brought up, followed by very distressing dry retching. Profuse salivation and perspiration may accompany the vomiting, or sometimes there is a watery discharge from the nose. In the group of patients investigated nausea followed by vomiting was found in 66 per cent, whilst nausea alone was felt by 22 per cent. This leaves only a small group of 12 per cent who had no symptoms referable to the stomach. These figures are very similar to those given by other writers.

Urticaria and angioneurotic oedema have

been noted by some observers, and 70 per cent of my patients complained of feeling shivery during an attack, symptoms all pointing to a generalised vasomotor upset. In a few, hæmorrhages in the conjunctiva, the retina, or from the nasal mucous membrane have been reported. This disturbance of the autonomic system is further demonstrated in many cases by a feeling of discomfort in the region of the epigastrium, and it has been demonstrated by X-ray examination that normal peristalsis is completely inhibited during an attack. 20 per cent of my patients complained of polyuria, and two of these noted a previous anuria. Cardiac symptoms such as palpitation and pain in the region of the heart were also met with.

MOTOR SYMPTOMS.

Irritative motor symptoms are rare, but when they do occur they are usually in the face and next often in the upper extremities, and consist of localised twitchings. Actual paresis or paralysis of a limb or affecting one side is occasionally met with, and so too is aphasia, or an agraphia may develop. Regarding this aphasia as found in

migraine, there seem to be two distinct types, though they are often present in one patient. The first type is where the person knows perfectly well what he wants to say, but has great difficulty in articulation owing to the paresis of his lingual muscles. In the other, cerebration is slow and confused, and though the power of speech may not be impaired, the patient is unable to put anything into words.

SENSORY SYMPTOMS.

Some disturbance of sensation is frequently experienced during an attack. The most common is a feeling of tingling or numbness in one or other hand which may spread up the arm. A similar sensation may be felt in the lower limbs or down one side. One of the fifty said that during a bad attack the whole of her body went numb; another that all her "inside" felt paralysed. Paresis of the gums or tongue on one or both sides may occur. Altogether, 42 per cent of my cases experienced in varying degrees a sensation of tingling or numbness. When the headache is definitely one-sided, paræsthesia usually occurs on the opposite side of the body, though this is not always the case.

SPECIAL SENSES.

Tinnitus or temporary diminution of hearing may come on during an attack, and there is definite photophobia in most cases. A metallic taste may be noticed in the mouth and hallucinations of smell are not infrequent. As visual disturbances are of such outstanding interest I propose to deal with them under a separate heading.

Giddiness is a symptom which is very seldom mentioned in the ordinary text-books on migraine, but in eight of these fifty patients it was a prominent feature of the attack. Several whose headaches came on first thing in the morning felt giddy even while lying in bed, and everything in the room appeared to go round. In one where the headache was right-sided, the giddiness was much exaggerated if she lay on this side. In two this symptom became so extreme that the patients actually passed into unconsciousness. One who suffered from very severe attacks of headache and giddiness in the early morning wrote that since wearing the glasses constantly her giddiness, sickness, and headaches had completely disappeared. This patient actually had been wearing glasses which were simple spheres before I saw her,

and the only alteration I made was to add a small plus cylinder to each.

TYPES OF MIGRAINE.

Simple Migraine is the commonest type, and consists of periodic attacks of headache together with mild nausea. The attack itself may last for a few hours and is not followed by any sequellæ.

Ophthalmic Migraine, according to most observers, occurs in about 10 per cent of cases. The visual symptoms which characterise this type may appear before, during, or after the headache. As an ophthalmic surgeon it is natural that one should see more cases of ophthalmic migraine than the ordinary physician, but nevertheless, I think that this figure of 10 per cent is much too low. So very often when dealing with these patients it is only after most careful enquiry that one is able to elicit a history of visual symptoms. Thus, in my series, 28 per cent had misty vision in varying degrees up to complete blindness at some time during their attacks; 26 per cent saw zigzags or fortification signs; and another 22 per cent saw other visual phenomena such as moving spots or

stars, wheels, balls of fire, etc. Altogether, 60 per cent had visual symptoms of one kind or another. A few patients are very rich in these phenomena whilst others are equally poor, perhaps seeing only moving spots, and these just occasionally. One of my patients who suffered from severe migraine reported that during the height of an attack she would become more or less unconscious for several minutes during which time she would see people in fantastic dresses, or animals of peculiar types and shapes. The disturbances noted by different patients vary tremendously, but remain more or less the same for each individual, though they may vary in degree in different attacks. A frequent description is that of a brightly coloured spot, large or small and of various shapes. This may be stationary or move about and gradually increase in size until it occupies a part or perhaps the whole of the visual field. Others complain of numerous spots which may be black or multicoloured and which fly about through the affected part of the visual field and are frequently termed "shooting stars". Another common effect is that of a shimmering or wave-like distortion of objects similar to that seen

through aqueous vapour rising from the ground on a hot day. Other patients describe this as being like the reflections seen on the surface of a pond into which a stone has been thrown.

The typical zigzag effect is usually seen to one or other side of the visual field, and consists of an irregular band of light which darts hither and thither always in rapid motion. The fortification or wall figure, on the other hand, moves slowly, but it too is usually seen round the edge of the field. These disturbances invariably present a homonymous distribution. The resulting scotoma may remain small or it may affect the complete half of each field. In a few it spreads and obliterates vision entirely. When these visual disturbances are one-sided the accompanying headache is usually felt in the contralateral side of the head.

Quite often a patient will complain of losing the vision of one or other eye for a short time, but on careful questioning it is generally found that it is one half of the field which has been lost, with the involvement of both eyes.

The teichopsia may last for only a few

seconds though it is usually troublesome for about 30 to 40 minutes. On the other hand, it may last for as long as 2 or 3 hours. Occasionally visual disturbances may persist for many years without the development of any headache. In a few a scotoma or a hemianopia may persist for several weeks or may become permanent. In these latter cases it is just possible that it is not true migraine with which one is dealing, but a 'symptomatic' migraine which has a definite organic basis.

In ophthalmic migraine any of the other motor, sensory, or sympathetic disturbances already described may be associated with the visual symptoms.

Ophthalmoplegic Migraine. This was first described by Gubler in 1860, and again by Moebius in 1864, but it was Charcot in 1890 who described the ophthalmoplegia as a symptom of migraine. The condition has been observed in very young patients, whilst in others it only makes its appearance after the patient has suffered for many years from the simple type. Many writers doubt the identity of the two conditions. For one thing, an hereditary tendency is not nearly so often found in this type as in the more usual variety. Thus in twenty-nine

cases reported in the literature only four gave a history of headache in any other member of the family. Another peculiarity is that in ophthalmoplegic migraine the pain is very often felt behind the affected eye. In practically all cases of this kind which have gone to autopsy a definite organic lesion has been found in the nerves themselves or around that part of the brain-stem from which they emerge. In the majority there is no visible aura comparable to that found in the ophthalmic type. These facts would rather suggest that ophthalmoplegic migraine has an individuality of its own. It is usually the muscles supplied to the 3rd nerve which are affected, and the paralysis may appear immediately following an attack or after an interval of from one to ten days. All the extrinsic muscles supplied by this nerve may be affected or only a few, and in some cases the intrinsic muscles are also involved. In a patient of my own one pupil remained contracted for several weeks after the cessation of the headache. A few cases have been reported in which there was paralysis of the 4th and 6th nerves. Complete restitution of function may occur in a few days, or several months may elapse before this takes place.

Robin states that in his opinion there is usually no relation between the ophthalmoplegic and the ophthalmic types. He believes that the condition is caused by a neuritis involving the nerve-trunks in the sphenoidal fissure, or by a fibroma or fibrochondroma pressing on the nerve. Likewise, a vascular lesion such as an aneurism in the Circle of Willis might act in the same way. The difficulty is in accounting for the few real cases of ophthalmoplegic migraine which occur in patients who have suffered for a long time from the usual variety of the disease. In some of these which have gone to autopsy a flattened oculomotor nerve has been found where it is crossed by the posterior cerebral artery. Thus it is possible that transient pressure on the nerve during an attack may have produced the palsy.

ATYPICAL FORMS OF MIGRAINE.

Occasionally a patient who has previously suffered from the usual type of migraine may lose one or other of the characteristic symptoms, and so the later attacks may present a somewhat atypical picture. The headache may disappear and be

replaced by bouts of abdominal pain associated with nausea and vomiting and often diarrhoea. If the past history is not very carefully taken in such a case the correct diagnosis may well be missed.

Here, presumably, the disturbance has become localised to the vagus centre in the medulla and so upsets the normal functioning of the stomach and alimentary tract.

In the same way psychic disturbances, if they are marked, may help to confuse the diagnosis. Thus, many patients are extremely irritable during an attack, and a few are disoriented as to time and place, and often there is complete amnesia. If, at the same time, there are hallucinations of sight and hearing, it is not difficult to understand how a wrong diagnosis might easily be made.

THEORIES OF CAUSATION.

(1) The central theory of migraine.

Moebius was an advocate of this theory, believing that the symptoms were due to irritation within the cerebral cortex. Other observers, recognising the evidence of cortical irritation, suggested the presence of adhesions between the dura

and cortex. A lack of free circulation of the cerebral spinal fluid, due to blocking of the foramina between the ventricular cavities, has also been cited as a cause. There is, however, very little pathological evidence in support of this, nor have adhesions ever been found between the dura and the cortex at postmortem examination.

(2) Duodenal stasis theory.

McClure and Huntersinger in 1928, as a result of X-ray examination of a number of migraine patients, reported some evidence of duodenal stasis and suggested that, as a result of this, toxins were produced which might cause the attacks. There is little doubt that during an attack of any severity there is very definite interference with normal peristalsis, but this surely is a result of the general upset and not a cause. Again, it is scarcely ever one of the initial symptoms, and usually is only present after an attack is well under way. There is one other difficulty which I have in accepting this theory, namely, that if normal stomach function has ceased and actual sickness is taking place, it is very hard to believe that anything at all is being absorbed into the

blood stream. This is supported by the fact that drugs given by the mouth at this stage have little or no effect.

(3) Toxic Theory.

It is easy in little understood diseases such as migraine to predicate a toxic causation. Numerous writers believe that as a result of intestinal stasis, toxins are accumulated in the body, which having reached a certain degree of concentration, will then produce an attack. It is an essential part of this theory that one must also postulate a localised sensitivity in certain parts of the central nervous system. It seems to me that very little evidence has been brought forward to support this theory and it is largely a matter of surmise. If toxins act at all it is probably in a general way by reducing the patient's vitality rather than being themselves the specific cause.

(4) Hypophysial theory.

This presumes that there is a periodic swelling of the pituitary body which presses on the surrounding structures and so gives rise to many of the symptoms, or that the gland being too large for the sella turcica, is pressed on with a resulting

headache. In eighty-nine cases whose skulls were X-rayed by Balyeat no special abnormality was found. Supporters of this view emphasise the frequency with which migraine attacks are associated with the menses, when, presumably, the gland undergoes physiological enlargement. This explanation, however, does not hold good in male patients, and in many other cases in which the attacks do not cease at the menopause. It is likewise very difficult to see how any pressure exerted by an enlarged pituitary gland could cause many of the visual and sensory symptoms which can only be due to irritation of the cerebral cortex.

(5) Endocrine theory.

A dysfunction of the pituitary gland which would, in its turn, upset the general glandular system, has been suggested as a cause. Since there is often a relief of symptoms during pregnancy and after the menopause, it has been thought that the aetiology of the disease might be pluriglandular in type. If the headache were pituitary in origin one would expect to find other signs of pituitary dysfunction such as osseous, genital, or pigmentary changes. Because attacks of migraine frequently occur at the periods, many writers deduce from this

that it is due to, or associated with some alteration of the internal secretions, but surely a more simple and as good an explanation is just this; that at these times most women feel definitely out of sorts, and should they be liable to any indisposition it is most likely to make itself felt at those particular times. It is frequently said that the attacks cease during pregnancy, and though this is often the case, I had just as many who said that their attacks were more troublesome at this particular time. A great many women feel particularly well while pregnant, and so it is not surprising that the headaches are then less severe.

(6) The allergic theory.

This theory is based on the assumption that the patient becomes specifically sensitive to certain substances and that there is localisation of the sensitivity in the cortex of the brain. The contention is that just as an urticarial wheal may arise anywhere on the skin, so may a similar patch of œdema be produced somewhere in the cerebral cortex, giving rise to the headache, visual symptoms, etc. In support of this theory the hereditary factor is mentioned as being common to both migraine

and the allergic diseases, and likewise the occurrence of an eosinophilia. This latter statement, however, was not corroborated by an investigation carried out by Riley, Soltz and Brickner who found that 4 per cent of eosinophils was the highest number present in their series. Some writers maintain that migraine is definitely an allergic condition, and that it is interchangeable in the linkage with asthma, hay fever, and urticaria. If this is so, one would expect more frequently to get a history of one or other of these diseases from migraine sufferers. Only from 9 per cent of my own patients did I elicit a history of allergy in themselves or their families. Many allergists contend that it is the common foods which are the most frequent offenders, as for instance, milk, wheat, eggs, etc. These foods are consumed daily by everyone and it is difficult to understand why an attack should only be produced occasionally and not every day, and furthermore, how can one account for the definite periodicity so commonly met with in migraine patients? Many physicians believe that an idiosyncrasy to this or that particular food is the sole cause of migraine. With this in

mind I went into the question of food allergy with every patient. Of the fifty, 10 definitely blamed cocoa or chocolate, and several of these also thought that fats in general did not agree with them; 6 others blamed this or that particular food, but in no instance were they so certain about it as those who attributed their attacks to chocolate. Two only, blamed alcohol, but it has been explained to me by an allergist that it is not so much the alcohol itself, but the fact that food taken with alcohol is much more quickly absorbed, and so is less completely broken down. I must point out, however, that though food was mentioned by a few as a causal factor, it was in no case the sole exciting agent, but was only one of several given by the patient.

I fully appreciate the fact that it is quite possible for a food to produce an allergic reaction and yet for the patient never to suspect that his symptoms are due to it. Especially is this the case if it is one of the commoner food-stuffs which is to blame. At the same time, I cannot help feeling that if migraine were the allergic condition which some observers would have us believe, one would more frequently find patients

who could definitely point to this or that particular substance which they knew from experience would engender an attack. George H. Hyslop quotes a case of a patient who would occasionally have a migraine headache several hours after eating pork. He could eat this without trouble when he was in good condition, but was sure to have a headache if he took it when tired or under emotional stress. While on this subject, I would like to mention that several of my patients, after wearing their glasses constantly, could take certain foods with impunity which previously they dared not touch.

(7) Vasomotor theory.

Supporters of this theory maintain that the symptoms of the disorder are the result of a disturbance in the cerebral vessels. According to some it is due to a vasoconstriction producing an anæmia of the part, whilst others think there is a vasodilatation with resulting œdema and congestion. This idea has been elaborated by Curschmann who argues that the distribution of the circulation throughout the body depends on a balance between the vasoconstrictor and vasodilator impulses which are controlled largely by the sympathetic nervous

system. In some persons this balance is unusually delicate and when it is upset in the cerebral vessels the syndrome of migraine occurs. Following the same idea he would argue that when the pulmonary circulation is disturbed an attack of asthma is produced. In Curschmann's opinion many different types of stimuli or irritants may serve to disturb this vasomotor balance, - psychic, visual, toxic, etc. The following facts are all in support of the vasomotor theory.

- (a) Transient nature of the attack.
- (b) No residual disturbances as a rule.
- (c) Vasomotor disturbances in the face and conjunctiva.
- (d) Frequent relief from vasomotor drugs.
- (e) Ophthalmoscopic examination is usually negative, but spasm of the retinal artery has been observed during an attack.
- (f) Pressure on the temporal or carotid artery on the side of the headache will immediately relieve it.
- (g) Throbbing character of the pain.

In those cases where a scotoma or a hemianopia persists for several weeks, or for that matter, has become permanent, it is possible that a definite change has taken place in the corresponding part of the brain as a result of the repeated interference with the circulation. In connection with this vasomotor theory I have been particularly

interested in some of the more recent work done on the cerebral circulation. Cobb, in 1933, demonstrated that the cerebral vessels have vasomotor control, and maintained that there were no end arteries in the brain. Fay, in a study of the cerebral vasculature by X-rays showed that there is an anastomosis between the anterior, middle and posterior cerebral arteries, and also between the vessels of the right and left hemispheres. These investigators also maintain that the capillary bed of the cortex and basal ganglia is one continuous network. If this is so, parts of the brain controlling very different regions of the body are linked up by their vascular supply, and so a disturbance in the cerebral vessels might well cause the multiplicity of signs and symptoms with which one meets in migraine.

It is difficult to see how the visual symptoms could be produced in any way other than by irritation of the occipital cortex, or if not actually the cortex, at least the occipital lobe, giving rise to the fortification spectra and scintillating figures. This irritation of the nerve cells might well be followed by a temporary inhibition of

function causing the scotoma, the retina all the time playing a passive role. Similarly, the tingling felt in the arms, face and tongue is presumably due to irritation of the cells supplying these parts in the sensory area of the brain, and the numbness corresponds to the scotomata. The motor disturbances are likewise of cortical origin, while the gastric and vasomotor symptoms must be due to some upset of the sympathetic nervous system.

EYE STRAIN AS AN ACTIVATING AGENT.

When interviewing my patients I made a special attempt to try and find out what was most likely to induce an attack. As I said before, each one was asked a definite series of questions so that nothing might be missed. Out of my fifty patients 19 blamed travelling, and most of these found sitting with their backs to the engine far more trying than when facing it. Others who were upset by motoring much preferred a front seat to a back one. The only explanation of this which I can offer is that there is more obvious movement of the objects passed when viewed from the side than when seen head on. The cinema was also responsible

for 19. When on this question of the cinema, I am sure that the moving picture seen on a screen is one of the best tests for finding out and bringing to the patient's notice any latent eye strain. This is particularly true of low astigmatic errors. In the same way, strain is quickly felt if there is any appreciable degree of muscle imbalance, and especially so if hyperphoria is present. With 15 patients movement, by which I mean an unusual degree of physical activity, might predispose them to an attack. Worry and excitement I have grouped together, though of course they are totally different conditions; these were given as a causal factor by as many as half of the patients investigated. I think it is true to say that excitement is more a factor in young people, as for instance that of children preparing for a party or a theatre, whereas worry is much more found in adults. Where excitement is given as a cause by an older person it is usually found that it is more a state of apprehension with an element of fear in it, rather than of pleasurable anticipation. Near work, by which is meant continued close application, was given by 22 as likely to bring on a sick headache.

It is a common belief that the headache produced by eye strain is always felt at the time when the eyes are being used, but though this is frequently so, it is certainly not always the case. Very often, from presbyopes who require stronger reading glasses, one will get a history of early morning headache due to prolonged reading the night before.

In quite a number the attacks were brought on by looking at any bright object such as the reflection of the sun's rays from some highly polished part of a motor car. Looking at a much-patterned carpet, wall-paper, or material might be quite sufficient to initiate the train of symptoms in very susceptible individuals. One of my patients was a machinist and always had an attack of migraine after working on striped material. Another told me that she would practically always get an attack whenever she dressed her small son in a particular striped suit. Certain colours may act in a similar way, and this same patient was very much afraid of bright red, as for example, a bed of geraniums. It may be argued that looking at a much-patterned material may induce an attack by suggestion, but I do not

think this is so, otherwise there would be no improvement after the refractive error had been corrected.

Only three blamed noise, and this is rather illuminating when one compares it with the very large number whose attacks are brought on by some irritation of the ocular mechanism.

There are several comments I would like to make on the methods which I employed in the purely ophthalmic part of my investigation. Each patient was first examined by the ophthalmoscope, and also by the slit-lamp if necessary, to make sure that the media were clear, and notes were made on the condition of the retina. A very careful retinoscopy was then done, followed by the subjective examination. If the vision did not come up to 6/6 and this could not be accounted for, or if I was not sure of the axis or strength of the correcting cylinder, or if there was any doubt about the spherical error, I insisted on making a second examination under a cycloplegic. For this purpose, in the majority of cases, homatropin and cocain drops were used, and I made very certain that the ciliary muscle was completely paralysed before making the second examina-

tion. On the question as to whether or not a cycloplegic is necessary, the finding I have come to is this; that in middle-aged patients with relatively large pupils and an appreciable astigmatic error it is not absolutely necessary to use a cycloplegic if sufficient time is taken over the subjective part of the examination to make quite sure of arriving at the correct axis. In younger patients, however, and by this I mean anything up to 30, where the astigmatic error is low, I do think it is absolutely necessary to use complete cycloplegia. In several cases of this type where I first ordered glasses without using drops, I found on a subsequent examination with a cycloplegic, that the axis noted on the first examination had been wrong. Where the correcting cylinder is a low minus one I think it is all the more necessary, as a patient with this error has much more difficulty in appreciating varying positions of the cylinder. In some cases I found it necessary to see the patient a third time before being sure that I had arrived at the correct prescription.

In the subjective part of the examination I always used an astigmatic fan. This, I think, is

very important, and I am amazed that this most useful test is not employed by more ophthalmic surgeons. In my opinion, it is quite impossible for most patients to appreciate any movement of less than 10 degrees with a + 0.25 cylinder, and in the same way they will not appreciate a movement of much less than 20 degrees with a minus cylinder of the same strength. Because of this, if one does subjective testing without the aid of a fan, it is quite possible to be several degrees out in the axis of the correcting cylinder. So very often when doing a retinoscopy one discovers the need of a cylinder in the vertical or the horizontal meridian, but in the majority of these cases it is found at the subjective examination that the correct axis is to one or other side of the 90 or 180 degrees line. In most of these cases the use of a fan will immediately give the surgeon a guide to the true axis.

When on this question of astigmatic fans there is one point which I would like to make. Most of those on the market, and this is certainly true of all the cheaper ones, consist of radiating lines forming a half circle. These are perfectly satisfactory where a plus cylinder lies somewhere

in the vertical meridian, but if the cylinder is a horizontal one they are not so helpful. It is then impossible to tell whether the correcting cylinder should be at 10 degrees, 180 degrees, or even 170 degrees. A slight improvement on this type of fan is the one having two more lines above the horizontal on each side. A still further improvement is to have the complete circle of radiating lines in which case one has a double check, and the clearest line on one side has an equally clear one diametrically opposite. Instead of having single lines it is better to have a thin double line and so when the astigmatic error is a low one, the patient can easily pick out a small group of clear double lines, while the others appear indistinct. In a few of the more expensive fans the lines are too far apart, and on account of this lose much of their accuracy. In my opinion, 10 degrees is about the correct distance. I have had a special fan made for me incorporating all these features, that is, a complete circle of double lines at 10 degree intervals, and I find it most satisfactory in every way.

While on this subject I would like to say a little about the cross cylinder. Like the astigmatic fan, I find this a most helpful instrument

and use it on every case. Its greatest value, I find, is in verifying the axis of the correcting cylinder. When used as a means of ascertaining the strength of the cylinder I think it is often misleading. In many cases, had I relied on it alone, I would have ordered a much stronger cylinder than the patient would have comfortably worn. It is puzzling why this should be so with some patients, when with others it gives an exact indication as to the strength of the cylinder.

I have come to the conclusion that to give an accurate prescription it is necessary to do a retinoscopy under the most favourable conditions and to follow this by a subjective examination at which both an astigmatic fan and a cross cylinder are used. Very occasionally, when the cylindrical error is a small one, the indications got from the fan are somewhat confusing, in which case it may be necessary to rely on the cross cylinder alone, but I am never quite happy when this is so. In the great majority of cases the axis of the correcting cylinder as determined by the astigmatic fan and the cross cylinder will be the same, though it may take a certain amount of time and patience to get this quite right.

In the few cases where the findings do not correspond, they will usually be found to do so after using a cycloplegic.

I make no apology for what might appear to be a digression from the main subject into a discussion on refraction. The very essence of the treatment of migraine from an ophthalmic surgeon's point of view is the absolutely accurate prescription of glasses, and this can only be done with good instruments used in the best possible way.

The question of muscle imbalance is an important one in the treatment of this condition. Quite a large number of my patients had this in one form or another. Hyperphoria undoubtedly gives rise to most trouble, and after this I would place esophoria in young patients and exophoria in old. I have always followed the rule of correcting practically the whole of the vertical imbalance, and the great majority of patients will quickly accept this. In a few, however, and especially in those where one is incorporating a prism for the first time, I have found it wise to undercorrect at the beginning, and later on work up to the full correction. In ten of my fifty I ordered prisms to correct a vertical imbalance. This makes a total of 20 per cent; a

percentage certainly very much higher than that found in the general run of patients. With an exophoria or an esophoria which I thought was giving rise to strain I would, as a general rule, correct half or rather less than half of the total amount.

In quite a number of cases migraine first manifests itself about the presbyopic age. This often coincides with the menopause and so is ascribed to the change of life, but in many of these cases the symptoms will disappear when correct reading glasses are worn.

TYPE OF REFRACTION.

Practically every variety of refractive error was met with, and in a large number the error was quite small. It is because many of these patients see so well that neither they nor their doctors think of suggesting an eye examination.

Out of the fifty cases in the series 26 were found to be suffering from compound hypermetropic astigmatism; 14 from simple hypermetropic astigmatism, and only one from simple hypermetropia. There were 7 with compound myopic astigmatism, and 2 with mixed astigmatism. It is a well recognised thing that compound hypermetropic astigmatism is the commonest refractive error found as a cause of ordi-

nary headache, and so one is not surprised to find this type predominating in migraine patients. I have long been of the opinion that hypermetropic astigmatism against the rule was more liable to give rise to trouble than when it was with the rule, but in this series I found that the numbers against were equalled by the numbers with the rule.

RESULTS.

In estimating the figures of those who have been cured or helped by treatment it is difficult, when dealing with a disease like migraine, to state categorically that so many have been cured and so many improved, etc. All the patients dealt with in this series have now had their glasses for some five to ten months. In estimating the number who have been cured I have put down only those whose attacks have ceased altogether since wearing their glasses. I should qualify this last statement by saying that in a few cases there were several slight attacks immediately after wearing the new correction, but once the eyes had completely settled down there was no further trouble. These naturally I have included among the cured. In the next group I have placed those who have been much improved, and in coming to

this conclusion I have compared the frequency and severity of the attacks before and after wearing the glasses prescribed. In many cases there was a decided drop in the number of attacks, and in still more the headaches have been less severe, shorter in duration, and with little or no sickness. The third group I have described as being slightly improved, and in it I have included those patients who have been certainly better after wearing the glasses, but in whom there has not been that decided improvement found in those of the second group. In the fourth and last group I have included those who were no better.

In group No.1. there were three men and twenty-one women, making altogether a total of 48 per cent whom one could describe as being cured. Of these only two were hospital patients. When I refer to these people as being cured I mean that they will be free from attacks as long as their present glasses accurately correct their refractive error. Should this change or should their presbyopia increase, it is quite possible that there may be a recurrence of the headaches, when a fresh examination will be necessary.

Group No.2. contains three men and eight women who reported much improvement, making a total of 22 per cent. By way of showing exactly what I mean by much improvement I will quote several statements made by these patients. One man who was getting severe attacks lasting from three to four days and recurring about every three weeks reported "One slight attack in three months". Another male patient who suffered from severe attacks every fortnight reported that "During the first two months after wearing the glasses the attacks were less frequent and very much less severe, and during the next two months they ceased almost entirely". In his case the sickness completely disappeared and the headaches were of very brief duration, whereas previously they had persisted throughout the day. A reply from a woman patient who was having an attack once a week which usually lasted twenty-four hours, was to the effect that after wearing the glasses the attacks were much less frequent, much less severe, and there was no sickness. Another woman who was having them every fourteen days and who was developing a status migrainous, reported that her attacks were now "Very much less severe, less frequent, and

the sickness has ceased". One young girl who obviously did not wear her glasses constantly, said that her attacks had lessened and were not so severe, but that they recurred if she left off her glasses.

In group No.2. two men and ten women, making 24 per cent, were slightly improved. The patients included in this group all said that their attacks had been lessened in some way. For instance, one reported a lessening in intensity, but not in number. Another girl who had previously been sick every day said that this had been reduced to not more than once a week. One of the men whose attacks had been reduced in severity, though not in number, reported that the polyuria from which he suffered had ceased.

Group No.4. is made up of three women in whom there was no improvement; two of these had been ill and had never worn the glasses, and the third refused to come back for an examination under a cycloplegic.

Of these fifty patients, 9 were examined in hospital and a still larger number at a National Ophthalmic Treatment Board Clinic. These patients, I feel, have not been prescribed for so accurately as those seen in one's own consulting room. The chief reason for this is that when working in hospital or

at a clinic one has not the necessary amount of time to devote to them, and also the instruments at one's disposal are not so good or accurate as one's own. Another factor is that hospital patients as a rule have not the same amount of intelligence as the better educated people one sees privately, and so they give the surgeon much less help at the subjective part of the examination. This is particularly the case when dealing with low astigmatic errors where one is bound to rely on the patient's answers when finding out the axis of the correcting cylinder.

Several of my patients declined to wear the glasses constantly and only used them for near work, and though they were much relieved of their symptoms by doing so, they still suffered from periodic attacks.

As a result of my experience in dealing with these migraine patients I have come to the conclusion that it is absolutely necessary for them to wear their correction all the time. This conclusion has rather been forced on me by seeing the very marked improvement in patients who had only a very low refractive error.

Another most important point, and one which is frequently neglected, is to make sure that the glasses fit very accurately. It is surprising how often one sees patients whose glasses are obviously tilted to one or other side. With these migraine sufferers I always insist that they go back to the opticians about every three months to have their frames readjusted. I do not want to labour this point unnecessarily, but several of my patients wrote reporting no improvement after wearing the glasses I prescribed. On seeing them again I discovered that they had a poorly fitting frame, and when this had been carefully readjusted there was an immediate amelioration of their symptoms.

CONCLUSIONS.

No theory has yet been advanced which would account for all the symptoms found in this malady. To begin with, one is dealing with a patient who, to all intents and purposes, is perfectly normal apart from the fact that every so often he is liable to be prostrated with a blinding headache. The disease is a functional one and bears a marked resemblance to asthma and epilepsy, and just as some are born with a predisposition to suffer from one or other of these

diseases, so the migraine patient has a constitution predisposing him to suffer from sick-headaches, and this is inherited by the large majority. Over sixty years ago Liveing described migraine as a "nerve-storm", and though this has no precise scientific meaning, it is a useful term, and with all our increase in knowledge it is doubtful if we can describe the pathogenesis of the condition any more accurately today. Of all the theories advanced I think the vasomotor one fits best the picture of signs and symptoms. According to this, the balance between vasoconstriction and vasodilatation is very delicate in some people, and when this is upset in the cerebral vessels that part of the brain so supplied is irritated, and an attack of migraine ensues. This balance is easily upset and so an attack may be brought on by worry, excitement, exhaustion, or ingestion of some food to which the patient has an idiosyncrasy, but of all these accessory causes eye strain is, in my opinion, by far and away the most common. Here we have a source of irritation and loss of nervous energy going on day in, day out.

REFERENCES

- ALLAN, W. : The sex ratio in Migraine. Arch. Neurol. and Psychiat. xxvll, 1932, 1436-1440.
- ANHAULT & WEPFER, : Quoted by G.H.B. Black. Med. Jl. Australia, 1934.
- ARETAEUS OF CAPPADOCIA. : Heterocrania. In: Corpus Medicorum Graecorum. Ed. Hude. 1923.
- BALYEAT, R.M.: Migraine: Diagnosis and Treatment. Philadelphia, 1933.
- BERNARD, C. : Influence of section of the Sympathetic. In: Léçon sur la physiologie et la pathologie du système nerveux. Paris, 1858.
- BRAY, G.W. : Recent Advances in Allergy. Philadelphia.
- CHARCOT, J.M.: Quoted by C. Gerini, 1927.
- COBB, Stanley: Anatomy and Physiology of the Cerebral Circulation. 1933.
- CRITCHLEY, M.: Mechanism and Treatment of Migraine. Practitioner, cxxxiii, 1934, 54-61.
- CURSCHMANN, H.: Ueber Kindermigrane. Munchen. Med. 1922.
- CUSHING, H. : In: Keen, W.W. : Surgery: Its Principles and Practice. Philadel. 1908. Vol.3.
- FAY, Temple, : The Cerebral Vasculature: Preliminary Report of Study by Means of Roentgen Ray. J.A.M.A. 84: June 6th, 1925.
- FLATEAU, E. : Die Migrane. Berlin, Springer, 1912.
- GALEN, : De compositione medicamentorum secundum locos. In: Opera omnia. Ed. Kuhn.
- GOWERS, W.R. : The Borderline of Epilepsy. London 1907. Also, Brit. Med. Jl. Dec.1906 & June 1909.
- GÜBLER. : Quoted by H.A. Riley in Migraine. Bull. Neurol. Institute, New York, 1932.
- HARE, F. : The Mechanism of Pain in Migraine. Med. Press and Circular, June 1905.
- HYSLOP, G.H. : Migraine: Suggestions for its Treatment. Med. Clin. North America, 1934, xviii.
- LAING, G.H. : Migraine Types in relation to Gastro-intestinal Tract. Med. Clin. North America, xi, 49-54, 1927.
- LEPOIS, C. : Selectiorum Observationum. De Hemisrania. pp. 67-77. 1714.
- LIVEING, E. : On Megrim, Sick-headache and some Allied Disorders. London, 1873.
- MCCLURE, C.W. & HUNTERSINGER, M.E. : Paroxysmal Headache. II. Observations on the Etiology, Symptomatology and Treatment of the Migrainous State. New Eng. Jour. Med. cxcix, 1928.

REFERENCES.

- MOEBIUS, P.J. : Die Migrane, Wien, Holder, 1894. In: Spec. Path. u. Therap. (Nothnagel) 1899.
- PASKIND, H.A. : Relationship of Migraine, Epilepsy and some other neuropsychiatric disorders. Arch. Neurol. and Psychiat. 1934, xxxii.
- POOL, J.L. : With Nason, G.I. Cerebral Circulation, xxxv. Comparative effect of Ergotamin Tartrate on the arteries of the Pia, Dura and Skin of cats. Arch. Neurol. and Psychiat. 33. Feb. 1935.
- REILLY, T.F. : Headache: its causes and Treatment. Philadelphia, 1926.
- RILEY, SOLTZ, & BRICKNER, : Unusual types of Migraine. Bull. Neurol. Inst. New York, 1935. ix.
- ROBIN, I.G. : The Eye in Migraine. Guys Hosp. Gaz. 1934. xlviii. 321-322.
- SMITH, J.C. : Om Migraenens Arvelighedsforhold. Binliot f. Laeger, cxiv, 1922.
- STIEGLITZ, E.J. : Migraine physique. Amer. Jl. Med. Sc. 1935. clxxxiv, 359-364.
- STRUMPELL, G.A. : Lehrbuch de speciellen Pathologie und Therapie der inneren Krankheiten, Leipzig, 1926.
- TIMME, W. : Oxford System of Medicine. p. 654a. Nelson's Loose-Leaf Living Medicine, T. Nelson & Sons, New York. VI, 654A-654H.