ON MEGRIM - A DISCUSSION.

In his paper "On subjective visual sensations", Sir W. Gowers states that:- "frequent as is migraine and frequent as are the associated visual spectra, the ability to observe such quickly changing phenomena is rare. Still more rare is the ability to depict them and to describe them with needful fulness and precision. But the example afforded by Dr. Airy should not be lost on the many members of our profession who are similar sufferers. We may learn much regarding the action of the cerebral centres from such observations ..... I hope that from the many who suffer and have the ability to supply the record, some facts may be furnished which will enable us better to understand that which is now mysterious. We cannot tell what additions to knowledge may result from facts that seem simply curious." I, therefore, do not hesitate to say that no apology is needed, even although little that is original may be added to what we already know of this interesting disease, for putting on record the various phenomena associated with the attacks of one who has suffered from this trouble for many years. I may at least be able to corroborate the statements of Airy, Liveing,

Latham and others, and point out what has in my own case been the most satisfactory mode of treatment after a long and exhaustive trial of the many remedies at our disposal. I have not been able to collect a large series of cases, apart from my own, but in any case it is difficult to extract that which may be looked upon as a true record of the various symptoms which make their appearance during the course of a moderately severe attack of migraine:- such, for example, as the colours seen in the visual spectra - the relation of the colours to one another - the sensory aura met with in the arm and other parts of the body, or the confusion of mind so often present in many during the height of the attack. I shall accordingly chiefly confine myself to a record of the various ways in which I am myself affected, rather than to what one may learn from others, who are not members of our profession.

I propose, therefore, to give a synopsis of the disease - its early history and its association with headaches of other types, its aetiology, symptoms, diagnosis and lines along which treatment should be carried out. At the same time I propose to enter into a discussion of my own case, and point out in what directions I correspond with the standard types recorded or in what way I may differ materially from them.

History.

The earliest authenticated record of a case of headache is perhaps that mentioned in the second book of Kings, IV., 19, where one finds the following words:- "And he went unto his father and said 'My head, my head'": -- but it was not until quite modern times that any classification had been attempted. Dr. Smart of Edinburgh, about four years ago, in a clinical lecture on the subject, gave the following classification:-

Headache due to -

I. Organic Disease of
- (a) Brain, e.g. Tumour.
- (b) Cerebral Membranes.
- (c) Bones of the skull.
- (d) Scalp, i.e. Rheumatism.

II. Toxaemias
- (1) Fevers.
- (2) Anaemias.
- (3) Uraemia.
- (4) Constipation.
- (5) Gout.
- (6) Dyspepsia.
- (7) Foul Air.

III. Increased arterial Tension e.g. Nephrites.

IV. Venous congestion —
- Heart disease.
- Asthma.

V. Reflex causes (1) Nasal adenoids.
(2) Ocular errors of refraction.
(3) Dental irritation.

VI. Functional nervous disorders -
(1) Mental overstrain.
(2) Debility.
(3) Hysteria.
(4) Neurasthenia.
(5) Neuralgia.
(6) Migraine.

It is with the last (No. 6) that I wish especially to deal, but I will have something to say as to whether it may not often be induced by the circulation of some toxine in the blood, thereby irritating the central nerve centres and inducing the onset of an attack.

The word "megrim" is the shortened form of Hemicrania and may be defined as that of spontaneous attacks or fits of pain in the head, limited to one side, though often not distinctly bounded. Migraine was known to the older authors; Bartholin in 1684 describes a case of "Hemicrania Periodica" but on closer investigation this seems to have been a case of supra-orbital neuralgia occurring at fixed times of the day. There was amongst the older authors a

certain degree of confusion regarding this disease with that of supra-orbital neuralgia. During the years 1870-78, Lebert, Stokes, Clifford, Allbutt and other pathologists regarded the disease as one of genital neuroses. They localized the pains in the frontal nerve endings and divided the disease in a somewhat arbitrary manner into varieties which had as their existing causes certain organs of the body. Thus, for example, migraine stomacale, uterine plethorique, &c. Later on, Piorry placed the seat of the disease in the nerves of the Iris. Romberg, on the other hand, believed the disease to be due to an Hyperæsthesia of the brain or central pain, and thus, once and for all, sharply discriminated the disease from a peripheral neuralgia. He accordingly named it Neuralgia Cerebralis. It was sometime after this that Du Bois Reymond established the theory that migraine was caused by a unilateral tetanus of the vessels of the head or in the district supplied by the cervical sympathetic. Moellendorf, however, could not agree with this view and sought to show that migraine depended upon a unilateral relaxation of the vessels of the head as a result of loss of energy of the vaso-motor nerves. Zeimsen, Brunner,

Berger, Holst and others, however, took up a view intermediate between these, in that they said neither of these views explained all cases,

They stated that many were certainly vaso-motor in origin, but that this class must be divided into "sympathico and angio-paralytic types". Again, Haig and others have attempted to associate the attacks with a disturbed uric acid output, whilst Clauss regarded the disease as being due to Arthritism, nervous diathesis, chlorosis and anæmia. C. E. Herter attributed migraine to a toxæmic condition (probably albuminous) being absorbed from the gastro-intestinal tract. Dr. Edward Liveing, in his excellent book on Migraine and Sick-Headache in 1873, pointed out that the disease might be attributed to a nerve-storm and seeks to closely link the disease with other neuroses. Murchison (Croonian Lects. p. 109) in 1874 agreed with those authors who regarded certain cases of megrim of toxic origin being symptomatic of gout and of some other disorders.

In my own case I think I may claim to a certain degree of rheumatic element, but a discussion of the various theories regarding megrim will be gone into at a later date. Having thus given a short resumé of the history of the disease, I shall now confine myself to a discussion of the etiology of the condition.

Etiology.- Migraine seldom starts after the age of 30. The disease usually makes its appearance about the period of the second dentition if the disease is hereditary, and if not so disposed, then the onset is delayed until about the 14th-20th years. (Tissot, Traité des Nerfs, p. 353-4). Liveing (On Megrim, p. 24) also states that the last determining influence is early adult life, when the strain of business and work has occasioned the onset in those who had previously escaped. In my own case, my earliest recollections of the disease date back to about my seventh year. There is undoubtedly a history of heredity of which I shall speak later. I can thus bear out what has been said about the disease starting early in those in whom there is some hereditary taint. Most authors are agreed that the disease usually abates before the age of 55, and that it is practically unknown in old age. Indeed Liveing (p. 26) states that a maximum severity is often attained by the age of 30. In my own case the severity of the attacks and the frequency of their recurrence appeared to me to be at their greatest about six years ago, when I was 22 years of age, but how far this may have been due to the strained life of a student I shall again refer. In women the disease is often

more frequent during the catamenial period and shows a marked tendency to abate with the menopause. Thus it may be taken that the disease declines in both sexes before the onset of old age. There is no question but that in the large majority of cases there is a distinct history of heredity. Liveing (p. 28) regards this as a feature of the disease which helps to link it to the natural family of neuroses. On enquiry I find that from boyhood my father suffered from "biliousness" with occasionally partial blindness and luminous zig-zags seen before the eye. There was no history of headache on my mother's side. My two sisters do not suffer from the disease. My father's brother also suffered from biliousness, but no other history can be made out. There is no record of insanity or epilepsy in the family, but it is interesting to note that the disease has been handed on from father to son - the female side not suffering. That hereditary transformations do occur, such as insanity or epilepsy in parents and migraine in son or vice versa, has been clearly pointed out by Liveing (p. 31). He also mentions a case where the mother was migrainous, and who had a child who suffered from Chorea, and states that this was probably from the father's side, who was rheumatic. I should like this to be noted in what I shall presently

have to say in the relation of migraine to rheumatism.

As a general rule hereditary transformation does not occur but the disease is handed on in the same form - if father suffers from blind headache, so also the son. In my own case it was clear that my father suffered from blind headache, but I, myself, suffer from all the four varieties or combinations of these, described by Liveing. Thus it does not seem unreasonable to state that although hereditary transformation does not usually occur, yet it may certainly resort to a severer type of the same disease in the offspring. Rhomberg, Labarraque, Lebert and others are of the opinion that the disease is slightly more prone in women, but that this is not due to the uterine influence except in so far as it acts as an exciting cause. In most cases a history of hereditary was found. The disease is paroxysmal and intermittent in its character, leaving the patient in good health during the intervals of attacks. Tissot (Traité des Nerfs, p. 386) states that the affection follows a nearly uniform course in each individual. This, I cannot agree with, for sometimes I suffer from an attack associated with visual and sensory disturbances, whilst at other times I may have an attack constituted by sensory disturbances alone. The attack

usually passes off gradually. Tissot (Traité des Nerfs, p. 385) says that in one of his cases the paroxysm was indefinitely prolonged, unless vomiting occurred. I can partly agree with Tissot here, for I have often noticed that I have had a recurrence of the attack in a short time if the previous attack had taken place without vomiting, and if vomiting did not occur during the second attack, a third often occurred about a week later. But I have still further noticed that one bad attack induces another, so perhaps similarly a slight attack may induce a fresh one. I can clearly recall to mind as a boy how I used to dread an attack, because it meant usually a repetition of the process at a near date. I would perhaps be clear for four months from any attack whatsoever, but on my being incapacitated by one attack, I was sure to be laid up with another before a month had passed. This is quite contrary to the experience of Liveing (p. 40), who records a case where the patient did not wish for long intervals of attack, because then the attacks were much worse. I cannot say that in my experience length of time between attacks has made them more severe. Tissot (pp. 384-85) says that true migraines which recur oftener than three times a month or less than four times a year are rare.

Referring to myself again, I find that I may have less than four attacks one year and considerably more than double that number during another.

As Liveing states, the exciting causes largely depend on the idiosyncrasy of the patient and are chiefly those of gastric disorder, catamenial period in women, and emotional disturbance. Gastric disturbance is the commonest cause attributed by the public, - "An attack of the bile". This is much over-exaggerated, although there is no doubt that gastric disturbance can and does act as an exciting cause. Du Bois Reymond noticed a history of constipation in his cases. Regarding myself, I certainly have noticed that by careful attention to the bowels, attacks have been lessened in number, so much so that I never permit myself to become to any degree constipated. I have never noticed that food or heavy meals has produced an attack - the attack comes on quite apart from meals and meal hours, except in one case, viz., the eating of shellfish, especially lobster, but I shall refer to this under sensory impressions, to which I think it belongs. The catamenial period is also said to be an exciting cause and cases have been recorded where the first attack started with the commencement of the menses and recurred regularly for many years during the occurrences of

the 'courses'. Fatigue also is an exciting cause. I remember having a very severe attack after a heavy cycle run of 40 miles against the wind. Mental emotion is a frequent excitement. I used to be in a terror during the few days of professional examinations lest I should be incapacitated on one of the days. I was fortunate in most cases to escape, but I usually had severe attacks after the exams were over - the excitement seemed to keep me going, but the rebound came afterwards, and I seldom escaped. During the six weeks of the final professional examination, I had no less than 14 attacks of varying severity.

Sleeping and waking appear to have some effect (Liveing, p. 52). Dr. Airy remarked that when he awoke with it, he had dreamt of the Zig-Zags and awoke with the headache. This has never occurred to me; on every occasion on which I have awakened with the attack on me, I have had no dreams of light, but on waking, the first thing I noticed in each case has been that my tongue was numb and tingling and that I had disorders of speech. The light effects subsequently occurred followed by vomiting and a gradual cessation of the symptoms. Liveing (p. 54) mentions sensory impressions as an exciting cause, such as glaring lights, loud noises, strong odours, strain on the eyes,

late reading, &c.

In talking of gastric disturbances I have mentioned that lobster brought on an attack. I have noticed on eating lobster or crab that an attack is at once brought on, but this is not all. When in my first professional I had to dissect a cray-fish in the class of Zoology, then a very severe attack of migraine came on. This I would have thought as possibly a mere accident, had I not had a present given me of a lobster a few months ago and after looking at it as brought to me, an attack of migraine started. I, therefore, am led to think that this is a case where the sensory impression of the smell produced the attack, and that the mere eating of the lobster would have had no effect.

I may here refer to my patient Mrs. T., age 55, who has suffered from megrim all her life. She put her attacks down to biliousness, but on enquiry she also told me, that when owing to her trade (fish curing) she had to deal with certain oils, the smell of them used to sicken her and produce an attack. Here again I think the sensory impression of the smell acted as the primary excitent in one who was prone to the disease.

Liveing states (p. 55) that the disorder of sight is the first in the series of phenomena, i.e. that the

part of the sensorium concerned in sight is the point of departure for the nerve storm, and hence it need not create surprise that in cases like those of Mr. Piorry and Dr. Airy, for example, where visual phenomena were highly developed, anything which tried the sight should (have determined a paroxysm. I must again disagree with Liveing in stating that the point of departure for the nerve storm is that portion of (the sensorium concerned in sight. I am frequently attacked with migraine beginning with the sensory aura in the hand or arm, or throat mouth or tongue, and not until 10 minutes to ¾ hour afterwards do the visual spectra make their appearance. I think this fact interesting, in view of the theory of nerve storms. Liveing had not met with any who ~~have~~ attributed their sufferings to change of season. I agree with Tissot, who believes contrary to this. For my own part, I am comparatively clear of attacks during the summer months. It is the late winter and early spring in which I am most prone to visitations. Three years ago, after being a year in hospital as house surgeon, I went abroad as surgeon to a ship. During my whole 8 months in the sunshine and warm weather of the East I had not the least semblance of an attack, but I had not returned to this country long before I had an attack of Rheumatism in my right shoulder which was followed by a severe attack of

migraine on the right side. This was followed by another and yet other two attacks before the summer weather again came in, and I was once more clear until the next winter. I have noticed that damp murky days - rheumatic days - are those that I fear most, and this is again another point in favour of Rheumatism as an exciting and prevailing cause.

Accessory causes, such as prolonged lactation, diarrhœa, leucorrhœa, want of out-door exercise, have all been set down as having an effect on the frequency of the attacks. Sir Lauder Brunton goes so far as to suggest carious teeth as a cause. I was called in a few days ago to see a child, 12 years of age, ~~in general practice~~, who had ~~has~~ a fall two years previously with a resulting consussion. Since then she had been ~~the~~ subject to headaches. There was no hereditary manifestations on either side of the family, unless a history of rheumatism in the mother. I found her suffering from an attack of migraine of the fourth kind, having had the visual spectra and tingling in the right arm and hand, followed by vomiting. I think this interesting, in so far as it may suggest the question whether "accident" may not sometimes determine the onset of migraine per se, or whether the accident merely brought out the trouble in one who had inherited

a rheumatic tendency. I shall speak of this case later.

Having thus mentioned the chief causes of the disease, I will proceed to discuss the symptoms, but before doing so, will give a resumé of the chief outstanding symptoms of the four types of migraine described by Liveing. Throughout I shall follow closely his writings, pointing out where I differ from him and at the same time adding anything that may appear to be of general interest. I want to draw particular attention to a fact that seems not to have been hitherto noticed, viz., the presence of very early premonitary signs before the onset of an attack of migraine - considerably earlier than those of the visual spectra or sensory aura. The importance of this observation lying in the possibility of arresting or cutting short an attack by treatment, which otherwise would resist any form of medical administration. I shall both refer to this subject under symptoms and again under the treatment of the disease.

The exciting causes of migraine are thus:- (1) Heredity, (2) Rheumatism, (3) Gastric disturbance, (4) Fatigue, (5) Mental emotion, (6) Catamenial period, (7) Sensory impressions of taste, sight and smell, and lastly perhaps Accident.

Liveing divides the attacks of migraine into four great phases:- (1) ordinary simple hemicrania, (2) Blind-headache, (3) Sick-headache, and lastly (4) a severe type where sensory disturbances occur.

I. In (1) the ordinary simple hemicrania, the patient on waking is conscious of a general feeling of disorder and slight pain in the region of one or other temple. The pain does not usually overstep the middle-line. It is culminating in its character and reaches its fastigium about mid-day and passes off towards evening. So long as the patient is at rest the pain is bearable, but it is aggravated by anything increasing the blood pressure within the head. It responds to each beat of the temporal artery. There is marked pallor of the countenance, the eye on the affected side appears sunken, small and reddened. As the termination of the attack approaches, the ear of the affected side is reddened. The onset of sleep usually ends the attack. The scalp frequently remains tender at one spot the following morning.

II. Sick-headache. These may come on at various times of the day with frontal headache, which gradually increases until the pain becomes most intense. This is accompanied by an intolerable sense of nausea and sooner or later vomiting sets in. There is now a good

deal of depression and collapse of body and mind rather like the condition brought about by sea-sickness. Sometimes vomiting terminates the attack, whilst in others it affords no relief. The vomiting may be more or less in severity; at first the vomited matter consists of any food that may be in the stomach, afterwards it consists of mucous variously discoloured by biliary matters, and then bile itself, to be followed by simple retching. From the onset of the attack the appearance and expression of the patient is greatly altered. There is a blankness of mind and a waxy pallor with lustre-less eyes. Towards the end of the paroxysm, vomiting ceases and the patient falls into a deep sleep and awakes as a rule feeling refreshed, the headache is lessened, and by next morning has passed away.

Sub-class - where no headache. Sick-giddiness.

III. Blind Headache.- In this type there are many features which are common to it and the previous two manifestations. Thus a history of heredity is usually noted. It commonly commences about the age of puberty and occurs at regular intervals thereafter. After some exciting primary cause, such as emotion, gastric disorder and so forth, disorders of vision are noticed, there is absence of discernment over some part [of

the field of view, giving the sensation of blankness. This is often followed by some spectral appearances replacing the former blankness. These usually consist of intersecting zig-zags, luminous and in rapid motion. After these, eye symptoms begin to subside, which they usually do in 5-15 minutes, the headache comes on, which has the same bi-lateral character as before, and is attended by nausea and even vomiting, and sometimes ~~some~~ mental disturbance as well. Vomiting may terminate the paroxysm, but usually the patent falls into a refreshing sleep and next day is comparatively speaking well again.

IV. Fourth condition or that in which disordered sensibility occurs.

These cases do not appear to occur quite so frequently as do the other three types. The condition indicates a wider affection of the brain. In this class the sense of touch is largely disordered, especially in the arm, fingers, lips and tongue, sometimes impairing the faculty of expression and often causing disorders of speech and mental confusion. The attack usually commences perhaps after some exciting cause, with a feeling of numbness and also sometimes tingling in the arm of affected side. This may spread up to the face, lips and tongue. After this,

the headache comes on and may be followed by nausea and vomiting, and again sleep may terminate the paroxysm. These disturbances of sensation may also be accompanied by a state of mental confusion and disorders of speech, and in addition eye symptoms may also make their appearance. Thus type IV. may be composed only of disordered sensation but may combine all four conditions in varying degree.

Having thus pointed out the four leading types recorded by Liveing, I may describe the various ways in which I am myself affected before I venture to discuss the symptoms seriatim. I will not, however, begin with type I., but will take the order in which I am most frequently affected. A detailed description of the eye symptoms will be given subsequently.

The most common manner in which I am affected is the fourth of the above types. I referred under etiology to the presence of the very early premonitions. These were brought under my notice for the most part quite accidentally and I will relate in what way under each type of attack. I do not quite follow the exact lines laid down by Liveing in his classification in any of the attacks. I will illustrate this

by reference to an attack from which I suffered about six months ago. I had just had breakfast and sat down by the fireside for a few minutes, after which I went to the piano to play some tune, which I usually do for a little while previous to going out to work. I may mention that I play largely "from ear" and consequently had no music before me. After playing a few bars I found that my right hand refused to take a run, there seemed to be some loss of the suppleness of my fingers and I got no further on in the piece. I tried another, which was a 'popular air' and finely rooted in my mind, but again broke down - I knew I was in for an attack of migraine. I had no medicine with me at the time and took nothing. Two hours to two hours and a half later a small spot, bright in character, about the size of a pin head, appeared in the outer and upper part of the combined field - seeming to be on the upper and outer part of the right eye. This spot started just to the right of the fixing point. It increased in size and assumed the character of luminous zig-zags, which started from a little to the right of the fixing point and danced out of the field of vision. The zig-zag spot became more and more oval (it was observed) and there was a space near the fixing point of the eye where the bright zig-zag was not complete. The zig-zag was highly

motile and after dancing out of the field of vision, its place was taken by another and the same process repeated again and again, often ending in a dancing and whirling bright spot of light from which many sparks seem to fly out. These zig-zags are distinctly luminous, being of that peculiar bright almost indescribable colour of an electric spark as it passes from the screw to the hammer of a Rhumkorf's coil in an electric battery. At this time there was distinct blindness. It is impossible to see objects on the outer side of the zig-zags, but the area of blindness is limited by the zig-zags. On closing the eye the zig-zags are still present and it is impossible to see distinctly with the right eye. The conjunctiva of the right eye at this stage becomes red and angry-looking. The pupil is also contracted, though not out of proportion with the contraction of the left pupil. The face would be seen to be pale and the eye sunken (in some cases the face may be flushed). At this time too, the mouth begins to feel dry and parched and often the secretion of saliva is in marked abeyance. After the eye symptoms have lasted about 20 minutes, and whilst they are still going on, a feeling of tingling comes on in the

right thumb and forefinger accompanied by a degree of numbness. The tingling is like the pins and needles caused by pressing the ulnar nerve as it passes in relation to the elbow joint. This feeling of numbness and tingling gradually passes up the right arm in a band which feels to be about four inches long. As it passes up it is accompanied often by an indescribable feeling of tightness - the vessels seem to be pressed so tight that no blood can pass. This feeling of numbness and tingling ~~with me~~ does not seem to pass beyond the insertion of the deltoid muscle. At this time a cold shiver comes on and the bowels feel as if they were loose and digestion appears to be at a standstill. All this time let it be noticed the eye symptoms continue and there is no headache. Tingling now appears at the tip of the tongue and gradually proceeds backwards, more especially on the right side until it reaches the uvula. The sense of taste is ~~now~~ largely destroyed and even water feels and tastes peculiar, rather like copper. Tingling again begins about the finger tips and again proceeds up the arm to the deltoid. Then tingling on the tongue which as before passes back to the uvula. The eye symptoms have now meantime disappeared, i.e. in the loss of the luminous zig-zags, although there is still disordered vision in the presence of blindness in the

outer part of the field of the right eye and inner part of left.

When the tingling has affected the tongue and uvula in this manner, speech disorders occur. My mind has been perfectly clear all along and I can watch the development of all the symptoms as they occur, but at this time speech becomes difficult owing to the condition of the tongue with the tingling and numbness and so forth. The saliva refuses to flow and the mouth becomes exceedingly dry and then it is found difficult to pronounce words - to get the tongue round the words so to speak, and I seldom try. This condition lasts so long as the tingling and numbness persists in the tongue, after which it more or less passes away, except a trifling difficulty of which I shall speak later.

At this stage a dreadful nausea comes on, and I begin to try and retch, but in vain. This is about one hour after the onset of eye symptoms. The tingling now usually proceeds to the right lip and at this time vomiting occurs and the eye symptoms in the form of luminous zig-zags come back. The vomiting continues violently, at first the vomited matter consisting of food and then later of bile tinged mucous, and then bile alone, and lastly of mucous

alone, sometimes streaked with blood. The vomiting continues at intervals of every few minutes for the period of three hours or more and at this time the headache has come on and is most severe. It is frontal, hardly limited to one side, but certainly more painful on the right side than the left. It is throbbing and the strain of vomiting makes it almost unbearable. By this time all tingling and eye symptoms have disappeared.

The upper and lower lips of the right side, however, appear to be swollen and feel uncomfortable and there are slight disorders of vision in that things dont look quite right. There is a momentary blindness which seems to pass over the eye. A feeling of drowsiness, the sickness becomes less, then sleep supervenes, only to waken in the matter of an hour or a little more, on the point of vomiting. This vomiting sometimes seems to start the luminous zig-zags over again, but they do not persist longer than a few minutes. After vomiting for perhaps 10-15 minutes, sleep follows, only to waken once more, on the point of vomiting, but this time the retching is not so severe and may only last 5-10 minutes. Once more,, sleep until another attack of vomiting occurs, which is again still less severe. Finally sleep for three

to five hours and awake better except for one or two after prodromes of which I shall now speak.

The countenance may now be observed to be markedly flushed, especially on the right side, the right ear may also be found to be redder than the left. The temporal artery may feel hard to the touch and markedly pulsatile. The forehead, if there had been much vomiting, would appear to be faintly mottled as if some of the minute capillaries were dilated. In some instances, however, the countenance has remained pale throughout.

On waking after a refreshing sleep, while lying at rest the headache does not feel unbearable, but on movement it becomes very much worse. On rising to dress, the headache is extremely sore on every step taken. It is not unusual to feel giddy, but this and the headache gradually pass off and by the evening of the day following the attack, it has completely disappeared. But I may remark that for perhaps a day to a day and a half longer, there persists a slight liability to stumbling on the utterance of big words, the liability, to be sure, is very slight but still I can detect it, though in ordinary conversation it would not be noticed. It is difficult to get the tongue round certain words, and in the same way at the

piano it is found impossible to play any piece of music where a degree of delicacy of coordinated movement is required. As to sight, the eye on which the luminous spectra occurred feels weak and every now and then a small opaque spot may occur in the field of vision. If the eye is rubbed, the spot disappears. This may persist the whole day succeeding an attack. During this stage too, the face appears flushed and friends remark on how well I am looking, while I am feeling anything but well. The following day, however, I have returned to my usual state of health and remain so until the next attack.

I think it desirable that particular attention should be drawn to the following facts:-

(i) The slight loss of coordinated or of delicate movement (as evidenced in playing the piano) - an hour or so before the onset of the occular symptoms. I shall have occasion to point this out again in other instances and not only in movements but in the case of the occular symptoms themselves.

(ii) The appearance of the occular manifestations an hour or two hours later.

(iii) The persistance of the eye symptoms until vomiting has almost commenced and their occasional recurrence during the attack of vomiting.

(iv) The recurrence of the tinglings once or twice during the attack.

(v) The clearness of mind throughout.

(vi) The swelling of the lips after the tinglings have ceased.

(vii) The interrupted sleep to begin with.

(viii) And lastly the slowness of return to complete health of coordinated movements and complete normality of the eye.

I shall discuss these points under the heading of ~~the~~ symptoms.

I might also mention here the frequency with which pains of a rheumatic nature occur in my right shoulder and scapula either before or immediately after the onset of one of these attacks. I have not mentioned the occurrence of these pains in every form recorded, although I frequently, but not in every instance, have some manifestation of this kind. I think this of some little interest in the demonstration of how frequently rheumatism is present during these paroxysms of migraine.

The second most common form of attack from which I suffer is as follows:-

I am evidently ~~me~~ in perfect health, when perhaps from overwork, climatic conditions or other

exciting cause, I may feel my mouth become very dry, all secretion of saliva stop, and from the mere fact of want of lubrication of the tongue, difficulty is experienced in articulating big words. This dryness of the mouth and cessation of secretion persists for about three-quarters of an hour, when a small point near the uvula on the right side begins to tingle, the tingling now proceeds slowly forwards to the tip of the tongue and advances to the teeth and a peculiar tingling feeling seems to be in some of them. The tingling next advances to the lips on the right side and now difficulty of speech occurs, the tongue seems to go to the roof of the mouth and I can't articulate. Tingling may now start in the right thumb or finger of the right hand and proceed slowly up the arm as before. Then the tingling may again start in the mouth, lips and tongue. At this time an overpowering feeling of nausea arises and often simultaneously with this the eye symptoms make their appearance. These eye manifestations may occur in a similar form to those described under the first attack, but they may be different. There may be no zig-zags, only an opaque spot which increases in size to the outer side of the fixing spot and in this area there is loss of vision. Vomiting now occurs and the remainder of the attack follows out precisely that already indicated. The headache does not make

its appearance until after vomiting has started. Frequently, however, either before or after such an attack and also its predecessor though not always, there is pain in the right shoulder.

The points to be noticed here are:-

(i) The premonitary dryness of the mouth with cessation of the flow of saliva.

(ii) The subsequent disorder of sensation in the mouth, lips and tongue.

(iii) The progression of disorders of sensation proceeding downwards to the limb.

(iv) The appearance of the occular symptoms at a much later date than the sensory (quite contrary to Liveing).

(v) The subsequent termination as before.

The third ~~most frequent~~ and worst type from which I suffer is as follows:-

I may at the time have had pain in my right shoulder or some other exciting cause may start the onset of the paroxysm. Here again, I have noticed the presence of very early premonitary signs. The lids of my right eye appear to be "itchy" and I unconsciously rub them, the itchiness continues for about one hour to an hour and a half, when a small spot appears to the outer side of the field of vision

as in the first type. The luminous zig-zags may or may not make their appearance but these eye symptoms of whatever type continue for about fifteen to twenty minutes. Tingling now starts in the fingers of the right hand and proceeds upwards as before, but suddenly tingling occurs accompanied by numbness in the right toes and proceeds up the leg to the thigh. Tingling and numbness have at this time (about an hour after the onset of the eye symptoms) reached the tongue and lips and mouth, and a feeling of nausea follows. The tingling does not proceed to the left side, the right side only being affected. Difficulty of articulation now comes on, but in this case the attack seems to be very much more severe and the mind becomes more or less dulled and I suffer from paraphasia. Words are constantly misplaced. I may know quite well what I wish to say but cannot do so. For example, I may be calling for a basin and will say "bring me a teapot", and so on. At this time vomiting sets in and goes through precisely the same stages as before. The eye symptoms and the tingling and numbness coming on from time to time. The mind remains more or less confused until the sickness is abating and the attack is terminated by sleep as before. The headache is frontal and quite unbearable when at its height, which it reaches after the sickness has set in. It is not

strictly limited to one side, though if anything rather more painful on the right side than on the left. Next day I am very shaky and again I notice the after effects, such as a slight difficulty of speech, slight blindness of the right eye, and want of coordination in the fingers, as evidenced. These gradually passed off and I am once more well.

I would here draw attention to :-

(i) The more widespread affection with

(ii) Numbness and tingling extending into the leg.

(iii) The difficulty of speech and comparative fogging of the mind.

(iv) The after prodromes and their gradual disappearance.

The fourth example from which I suffer is one affecting the left side. The onset of this paroxysm is usually ushered in by eye symptoms with luminous zig-zags. I have further noticed here that in every attack the onset has been preceded by an itchiness of the lids of the left eye about an hour prior to the appearance of the opaque spot to the left of the field of vision. This spot enlarges just as in the right eye, and the zig-zags form as before. These eye symptoms continue for 15-20 minutes, when tingling

begins in the tips of the left fingers proceeding up the arm as far as the deltoid, tingling and numbness may sometimes start also in the left toes and proceed up the leg but not until after it has affected the arm. Sometimes, though rarely, the tingling affects the tongue and lips on the left side and nausea makes its appearance. I do not remember a single instance in which speech was affected in any way, and my mind has remained clear throughout the paroxysm. Vomiting sets in about an hour later and may as in the previous instances cause a recurrence of the ocular and sensory manifestations. The vomiting gradually abates from 6-8 hours and sleep as before terminates the attack. I have also noticed this variety ushered in by early prodromes of want of co-ordinated movements in the fingers on the left hand and a similar difficulty after the attack was over.

Here it would be well to notice:-

(i) The change of side in the paroxysm.

(ii) The presence of early prodromes as before.

(iii) The absence of speech manifestations.

(iv) Similar termination as in other cases.

The fifth example ~~from which I suffer~~ is one in which sensory disturbances are peculiar in affecting the lower limb first. I have been walking along the

road, when suddenly a small point in one of the toes of the right foot begins to tingle. This enlarges and tingling accompanied by numbness proceeds up the foot to the leg. The feeling of constriction now makes its appearance and passes up as a band about 3" long. Tingling begins in the finger tips of the right hand and tingling and numbness proceed up as far as the insertion of the deltoid. At that time about 25 minutes after onset the eye symptoms come on as before commencing as a small spot with laterally the rapid formation of luminous zig-zags. At this time tingling may start again in the toes and proceed up the arm or tingling may commence in the right lip or tip of the tongue and gradually proceed backwards towards the uvula. At this stage I become nauseated and soon vomiting starts as in the previous attacks. This is about an hour after the onset of the attack. The mind now becomes fogged and disorders of speech may occur, either difficulty of articulation or paraphasia or both together. Sometimes, however, the mind is clear throughout and speech disorders do not make their appearance. The headache now sets in and is most severe on the right side, is throbbing in character and quite unbearable. The vomiting continues for several hours with frequent recurrences of the eye symptoms and sensory auras, when

it gradually begins to abate and drowsiness comes on. Finally deep sleep, to awake feeling very much refreshed. The after effects are as before, viz., slight disorders both of vision and speech and the headache gradually disappears.

Here I would call attention to the :-

(i) Occurrence of the sensory aura first becoming evident in the toes of the right leg.

(ii) The late onset of the ocular manifestations.

(iii) The course of procedure of the sensory aura from the lower limb to the upper and from the upper to the lips and tongue.

(iv) The termination as before with the after prodromes.

The sixth manifestation may be described as the ordinary blind headache described by Liveing. I may suffer from it either on the right side or the left. I have never suffered from this form alone - it always has passed on to one of the other more severe forms, but recently I think by careful general treatment and early attention to the paroxysm the moment the early prodromes are noticed, this which would have run its course as one of the severer forms, has been cut short and the ordinary blind-headache has resulted. There is nothing particular to note in the

course of the attack, which follows the lines laid down by Liveing, except that early prodromes occur as before either with the itchyness of the lids or loss of coordinating power in the hand, without the subsequent development of sensory aura. The headache comes on without sickness and vomiting and persists for the rest of the day.. The eye symptoms may be either the zig-zag type or the opaque spot without zig-zags. After the attack is over, there are slight disorders of sight as before. I may draw attention to the fact that although the sensory aura did not make its appearance per se, yet there was evidence of the early prodrome of loss of coordinating movement.

The seventh and last form may be described as the ordinary hemicrania as discussed by Liveing in his first form. Here again it is quite recently that I have come to suffer from this phase and often before the onset of this headache, I usually have had either itchyness of the lids of one or other eye or loss of coordinating movements in the fingers, but the early administration of drugs has prevented the formation of one or other of the severer forms. This may not have been due to the taking of the drugs I shall mention later, but it is at any rate interesting to note that in many instances when I have suffered from

this type I have noticed the early prodromes of a severe form of the disease.

Having now outlined the various types of the disease from which I myself suffer I shall proceed to discuss the various symptoms in detail in their most usual order of occurrence.

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Phenomena of the Paroxysm.-

Liwing (p. 64) draws attention to a tolerably natural order of succession of the symptoms and points out that disorders of sight are the first to occur, followed by those of touch and muscular sense, and subsequently by impairment of speech and disordered ideation. This is succeeded by the development of the headache and nausea, ~~then sets in~~, and vomiting or sleep may terminate the paroxysm. From a reference to the symptoms described previously, it will be evident that such an order of succession is far from the rule, ~~in my own case.~~ I repeatedly have attacks in which the first disorders are those of sensation and of muscular sense and it is not until much later that the eye symptoms develop. The sensory disturbances too do not always work from above downwards or from below upwards. I am repeatedly affected with the disturbances occurring first in the tongue and lips and at other times starting in the upper limb and at

other times in the lower limb. In other instances I have noticed speech disorders and ~~disorders~~ disturbances of sensation in the tongue to occur almost simultaneously. The headache rarely sets in until nausea has lasted sometime or vomiting has actually begun. It will be found in all my attacks that sleep seems to be the termination - except for the after prodromes. The attacks in every case have been unilateral unless a certain degree of overlapping as regards the actual headache. I have never once suffered from disorders of sight or touch on both sides at the same time, but let it be noticed that in every case the disorders of sight and touch occurred on the same side as that of the headache. Liwing (p. 69) has found this to be very nearly equally divided. Many authors, including Gowers (subjective visual sensations) also B.M.J. speak of a greater frequency on the opposite side to that of the headache. That the paroxysm may now affect one side and now the other is evident from the facts laid down. Tissot (T. des Nerfs, p. 385) mentions a case where it attacked the sides alternately. With me, the right side is the oftenest affected, but I have noticed that when the left side is affected, disorders of speech are absent. I have been more liable to attacks on the left side during the past two years than at any time

previously. The left sided paroxysms have always appeared to be the least severe and the most amenable to treatment. From the fact of their more frequent development ~~now,~~ on the left side, when the attacks are not so severe as formerly, I take it that a left-sided attack in a person who habitually suffers from right-sided paroxysms, is an evidence of the gradual abatement of the disease. At an earlier date, I mentioned that my worst attacks were about six years ago and it may be of some little interest to state that left sided attacks have been very much more frequent during those six years than at any other part of my life.

The order of development of the symptoms in my own case and in a few others of those I have studied have always been ushered in by a development of early premonitions. Ross (Diseases of the Nervous System, article Migraine) speaks of patients feeling weary accompanied by attacks of yawning or sneezing 12-24 hours previous to the onset of the paroxysm. Again Mangelsdorf (Yearbook of Med. & Surgery, Gould 1905) describes in Migraine & Epilepsy a distention of the stomach on the day preceding the attack. Zeimssen (Encyclopædia) under Pathology, relates that in many

the attack is preceded from 12-24 hours by a slight depression, a sense of pressure in the head, weariness or paraesthesia in the higher organs, such as muscæ volitantes, tinnitus, &c. Some have observed violent gastralgia before the attack, whilst others mention extreme hunger. These early premonitions are interesting and I think important in respect of treating the paroxysm. But the appearance of weariness &c. is, I think due to so many causes that it is not so important as a guide to the onset of an attack as what I have related in my own case. Of far more value is the recognition of symptoms a few hours before the onset of the paroxysm and which symptoms are absolutely conclusive. I refer to the itchiness of the lids of the eye a few hours prior to the onset of the blind headache. I find this a very constant symptom in my attacks and in those of six other cases that have come under my observation. The prompt treatment then has led to some very startling results. The mention of muscæ volitantes and tinnitus is interesting and if noticed in many might act as a sufficient warning and guide to treatment. In those attacks in which the sensory disturbances were the first to occur and also in the others I mentioned, a loss of fine co-ordinated movements as in playing the piano should be looked for. This symptom I have

found a very constant and a sure sign of the onset of an attack. Moreover, it does not seem unreasonable that in a disease like this, where disorders of touch and muscular sense occur, to anticipate that something of this nature should take place. We have only to look to those cases in which disorders of speech occur and we are at once led to see how co-ordination ~~in speech~~ is lost in many instances. That the muscular sense and loss of co-ordination is not deeply affected and only disturbs the finer movements is seen by reference to the fifth type from which I suffer. I remember taking an attack of this kind whilst on a walking tour round the Pentlands. I had nine miles to walk with the attack on me, the paroxysm having started with tingling and numbness in the right toes, which proceeded up the foot and leg, yet I was quite able to walk except for the slight hindrance due to the peculiar feeling caused by the tingling and numbness in the foot -- the coarser co-ordinating movements of walking were not affected; yet I believe had I been able to test fine movements of the toes they would have been found wanting. These so-called fine co-ordinating movements observed as early premonitions in the hand, ~~as in playing the piano,~~ already described, I believe to be due to the loss of tactile sense

rather than a primary interference with motor functions which I think secondary. Thus the cause of these disturbances is relegated to sensory interference which thereby throws the movements out of gear. Again early premonitions have occurred in the form of a slight difficulty in articulation in the pronouncing of large words. Some two hours or so later the paroxysm would set in with tingling, starting in the tongue or lips - the co-ordinating movements of the tongue having been affected before the onset of the tingling. The occurrence of these early premonitions are of so constant a nature as to be worthy of observation, especially in relation to the treatment of the paroxysm.

The disorders of vision in megrim are usually the first to make their appearance during the course of an attack although this is by no means the order in my own case. These interesting symptoms have been well described by Gowers in his book "On Subjective Visual Sensations", and I shall adopt his classification. The disturbance may be unilateral or bi-lateral, the former being the commoner. There are many forms of unilateral spectra described of which the commonest is that which was seeb by Dr. Airy :-

(1) Expanding Angled Spectrum.- This is a variety

of eye symptom from which I frequently suffer; indeed it and a modification of it are the only forms of eye symptoms I have noted. Early in the day as already mentioned, itchiness of the lids may induce me to rub the eye, sometime after this a small bright spot makes its appearance on one side of the combined fields - usually in association with the right eye - just to the side of the fixing point. This small bright spot rapidly increases in size and assumes the character of luminous zig-zags (fortification figures) nearly at right angles to one another. These are no longer found to constitute a complete circle but near the visual line at the fixing point are found to become faint and finally disappear. The luminous zig-zags are of an expanding nature, but above are not so great at the commencement, so that they appear flatter and the angles lessen towards the fixing point. After the zig-zag oval has extended through the greater part of the field of vision, the upper portion also expands and the angular elements enlarge, except near the fixing point where they remain the same. This process may be repeated several times, finally the expansion near the centre ends in a whirling centre of light from which rays of light may be thrown off. The whole ~~business~~ process may again and again be repeated,

especially with the onset of vomiting or other symptoms as already described. This has been observed by Gowers (Subj. Visual Sensations, 25) who states "that sometimes after the expanding angular outline had attained its maximum, a fresh stellate body was observed near the broken extremities at or about the spot where the first commenced and similar in aspect. It was seen for a short time during the fading of the first spectrum, it then disappeared having the semblance of an abortive attempt to repeat the process." As already mentioned, on the day subsequent to the attack, small luminous spots to one or other side of the fixing point of momentary duration occurred at frequent intervals. These, I believe to be evidence of the gradual abatement of the paroxysm. Regarding the actual zig-zags themselves, many people have described them not only as being luminous but also of various colours. Red, yellow, blue, but seldom were two consecutive lines of a similar colour. In my own case the appearance of the zig-zags are more frequently of the nature of an "electric spark", that peculiar almost indescribable colour, which might have almost any element in it from red to purple. It is bright or dazzling in nature, and often appears of a peculiar whitish-blue colour and very bright. I have watched

most carefully in many of my attacks to detect distinct colours, and although I have felt that they were like what Airy has described "red, yellow and blue", yet, I have not been able to convince myself of such being the case. Sometimes the appearance was like a rainbow, but in rapid dancing movement, but I think the electric spark describes my appearances most fully. These luminous zig-zags are seen whether the eyes are open or closed and occur in the outer half of the combined field of vision. Accompanying this luminosity and fortification figures there is an area in which inhibition has taken place and loss of sight resulted. This area occurs within the luminous zig-zags and is bounded by them. It is most intense nearest the zig-zags and fades away towards their broken ends near the region of the fixing point. On closing the eyes this area of inhibition is found to be faintly luminous and appears to be of a dull yellow colour, on observing this area more carefully it is found to be made up of faint lines parallel to the outlines of the zig-zags. This area of inhibition is also one of subdued discharge. I have noticed that this area of inhibition and subdued discharge often persists after the zig-zags have disappeared and disturbed vision occurs. On closing the eyes in such instances the

faint luminosity is observed. This condition lasts until the termination of the vomiting in many of my paroxysms.

Another form of eye symptoms is one in which no zig-zags appear at all. I may be busy with work, when suddenly, on looking down, the right (or less frequently the left) arm appears or looks funny. I cannot describe it, it is not a condition of megalopsia or micropsia but just a feeling that it does not look the same - it is probable that an area of inhibition has set in, in some part of the field of vision, and thus the sight disturbed, but I have never been able to find out the exact locality of that area, at any rate about five minutes after this, a small faintly luminous spot appears to the side of the fixing point. This spot is more luminous when the eyes are closed. It slowly enlarges until it is about the size of the inhibitory area, bounded by the luminous zig-zags in the previous condition. It is of a faint yellow colour but it is peculiar that in this instance no luminous zig-zags make their appearance at all, but this spot of dull luminosity appears to be just like the area of inhibition in those cases where the luminous zig-zags have occurred. Thus the process of inhibition has occurred without the process of discharge. This spot gradually disappears and with it the disturbance of inhibition goes away. I could not

detect any linear appearance as occurs in the area of inhibition in those cases where the luminous zig-zags are present. In those attacks in which this form of eye symptom is present, the duration and severity of the paroxysms were never so great. There was less vomiting, if any, and less general disturbance. Indeed when the eye symptoms occur, as here, without the zig-zags and only the opaque spot, they are usually late in making their appearance. The attack is usually ushered in by some sort of sensory disturbance, as dryness of the mouth and tingling starting in the throat or lips. I have already referred to this under the second variety of paroxysm. In those patients who have come under my notice this form has not occurred, indeed they all had the unilateral expanding spectrum already described.

The next form of unilateral eye disturbance is:-

(2) The Progressive Spectrum, and is one in which the outer part of the middle zone of the field of vision is affected. (Gowers, Subj. Visual Sensations, 28). "It is angular in character and progressive. At first a zone of darkness develops in the edge of the field diminishing inwards. At the inner side of this area inhibition, a small star or angled sphere appears, which develops into a series of fine angular lines of light, intersecting each other and are concentric with the edge of the field." This form, I

have never witnessed, nor have any cases come before my notice.

(3) Another unilateral form described by Gowers (Subj. Vis. Sens., p. 31) is that which he calls the Radial Spectra, and is one in which the inhibitory darkness develops in the outer and lower part of the field of vision. Within this area a bright zig-zag of uncoloured light occurs advancing from the periphery towards the fixing point but not passing beyond the area of darkness. Another sub-variety was where a bright stellate body appeared within the area of darkness and remained unchanged throughout. These two varieties then constitute the unilateral spectra met with. Much more uncommon is it to meet with a bi-lateral spectrum. Two varieties of this bi-lateral nature have been described by Gowers (Vis. Sens., p. 34) :-

1. The Central Spectra:- "In this form the disturbance occurs at the fixing point where a small spot of darkness appears and grows rapidly, becoming more intense in the centre. Objects are only seen on the outer side of each field. It usually remains like this unchanged until with the onset of the headache it passes away."

2. The pericentral spectra is the last form of bilateral spectra described by Gowers. In this form an area of inhibition appears at the lower part of the field of vision, but which rises until it occupies the region of the fixing point. At this time a small bright circle of lines appears around the fixing point and at the same time the central cloudiness disappears. The angled lines of this circle are irregular, but equally so in the whole circle. This bright circle enlarges until it occupies one-third of the field of vision. At this time it is seen to be composed of bright intersecting lines, in the interstices of which are seen bright circles of light. This circle of light gradually disappears and so also the area of darkness and the headache comes on which is unilateral.

These then constitute the leading varieties of visual disturbance met with in megrim. The bilateral varieties being distinctly uncommon and their interpretation a matter of great difficulty. I shall discuss the localization of these ocular disturbances in connection with the ophthalmic theory of megrim.

Liveing (pp. 84,85) states that the disorders of common-sense or touch are the next in order of appearance. But he affirms that this occurs only in the minority of cases and makes its appearance after the affection of sight, and also that the sight disorder may not be completed until after the touch symptoms have set in. This is not my experience. The disorders of touch indeed occur just as frequently or nearly so as the eye symptoms the attacks starting with tingling in the lips and tongue or hand or foot separately and the eye symptoms coming on some time later. Liveing states that tingling and numbness in the foot is rare. Except in my own case, I have not found it occurring in any of the six special cases studied by me. Two of these, however, had attacks starting with disorders of touch before that of vision. As stated by Liveing (p. 85) the disorders of touch are twofold - (1) numbness or impairment to external sensations, and (2) subjective sensations of tingling thrills, &c. It is rare for tingling to occur without numbness. Benjamin Travers (Diseases of Eye, p. 175) states "that at the instant of their appearance the sentient extremities upon the fingers and tongue are so benumbed that objects of

touch and taste convey a very indistinct impression as if some muffle were interposed." In my own case I made mention of how the touch symptoms and numbness had an order of progression. They always make their appearance at one part such as the tip of the fingers and proceed gradually up the arm, like a constricting band 3 or 4 inches long. This sense of constriction is interesting in so far as it may bear on the vaso-motor theory. As the tingling and numbness proceed up the limb, the parts first affected become clear. The tingling has the feeling of pins and needles rather like the current induced by a galvanic battery. Whilst these sensory affections are taking place in the tongue disturbances of taste occur, giving a peculiar metallic character to fluids taken. In some cases this loss of sensibility is accompanied by a loss of motor power. Liveing (p. 87) regrets that he has been unable to test this. I have already made mention of cases where sensory disturbances have started in the lower extremities, and more particularly on one instance when on a 16 mile walk. I had nine miles to walk when the attack came on, and was able to complete the journey although all the symptoms of the paroxysm occurred. Walking was only rendered a trifle more difficult owing to the numbness and tingling in the foot. Thus the motor power is largely retained,

although the early premonitions already described in loss of fine movements may also show a slight impairment of motor power, but here I think, as already stated, that the condition is brought about more from a disorder occurring in the sensory part of the brain ~~rather~~ than as a result of that, and consequent disorder in the extremities. M. Piorry (Mémoire, &c., 817, p. 412) has drawn attention to the course of these sensory disturbances from the extremities and outlying parts towards the cerebro-spinal axis. There is quite a resemblance between the disorders of touch and those of sight, for in both the disorder is first made manifest in those areas where there is the highest degree of sensibility and in both there is the striking ~~—~~ resemblance of a unilateral character. Liveing has pointed out that when sight affections become bilateral, then touch disorders occur bi-laterally also. It is much more frequent for the disturbances of touch to occur on the same side as that on which the headache has developed.

Mention has already been made, under early premonitions, that disorders of hearing occur; both Tissot and Airy have observed noises in the ears either before or during an attack. In several attacks from which I have suffered, I have hummed a

tune over and over again during the whole of the paroxysm, being quite unable to get it out of my mind. Tissot also has described in his case, disorders of taste occurring: this, as pointed out, is quite a constant feature with me, when the tingling is present in the tongue and lips. After the sensations of touch have fully developed, disorders of speech may make their appearance, and in my case are very much more apt to come on after tingling of the tongue and lips, accompanied by numbness, has started. In fact, I cannot recall an instance in which speech disorders were not preceded by disorders of sensation in the tongue and lips. If the sensory disturbances only took place in the hand and arm and did not proceed to the tongue, then speech disorders were at a minimum. These speech disorders may result from several causes. Thus any paralytic condition of the mouth and tongue may bring about defects. On the other hand, there may be mental impairment associated with confusion of ideas and failure of memory, and again there may not be so much want of recollection of the word to be pronounced, as a loss of the power to utilize the vocal apparatus so as to pronounce it (Liveing, pp. 97-8). In megrim, many forms occur, but in the severer types it may be impossible to tell what has been the dis-

turbing cause and it is often a combination of the above conditions. If we refer to my own case again, it may have been noticed that in many attacks, the mind is clear to the last and although ~~this is so,~~ speech disorders have often occurred.

I remember once travelling from Edinburgh to Aberdeen. I was smoking and suddenly my mouth became very dry and the saliva refused to flow, shortly after, the tingling and numbness began on the right side of the tongue and when the train arrived at Dundee I was unable to speak to the ticket examiner, who came round at that time. I could not get my tongue round the words, although perfectly clear in mind and knowing exactly what I wanted to say - I stumbled over the words so to speak. This attack was followed by eye symptoms after the speech disorders had occurred. Lebert says that in his attacks "there is failure and incoherence of ideas, a difficulty in finding words and a numbness in the tongue and last fingers of the right hand." Here, therefore, was a case in which failure of memory and confusion of ideas constituted the disturbance of speech, but these, ~~memory and confusion of ideas~~ may also in some cases cause the disorder, ~~to occur,~~ and it must also be noticed that

defective power of articulation may lead to the occurrence of an attack. Here, there was no ideational disturbance and the aphasia took the form of difficulty in utterance only. In some of the severer forms of migraine from which I have suffered, distinct confusion of mind has occurred. I remember on two occasions my mind was affected, but with me, the rule seems to be that I retain my faculties clear to the last. But yet another form of speech disorder occurs in megrim, - I refer to the symptom of "paraphasia". I have on several occasions suffered from this , as in the case where I wanted a basin and shouted to "bring me a teapot". But there was no impairment of memory. I knew what I wanted to say, but a wrong word was used. Lastly, in reference to these speech disorders, in every instance in which I have been afflicted, as also in those cases that have come under my notice, the attack has been one in which the eye symptoms and sensory disturbances have made their appearance on the <u>right</u> side of the body. This,

therefore, concurs with what has been said by Liveing (p. 102) on this subject. There is yet one other symptom which I have noticed and conclude would come under this category of speech disorders, and that is that in some of those cases in which speech disorders have occurred, an early premonition presented itself as follows :- I would be writing a letter say, and would be going to write the words "Bayswell Road, Dunbar", when I might find I had written "Bayswell Dua", and would then notice the mistake, the mind seemed to be running too far in advance of the word being written, and the first letter of the next word would be inserted - in this instance the "D and the u" of Dunbar - when writing the word "road". I have not noticed this peculiarity often enough to pronounce that it does occur as an early premonition, but I think it worth mentioning here.

The Psychical phenomena which make their occurrence during an attack may be either intellectual or emotional. The intellectual phenomena are well exemplified in the disorders of speech. There may be loss of memory and confusion of ideas as I have mentioned occurred in me in my most severe cases. Liveing (p. 111) refers to a case where a patient suffered from migraine throughout life and suddenly suffered from loss

of memory and confusion of ideas which were put down as threatenings of apoplexy. I have met with one case of this kind. Mr. H-. aged 60, had suffered periodically from attacks of what he called "biliousness", but on enquiry were without doubt attacks of Blind headache. A few months ago, he was excessively busy and worried over literary work, when he suddenly became confused, the eyes rolled in the head and he had diplopia, probably due to a passing strabismus, this was followed by a confusion of mind and when asked questions answered wrong words altogether without meaning. The attack gradually passed off, and the patient was almost well again next day and had no further trouble. I think it highly likely that this was an attack of megrim in which the earlier symptoms had been missed out. Emotional symptoms often usher in a paroxysm, such as irritability of temper, and undue excitement from any cause whatsoever. Liveing (p. 114) mentions a vague and unaccountable sense of fear or dread occurring in some before the onset of the attack. He also refers to the occasional occurrence,in children especially, of nightmare, somnambulism or trance. These, I have never come across and have myself never been affected in such manner.

Giddiness or Vertigo.- This is a comparatively

common feature of a migrainous paroxysm, more especially towards the termination of the attack. I, personally never feel any degree of vertigo. After the paroxysm has ended, and I get out of bed, I feel light-headed, but even after the very worst seizure, I never suffer from any degree of vertigo beyond this "light-headedness". There has never been any feeling of rotatory movement in my own case nor in any of the cases which I have studied. Liveing (p. 128) has noticed in people who are subject to megrim, that on rising suddenly from a sitting position, they become giddy. I have repeatedly noticed this in myself, although I do not suffer from any heart lesion.

The headache associated with the paroxysm may vary in its severity, position and nature. In my own case it is a very late symptom and I have seldom noted its appearance until after vomiting has set in and often it has been delayed until vomiting has ceased. It starts on the side of the head on which the body is affected, thus if the eye symptoms or the sensory disturbances are on the right side, then the headache is worst on the right side. It is culminating in its character and throbs with each beat of the temporal artery. It is not strictly limited to one side, but may extend over to the opposite side as well. At its

height it is almost unbearable and is intensified by any noise or strong light or on the slightest movement. Its duration varies, but it usually lasts the entire day following the attack. Its fastigium is reached shortly after the vomiting has ceased and in some instances when the strain of vomiting is at its height. It usually starts over the temple and affects the eye which feels painful to touch. Cases have been mentioned where the pain occurred at the back of the head, and as Sir J. Clarke says these cases usually occur in females and are associated with uterine disturbances. Great variations in the headache, however, are noted. In some people the headache only occurs for a very short time and is moderate in its severity, whilst in others it is excessively severe and may last 2 or 3 days. Zeimosen (Encyclopedia) has noticed that pressure on the carotid artery of the same side eases the pain. This, I have never found to be the case, nor does it increase the pain - the throbbing still continues the same.

Nausea and Vomiting.- Liveing (p. 137) considers that these symptoms normally follow the development of the headache. Such, as already stated, does not usually occur with me, but it is far more usual

for the headache to develop with the onset of vomiting or after it has started. Nausea usually occurs with me after the tingling has started in the tongue or when no sensory disturbances have occurred, some 60-90 minutes after the onset of the eye symptoms. At this time the stomach feels deranged, there is often an escape of gas from the rectum and the bowels feel loose. Digestion seems to be in entire abeyance. Sajous (Encyclopedia by E.D. Bondurant Mobile) has noticed a regular and well marked increase in the size of the stomach during an attack of megrim; this dilatation, he says, includes all parts of the stomach. He states that out of 409 cases afflicted with megrim only one presented a normal condition of the stomach. I have not had a sufficient opportunity of testing the accuracy of this statement, but there seems often to be an accumulation of gas in the stomach at this time. Vomiting then sets in, usually about 10-20 minutes after the feeling of nausea comes over me. Its violence depends on the virulence of the paroxysm and in a severe case vomiting may go on intermittently for several hours. All the stomach contents are expelled and laterly only mucous streaked with blood. Liveing (p. 138) regards vomiting as one of the

natural terminations of the paroxysm. "There is no doubt that it sometimes cuts short an attack. Many patients say "when I get sick I get relief" and have tried to induce sickness on this account. My uncle on my father's side was a tea-planter in Assam, he had long suffered from so-called biliousness, but from the family history, as pointed out, it appears to me that he must have suffered from megrim in some of its forms. He had evidently observed that the act of vomiting had terminated his seizure and used to bring about vomiting artificially, when he realized he was in for an attack. By this means he met his death, for he had also evidently an enlarged spleen from malaria, and one day when inducing vomiting, the strain had ruptured his spleen. This is interesting in view of what Liveing has said regarding vomiting as one of the natural terminations of the paroxysm. Fothergill and others said that vomiting gave relief by promoting the evacuation of some visceral irritant - undigested food or bile. This, I cannot agree with, for in the majority of my attacks, the stomach has been quite empty, except for the "wind" which I have already mentioned and its presence there I believe to be due to the paralysis of the action of the bowels, which I think occurs before the height of the paroxysm has been reached. Liveing (p. 139) believes that the act of vomiting gives relief by substituting one

form of nervous peroxysm for another, just as in sea-sickness. With me, however, vomiting is by no means always curative, although I always feel that I am well on towards the end of the paroxysm when vomiting has set in. Sleep, however, just as often terminates the paroxysm and I will now discuss this symptom.

Drowsiness is a frequent occurrence at the height of the attack, in some people it may verge on coma. It is not curative but is similar to the drowsiness occurring in epilepsy, and sometimes in asthma. I am usually perfectly clear in mind until after vomiting and then become drowsy, which is thus the commencement of the natural and refreshing sleep terminating the attack, and not the type of drowsiness suggested by Liveing (p. 142).

The paroxysm of megrim is most usually terminated by sleep. This sleep is refreshing and curative, and Liveing (p. 147) regards it as the natural result of the exhaustion of the nervous system produced by the attack. In some patients, however, sleep does not terminate it, but cure takes place gradually by a slow return to normal health.

In my own case sleep comes on some time after vomiting has set in and after three or four hours of it, I awake refreshed and feeling much better. This

is followed next day by slight symptoms, there is a gradual abatement of the headache, but I have noticed on playing the piano the same difficulty of co-ordination as occurs in the fingers at the outset of the attack, if the hand had been previously affected by sensory disturbances. Again, if eye symptoms had occurred, peculiar small luminous spots make their appearance, lasting only for a moment at a time; and lastly, if there have been speech disorders, then there still persists a very slight difficulty in pronouncing big words throughout the day following the attack. Thus, I believe that although sleep usually starts the termination of the paroxysm in most people by its restful action on the brain, yet I believe that the brain has been so disordered throughout the attack, that it takes not a little while before all these sensory and other disturbances have completely abated, and a return to normal health has occurred.

Pathology:- This is a subject which has been very widely discussed and many conflicting views have been established. Probably the oldest and most popular theory, especially by the laity, is that the disease is due to biliousness. The ancients believed

in the "cardinal humours" of which yellow bile and black bile were two. Various diversities in the condition of these were said to be the determining cause of a paroxysm and methods had to be adopted to eliminate them in order to bring about a cure. Most people believe and attribute their sufferings to bile and it is only on closer enquiry that other facts are brought out. I need only illustrate this with reference to one patient whom I saw the other day:- Mrs. T., age 55, had suffered for many years from what she called bilious attacks - fits of vomiting came on and she was incapacitated from her work for the time being. She had to lie down for most of the day and a heavy throbbing headache resulted and lasted over night. Next day, she was her usual self. She had noticed that heavy articles of food were most prone to bring on her attacks, such as butter, suet, &c., &c. On enquiry, however, I found that previous to the onset of her attacks she had the unilateral expanding spectrum described by Dr. Airy, with the typical area of blindness. On my questioning her, she had noticed that an itchiness of the lids of the eye occurred an hour or two before the onset of the attack. On further enquiry, I found that she had periodically suffered from the disease since her periods started, that her

mother had also suffered, and that she had frequent attacks of rheumatism. She was a "fish curer" by trade and had to deal with "oily substances" at special seasons of the year and that some of them in particular - as she says - she could not do with at all and the smell of them invariably produced an attack. (This I have already referred to when discussing sensory impressions). She has noticed that her attacks have become very much less since her change of life. During one of her headaches I compressed the right carotid and she said it relieved her headache. Here then is a typical instance of many of those who attribute their trouble to an attack of the bile. They have never considered the eye symptoms &c. until they are brought home to them. I think here too, the history of heredity, the history of rheumatism, the early date of commencement, the production of the attacks by smells, &c., and the comparative abatement since the change of life, clearly point to the condition being of some other nature than that of bile. More than 200 years ago, Sydenham protested against the bilious theory and many others followed in his wake. Symonds, in his Gulstonian Lectures in 1858, enters into an exhaustive study and comes to the conclusion that they

are nearly all of nervous origin. Certainly to my mind, because vomiting occurs during some of the attacks, that is not to say that the paroxysms are in any way related to the condition of the bile. In opposition to this theory, that of a sympathetic or eccentric origin was established. Here a nervous origin was placed at the periphery of the system, an irritation here inducing the attack. The stomach was again recognised as the chief seat where the irritation started and hence the name "Gastric Megrim" arose. This idea was also recognised at an early date in the history of medicine. Galen, Fothergill, and Tissot each supported this view, but one must again ask "how are we to attribute the great factor of heredity or the periodicity of the disease and many other features, which clearly point to the stomach condition being merely one of the many symptoms of the paroxysm ?" There can be no doubt, however, that sometimes indiscretions of diet may determine an attack in one who is predisposed to the condition. Another source of peripheral irritation which was laid down as a cause of the trouble, was the uterus. It has been repeatedly noticed that women often attribute their sufferings to some abnormal condition of the uterus or ovaries. It has also been noticed that

attacks have come on at the menstrual periods and that there is a tendency to cessation after the menopause. But then at this time from the onset of the menses there is a great strain on the whole system, not only of the uterus, and we also know that at puberty there is often a mental breakdown, as is seen by the development of insanity &c. at this time. Liveing (On Megrim, p. 253) says:- "there can be no doubt that the catamenial period - the period of ovarian and sexual activity in the human female - is intimately associated with, if not dependent on, a periodically recurrent activity of the nervous system." Thus it seems clear that really this occurrence of the attacks at the monthlys is just because at this very time the central nervous system is unduly excited and an attack liable to occur quite apart from the uterine influence.

Another source of peripheral irritation believed to induce a paroxysm was strain on the eyes and gave rise to the idea of "Ophthalmic Megrim" adopted by M. Piorry, who was the first to point out the occurrence of visual phenomena in Megrim. He believed that an exciting cause acts on the retina and iris, a morbid condition is set up, manifested by oscillations which

spread from the small circumference of the iris to the larger, giving rise to a gradually expanding luminous circle. Later on the disorder extended to the 5th pair of nerves and pain occurred. Then, if the affection extended to the sympathetic and 8th pairs of nerves, nausea and vomiting occurred, and if the nerves of the tongue become involved, these parts become the seat of vibrations, which are felt as oscillating sensations. (Memoire: "Du procédé opératoire à suivre, &c. § 822, p. 415). But how would M. Piorry explain some of my own attacks ? As already pointed out, I frequently suffer from a paroxysm in which the disorders of sensation are the first to make their appearance either in the tongue, lips, arm or leg, the eye symptoms coming on at a much later date. Again I have repeatedly suffered from attacks in which no eye symptoms have occurred, but yet all the other phenomena have become manifest. Clearly then an irritation of the iris is not the starting point of the paroxysm. Undoubtedly severe strain of the eyes may induce an attack of megrim in some instances, but then it is not the iris that is fatigued but the brain. Now that the conditions of Myopia and Hypermetropia have been described and understood it is easy to see how this

constant irritation on the brain may induce an attack. In virtue of this fact, I had my eyes tested a number of years ago, but there was no evidence of either myopia or hypermetropia, my eyes being perfectly emmetropic. We must, therefore, look to a central cause for the occurrence of the eye symptoms, and this is the view adopted by Gowers in his Sunjective Visual Sensations (p. 53) where he states that "the difference between the two forms of expanding spectrum, the one-sided oval of Airy and the pericentral varieties, suggest that in the former the discharge is in the higher visual centre of one hemisphere, while in the latter it occurs in both, combined by a perfect functional fusion." I think enough has been said to show that the primary seat is not in the Iris as M. Piorry has thought, and that although eye strain may act as an exciting cause, yet it is by no means the real cause of the condition. We must look somewhere else, than to the periphery for the essential cause and this brings me to consider the "central theories".

Various vascular theories have been brought forward in order to explain Megrim and its allied diseases. The view that there was a general plethora which caused

the disease, has long been given up, but a local plethora is admitted to occur. Indeed Lebert attributed one of his attacks with loss of memory to such a cause. The first great vascular theory brought forward is that of Active Hyperæmia and expounded by Dr. Parry. He pointed out that in Megrim, Epilepsy, and many other disorders, there was a determination of blood to the head and this induced the attack. He believed that the cause of this determination was an increased action of the heart. He elaborately describes how he has stopped many attacks of epilepsy, megrim and allied disorders by firm compression of the carotid on the affected side. Certainly in some cases of megrim, the headache may be lessened by pressure on the carotid - I have noticed it repeatedly but it does not occur in all and has never occurred in my own case,- indeed Zeimssen, Berger and others have noticed that in certain cases pressure on the carotid increases the pain. I do not think that increased action of the heart does induce an attack, for why do attacks usually come on in the morning or through the night, when least exertion is being put on the organ ?

As opposed to Parry's view, Dr. M. Hall put

forward the passive or venous hyperaemia or congestion theory, in which he would have us believe that epilepsy, mania, apoplexy, &c., are due to a spasm of the muscles of the neck "Trachelismus" or to a spasm of the glottis "Laryngismus" or to both - each and both act by affecting the venous return from the brain and according to their degree cause certain degrees of symptoms. Hall believes that the primary spasm is induced by an eccentric cause, especially emotion, or irritation in the bowels, uterus or elsewhere. This view, however, gives no satisfactory explanation, nor does it explain the symptoms which are so varied and typical. The more recent theories in association with vascularity are those of Du Bois-Reymond, Möllendorf and Latham. They each deal with a disturbance in the vaso-motor system and believe that the sympathetic nerve of one side is affected.

Du Bois-Reymond had noticed that in his own case at the outset of the attack, the face was pale and the eye sunken, that the pupil on the affected side was dilated, that the conjunctiva was red and that after the attack, the face and ear of the affected side became red and there was an increase of temperature, and it was then that the pain and headache

occurred. These symptoms, he points out, are those of a tetanus of the sympathetic nerve. He believes that this lowering of the blood pressure at the outset of the attack may account for the visual spectra in megrim. He admits that redness of the conjunctiva at an early date is against his view, but tries to account for this by the early relaxation in this area - this relaxation of the vessels occurring ultimately from exhaustion and being the cause of the increased warmth and also pain by the effect of dilatation of the vessels on the vaso-motor nerves. Liveing (p. 302) disagrees with this and thinks the cause of pain, on the contrary, ought to be sought for in a condition of anaemia and spasm. For my own part I feel that there are many points against this theory. Certainly in my own case I have noticed that my face was pale and the eye sunken at the commencement of an attack and that afterwards it was flushed on the affected side - but this is by no means a constant appearance, for the face is often flushed from the outset and may even be paler after the attack. Another point too, is that redness of the conjunctiva often occurs very early in the attack and is associated with the early itchiness of the lids, already described. How then could the stage of relaxation have been reached before the

eye symptoms, which, Du Bois-Reymond implies, are caused by contraction of the vessels, have started ? It may be more than two hours later before the visual spectra make their appearance. Regarding the dilatation of the pupil described by Du Bois-Reymond, this is by no means a constant symptom, I have never been able to detect it in myself, and those who have witnessed some of my attacks have stated that on the contrary the pupils were contracted or even both normal and that no difference occurred in the size of the one or the other. Opthalmoscopic appearances, I shall discuss presently, but I may here state that they do not agree with the theory here set forth that the eye symptoms are due to anaemia.

The second vaso-motor theory is that expounded by Möllendorf of Berlin. He takes a directly antagonistic view to Du Bois-Reymond, and tries to prove that migraine is due to a one sided loss of power in the vaso-motor nerves, affecting the carotid and thus establishing a determination of blood to the head. He believes that the sympathetic nerve is the seat of affection in Megrim and that the paroxysm is induced by mental emotion, strain on the eyes, &c. He noticed that pressure of the carotid artery at the level of the thyroid cartilage on the affected side caused the

headache to vanish whereas compression of the opposite carotid aggravated the condition. He also relates that by opthalmoscopic examination of the fundus during a paroxysm that the central artery and vein were dilated. He also describes a slowness of the pulse at the wrist and a softness of the pulse in the temporal artery, and as a result of this, the hands are icy cold and the face on affected side warm. He concludes by thinking that megrim is hereditary by a hereditary deficiency in the vaso-motor system.

There are many points described by Möllendorf, which I have noted in my own and other cases. I have been able to examine the fundus of two patients during the paroxysm, and in each case have noted a dilatation of the central vein and artery of the affected side. On several occasions, I have noted a slowing of the pulse at the wrist before the onset of vomiting in a paroxysm, but even this is by no means constant. It has never fallen below 50 , indeed the lowest was 55 beats per minute. At other times, however, I have noted a quickening of the pulse, especially towards the height of the attack. I have frequently tested on myself pressure on the carotid of the affected side without any relief of symptoms, thus I think the

appearances described by Möllendorf too uncertain to furnish an absolute cause of the disease.

The third vaso-motor theory was that introduced by Latham of Cambridge, who believes "that the primary cause is a morbid activity of the sympathetic nerve in consequence of a defective control or inhibition by an exhausted or enfeebled cerebro-spinal system." This produced an anaemia of the parts supplied, by a contraction of the vessels of the head, and thus the eye symptoms might be accounted for. He thought that the headache might be due to a secondary hyperaemia of the brain following exhaustion of the activity. This theory seems more of a compromise between that of Du Bois-Reymond and Möllendorf, and still places the seat of the disease in the sympathetic. Like Liveing, I feel that these vascular phenomena are too inconstant both in their appearance and progress to assign to them the real cause of the trouble. It is difficult to see how anaemia of the brain can account for the presence of the eye symptoms for I have suffered from attacks of megrim in which the eye symptoms have been entirely absent and yet again have started for a second time in the course of the same attack after the onset of the headache which is here attributed to be due to a resultant hyperaemia.

There are certainly many symptoms which can be well explained by one of these vaso-motor theories. Thus in my own case, after a severe attack I have noticed a swelling in the lips on the side on which tingling had previously occurred. Then again I have sometimes noticed a slowing of the pulse at the height of the attack. Also the redness of the conjunctiva at the outset and the dilated condition of the vessels of the fundus in those two cases examined. Then again I mentioned under my symptoms that a feeling of constriction occurred in the limb during the occurrence of the tingling. One might mention also the occasional flushing of the face and in others the paleness of the face during the paroxysms. All these symptoms are so divergent and so inconstant that it appears to me, that whilst anyone of them taken separately might strongly advocate one or other of these theories, yet collectively and on closer examination, owing to their irregularity it would appear that they result from the paroxysm rather than that they are actually the cause of it. Some other hypothesis must, therefore, be framed and this leads me to discuss Liveing's "Theory of Nerve Storms".

Here the author considers "that the fault does not lie in visceral irritations or anomalies of the

circulation, but believes that there is a primary and often hereditary vice which consists in a tendency on the part of the nervous centres themselves to the irregular accumulation and discharge of nerve force.... ... The immediate antecedent of an attack is a condition of unstable equilibrium and gradually accumulating tension in the parts of the nervous system more immediately concerned, while the paroxysm may be likened to a "storm" by which the condition is dispersed and equilibrium for the time restored" (Liveing, Op. Cit. p. 336). This theory he puts forward not only to account for the migrainous paroxysm, but according to the part affected, any of the other neuroses, such as epilepsy, eclampsia, mania, &c., &c. He argues that this explosive nature is shown by the intermittent character of these diseases, that, for sometime after an attack, the patient can expose himself to those very causes which excite the onset of a paroxysm; that one attack keeps away another; that the exciting causes are so diverse; that the symptoms of the paroxysm follow almost a definite order of sequence and lastly "that those who adopt other theories of neurosal affections have still to assume a minor degree of this very nature as described as the starting point of every seizure." In other words, that the circulatory

disorders are secondary and not the primary cause. Regarding some of these statements I have already pointed out how in my own case I differ from them. I usually find that one attack does not ward off another immediately and I have often felt disgusted at having had an attack as I have felt that another was sure to follow at a shorter interval. Then again, I used to notice one form of excitant which was very prone to bring on an attack in myself, viz., going to the swimming baths, more especially if I dived a lot, I used to get a slight chill which I think brought on the attack. If I went back two or three days later another attack started, so much so that I had to give up the swimming baths altogether, although hot baths at home seem to have no effect on me in this way. Then again as to the symptoms of the paroxysm following a definate order, I need only refer to th seven types from which I suffer and which have been already described. There it will be noted that the eye symptoms frequently recurred with the advent of vomiting, that sometimes the attack was ushered in by dryness of the mouth, followed by disorders of sensation and the eye symptoms did not appear until later on in the attack; that at other times the order of sequence of

the paroxysm seemed to be almost completely reversed, the attack being ushered in by disorders of speech, followed by disorders of sensation, and then by disorders of vision and so on. I do not mean to say that these phenomena are at all frequent but it is interesting to note that they do occur. I shall allude to these facts again under the discussion on Rheumatism. Liveing, in order to further prove his argument, draws a picture from the analogy of the healthy nerve actions and points out that the acts of sneezing, yawning, gaping, flushing, the natural development and gratification of our appetites, the emotions and the sexual instinct, are all striking examples of physiological nerve storms. He then proceeds to show how these can be disturbed by pathological causes, such as the disappearance of the appetite on the hearing of bad news, &c., &c., and then shows how any of these healthy nerve actions, pathologically, may constitute part of a paroxysm of one of the neuroses. Liveing then states, that although in the majority of instances megrim is an idiopathic neuroses of a functional kind, yet like epilepsy it may sometimes be symptomatic of structural lesions. He divides these forms of symptomatic megrim into three classes:-

(1) The Gouty or Arthritic Megrim.- Here he has noted that often the father may have gout and the son suffer from megrim, or a person may suffer from megrim and that he replaced at a later date by gout, the megrim entirely disappearing. Here the explanation put forward is that the excess of uric acid in the system exerts a toxic influence upon the brain and thus produced an attack. Liveing tries also to show that gout itself is one of a pure neuroses and therefore it is not surprising that megrim should occur in association with it.

(2) Malarial Megrim or Brow Ague.- Here again, megrim is shown to occur and Liveing quotes largely from the writings of Dr. MacCullock (an essay on the Remittent & Intermittent Diseases, including Marsh Fever and Neuralgia, Part. II., p. 31) who points out the occurrence of headache &c. as one of the commonest of the masked or irregular forms of intermittent fever, which headache, may present any of the features of this fever, such as the "tertian" or "quartan" types, or may alternate with the paroxysms of the fever. Again an attempt is made to show the neurosal character of malaria, such as the periodicity and intermission, the

stages of heat and cold, and the same tendency to force of habit as occurs in epilepsy, asthma and megrim. "We may even trace," he says, "in them indications of a limitation to certain periods of life so characteristic of many nervous affections. Thus we see that megrim is but one expression of that neurosal habit which is impressed on the nervous system by malarial influences."

(3) Megrim associated with organic disease of the brain.- This is also pointed out to occur in cancer and cerebral tumours, &c. In some instances megrim has assumed a severe form of cerebral disease and Liveing (Op. Cit. p. 422) states "that a hereditary tendency to premature cerebral disorganisation at a later period of life occurs in megrim or, that the constant return of the seizures impairs the nutrition of the brain and predisposes to haemorrhage and softening."

These then are the views of Liveing and we have to ask ourselves if they explain the nature of this interesting disease. No-one reading Liveing's book on Megrim and Sick Headache can very well help being impressed by the picture he draws of megrim being one of the Neuroses. He describes so closely step by step the many links binding it to the family of epilepsy, asthma, hysteria, &c. The suddeness of onset, the

periodicity of the attacks, the intermittent period in which the patient is quite healthy, the order of sequence of the symptoms during a paroxysm, the termination in vomiting, drowsiness and sleep and perhaps the passage of large quantities of limpid urine after the attack is over, and so on. I think that all these facts undoubtedly point to a disturbance in some part of the brain, instead of being the result of changes in the vascular system or a peripheral disease only. I do not think, however, that in every case there is a gradual accumulation of nerve energy which suddenly bursts forth in the form of a "nerve storm" and that after it is over the patient for the time being can expose himself to those influences which started the paroxysm, without any attack occurring. As I have already stated I cannot always do this and the same form of stimulus will start a further attack. I certainly believe, that, as Liveing says, the patient develops an "hereditary vice" or deficiency in the brain at the seat of the disturbance and that eccentric irritations will cause the explosion, but I am of the opinion that megrim is much more frequently of what Liveing calls a "symptomatic type" than he would lead us to believe. I have throughout this article laid stress on the appearance of Rheumatic features in many of my seizures and also in those of others. I shall

presently point out what bearing this has in relation to megrim, as evidenced by treatment on rheumatic lines. Liveing describes Megrim in association with gout, and, as stated, believed that this latter disease is one of nervous origin. He also thought that it was difficult to explain why the uric acid circulating in the blood, could confine itself to one part, and so cause the paroxysm, but that this difficulty was largely overtaken by the neurosal theory of gout itself. In a similar manner, he has attempted to show the occurrence of megrim in association with malaria, but what startling revelations have occurred since his day !! How greatly has our whole knowledge on this disease been enhanced by the recent discoveries of Laveran, Golgi, Sternberg, Manson and others !! One can no longer suggest that malaria is a paroxysmal nervous malady, but one must now relegate it to one of the specific infectious diseases, for has not a living parasite been discovered which is introduced into the human frame by the mosquito ? and has it not been demonstrated that this very parasite invades the blood corpuscles and thus it and its toxines conveyed to all parts ? Here, then is a newer view demonstrating the occurrence of megrim in association with this disease.

By the circulation of the blood these living parasites are brought into intimate contact with a certain inherited disposition and these and their poisons acting on a part which is below par have little difficulty in starting a paroxysm. Does this mere fact not suggest that certain toxines or organisms in the blood itself (and not the disorders of the circulation, which are, I believe, secondary) acting on a brain, which as Liveing puts it, is the recipient of an "hereditary vice", might be a frequent stimulus to the onset of a paroxysm. Liveing has referred to the Gouty diathesis and the accumulation of uric acid in the system as a cause of megrim in such subjects, but he finds difficulty in explaining why the uric acid should affect only <u>one part</u> of the brain. Might I be allowed to venture an analogy in another disease - Rheumatism.- I have tried to point out from time to time, the prevalence of this disease in some form in association with megrim. We further know that in this disease a rheumatic toxine circulates in the blood, it is further often hereditary in its nature, it is influenced by many extraneous causes, notably so the weather, and it is observed not to attack the whole frame subserviently, but that frequently, it may affect one or more joints, one time and at another, the muscles in the form of lumbago and so forth - in fact, some part of the body, which is temporarily below par. Why may not this toxine affect

the brain as well as the joints, muscles and other textures, more especially if there is at the same time some hereditary weakness ? May not this toxine, circulating into such intimate contact with the parts, induce the onset of a paroxysm of megrim ? I do not for a moment deny but that megrim may be idiopathic in a very large number of instances. I am thoroughly in sympathy with Liveing's views, but I merely wish to draw attention to a fact which may explain better a number of cases which meet with difficulty when relegated to his view, such for example, as those cases in which there is a disturbance in the order of sequence in the paroxysm and in which, therefore, there is some difficulty in showing how such a nerve storm acts which neither starts from above and proceeds downwards, nor from below and proceeds upwards, but affects those areas of the brain irregularly which are recognised as the anatomical seat of megrim. I shall again refer to this presently. Therefore, I may bring forward what I consider one more probable cause of the paroxysm in many cases, and to suggest thereby something which may be done in treating the condition. Undoubtedly, as admitted in my own case, exciting causes of a paroxysm have frequently been over-work, nervous worry, emotions, sensations of smell, &c., and others have

described food and so forth. But even here it is quite possible to conceive that by these influences, the brain, in a rheumatic subject, may be so lowered in vitality that now the toxines circulating in the blood have gained the upper hand and the attack has commenced. Again, another feature of megrim, of which Tissot has spoken, and which I am able to corroborate, is the influence of climatic conditions. May not this again point to rheumatism having something to do with the onset of the paroxysm in some subjects ? And we may ask, will such a theory explain all the symptoms of a paroxysm ? We have seen how the theory of nerve storms not only accounts for the nervous features but also those of the circulation and vaso-motor disturbances. Here again, the toxine is brought into intimate contact with the nerve centres and thereby induces the onset of the paroxysm. In those cases where the natural order in the sequence of the symptoms does not take place, we can assume that the toxines have affected those areas first which first are evidenced as symptoms. The vaso-motor disturbances are still accounted for by the poison irritating the vaso-motor centres in the brain. Moreover, the discrepancies observed by many authors of one another, such as in

the writings of Du Bois-Reymond, Möellendorf and Latham, such as the paleness of the face at the outset of the attack described by one and flushings by others, are readily overcome. Hence, I think all the symptoms of a paroxysm of megrim could be accounted for on this basis.

Therefore, I think, the pathology of the condition can be summed up as follows:-

(1) That Megrim is undoubtedly a disease of the nervous system of an intermittent and paroxysmal nature, closely linked to the family of other Neuroses.

(2) That the disease is not of peripheral but of central origin.

(3) That during the paroxysm, anomalies of the circulation occur, but that these anomalies are the result of and not the primary cause of the malady.

(4) That in the large majority of cases, there is an "hereditary vice" in a particular part of the brain which becomes of unstable equilibrium and a stage of discharge is induced by various extraneous causes.

(5) That although Idiopathic cases cannot be denied, yet there is a considerable percentage of them of symptomatic origin and occur in Gout, Malaria, and Organic Disease of the Brain.

(6) That the disease may also be symptomatic in those of a rheumatic diathesis, the attack being induced by the circulation of the toxines into intimate contact with a part which is the recipient of "an hereditary vice" or is below par.

(7) That many of those cases in which there is a difficulty in explaining some of the symptoms of the paroxysm, such as the paleness or flushings, the diversities from the usual order of sequence of the symptoms, the apparent influence which climatic conditions have over the frequency of its recurrence, &c., can readily be accounted for on the assumption of the circulation of toxines in the blood. Moreover, probably in those cases where such diversities occur and in many others a history of rheumatism will be elicited and in such, if benefit is to occur, the treatment will have to be conducted on anti-rheumatic lines.

We have admitted Megrim to be of central origin, where then in the brain will the lesion exist? We have seen how by M. Piorry, the eye symptoms were localized in the iris and how this view was exploded, and how Gowers localized these in the brain. From the other symptoms of the disease it is evident that

the second pair of cranial nerves are implicated as well as the sensory division of the Vth and VIIIth. I have spoken of motor symptoms and believed them to be of a secondary nature to the sensory. In some cases it has been shown that confusion of mind has occurred during a paroxysm and this, therefore, must point to the fact of an extension of the irritation to the hemisphere. Thus the disease seems to be one affecting the optic thalamus and the sensory ganglia between that and the vagus, with sometimes radiations to the hemisphere and to the sensory ganglia of the opposite side. Liveing (Op. Cit. p. 396) states that "since in the typical seizure the visual disorder is always the initial symptom, the headache the middle, and nausea and vomiting the last, we infer that the storm has its point of departure, or principal focus in the optic thalamus and that its normal course is from above downwards, or from before backwards in the sensory tract." It is, in such cases with which I am sometimes affected, where the order of sequence is far from that described above, that such might be accounted for on the assumption of rheumatism, and the consequent indiscriminate affection of these centres. The whole matter however is still much one of doubt and uncertainty. Yet another instance

where an unnatural sequence might well be conceived to occur and that is in some cases where the migrainous paroxysm was induced by organic disease of the brain, such as a cerebral tumour. The symptoms might well be divergent and wide of the common picture portrayed.

Diagnosis.- The diagnosis of megrim will be very largely on the history of the patient, the intermittence of the paroxysms, and of evidence of an hereditary predisposition or some rheumatic taint or other "symptomatic disease". One must not mistake the headaches of meningitis nor of advanced disease of the brain, although sometimes a cerebral tumour may produce the typical migrainous paroxysm. The cases which may present difficulty are cases like those already described such as Mr. A. which may present the symptoms of a threatened apoplexy and in which the earlier symptoms have been omitted. Here again, the history will be of great service and also the rapidity with which the patient recovers. Disorders of speech occurring during an attack must be discriminated from those other organic forms of aphasia. From epilepsy the disease is sharply eliminated by the absence of any motor or spastic symptoms.

Prognosis.- The immediate prognosis is good, but the general prognosis unfavourable as regards abatement. The disease will in most cases persist until old age or the change of life. In some cases prophylaxis will ward off the attacks and make the periods of intermission wider apart. Cases have been recorded where megrim has developed into advanced disease of the brain or has rendered the brain more liable to apoplexy, but these cases are rare.

Treatment.- Most authors consider that very little can be done for megrim in the way of treatment especially in those in whom the disease has persisted for many years, and in them, think that they must just patiently await the time when the disease will naturally decline. During the paroxysm they uphold that the less done the better, injudicious interference then will only aggravate the condition and prolong the attack. While agreeing, that in very many cases the condition is obstinate, yet I think a good deal can be done to lessen the frequency of the paroxysms and mitigate their severity. I shall first discuss the treatment of the paroxysm itself.

It seems to be the general consensus of opinion, that when once the attack has commenced the patient should retire to bed and keep the room dark. He

should adopt the attitude which gives most ease and if there is any feeling of chilliness, a hot-water bottle should be obtained and he should place this at his feet. Various remedies may now be tried, such as a full dose of brandy, which has occasionally been found to disperse the attack. Others again recommend large doses of Bromides XX-XXX grs. during the "blind" stage, upholding that this will disperse the attack. Strong tea or coffee, without milk or sugar, have also been recommended, especially in the milder forms. In virtue of the fact that in some people vomiting seems to terminate the paroxysm, the administration of emetics, such as Vin. Ipecac, &c., have been advocated. Trousseau believes that the severity of the seizure may be lessened by rubbing equal parts of cerate and extract of Belladonna on the temples, others again, on the assumption of the vaso-motor theories, have advocated the use of amyl nitrite, sodium nitrate, trinitrini, &c. and lastly, large doses of phenacetin, exalgin and citrate of caffein have all been employed. In my own case these remedies have all failed. I have tried a strong dose of brandy on several occasions and have always regretted my action afterwards, for the vomiting was longer in starting and was aggravated during its incursion,

being continued over a longer interval. Strong tea or coffee had usually a similar effect. Pot. Bromide in large doses was unsatisfactory and although in some rare instances the attack was cut short, yet I felt miserable as a result and wished I had allowed the paroxysm to go on uninterrupted, when I would have felt better. Usually, however, Bromides only delayed the onset of the symptoms, so also phenacetin in large doses and other remedies failed. A year or two ago I was senior resident in the Dumfries Infirmary and whilst occupying that position, I was laid up with what the senior physician said was a rheumatic tonsillitis. I was fevered and had difficulty and pain on swallowing. My throat was painted regularly and other treatment carried out. Aspirin was ordered in X gr. doses thrice daily. Before taking the first dose of this drug which had not yet been brought to me, I saw that I was in for an attack of megrim. Luminous zig-zags appeared in the right eye indicating the onset of a typical attack. It was at this time that the aspirin arrived and I took the X grs. prescribed. About 15 minutes later the eye symptoms disappeared and no sensory disturbances came on, nor did any vomiting occur subsequently. I slept a little and awoke with a slight headache on the right side. I thought this was remarkable and was determined to try the

effect of aspirin whenever the next attack made its appearance. I had about a month to wait and by this time the tonsillitis had quite disappeared and I was otherwise in perfect health. The attack started with itchiness of the lids of the right eye and the disturbance in the fingers already described and then the eye symptoms of luminous zig-zags developed. It was at the time that these had developed that I was enabled to get aspirin. I took grs. XV. this time and lay down in bed with the room darkened. Again the eye symptoms disappeared and no sensory disturbances, nor vomiting occurred. Headache developed on the right side, however, and persisted more or less throughout the remainder of the day. I was now determined to try aspirin if possible before the onset of the eye symptoms, and at my next attack I was able to administer the drug in a paroxysm which was beginning with the sensory disturbances as evidenced by the impaired movements of the fingers on playing the piano. Again, the prompt administration of the drug had cut short the attack, for no eye symptoms nor tingling of the fingers with numbness occurred, nor did vomiting nor speech disorders set in. On another occasion I did not take aspirin until both the eye symptoms and sensory dis-

turbances persisted for some considerable time. On this occasion the eye symptoms and sensory disturbances did not abate and vomiting occurred just as if aspirin had not been taken, in fact I thought the vomiting was rather more protracted. Sleep, as usual, terminated the attack. I now experimented with XV grs. doses of salicylates, and in my next seizures and found that they too, if taken early enough might arrest a paroxysm. They were not so efficacious in their action, I thought, as the aspirin. I now proceeded to try these drugs on others. The first patient was a Mrs. S. age 42, who had suffered from hemicrania for many years, confined to the right side and sometimes preceded by luminous zig-zags in the eye. She had been treated by various medical men and had taken phenacetin, exalgin, citrate of caffein, and other drugs without avail. Aspirin in X gr. doses gave her great relief. There was a history of rheumatism in her case. And so it was in many cases which came under my notice. There were others in which no relief was obtained, but in such cases, one generally found that no rheumatic history could be elicited. In some cases where the eye symptoms were followed by sensory disturbances and vomiting with headache, no good resulted, but here

again a rheumatic history was not forthcoming. In most of the cases I tried the drug on, the results were satisfactory. In the cases I will append there was a history of rheumatism in every one, and in all aspirin was of benefit. I have myself taken aspirin regularly for a month on end, at a time when attacks were prone, and during that time no attack of megrim occurred, but I have not so far attempted a prolonged use of the drug, owing to its depressant action on the heart.

This now brings me to consider the treatment to be adopted, between the paroxysm. The first thing to observe here is to obey the hygienic laws and the natural laws necessary for health. Look out for any abnormality, such as anaemia, chlorosis, &c, &c., and if such errors occur, let them be speedily corrected. See that the patient lives in a good healthy home. In professional people excessive brain work is a frequent excitement, as is also insufficiency of sleep. In such cases, rest from work, change of surroundings and removal of anxiety are bound to lessen the advent of the paroxysms. See that the individual is not leading too sedentary a life, and if so, insist on plenty of out-door exercise without at the same time

fatiguing the body. Of drugs suitable, many have been tried, and amongst those holding most favour have been sedatives, such as Belladonna, Hyoscyamus, and Bromides. In those instances where there exists some impairment in the nutrition of the nervous system, tonics must be prescribed, such as Iron, Strychnine and Quinine. Iodide of Potassium in virtue of its specific action has been given pretty widely, apart from its anti-syphilitic action and sometimes with good result, but it has no effect on me. Valerian and Valerianate of Zinc have been greatly lauded by Liveing and others, but I can give no opinion of them for I have never tried their effect.

One must also take good care to ensure the removal of all exciting causes. The stomach, in a state of disorder, has been admitted to be an excitant in some cases, therefore careful attention to the diet is necessary. I find distinct improvement from taking my food (which must be nutritious and easily assimilated) regularly and also by seeing that the bowels are kept in sound order. The emotions must be controlled as much as possible and unnecessary excitement avoided. Late reading, straining of the eyes and bodily fatigue must all be carefully relegated to the background. There is nothing like healthy open-air exercise without

fatigue. It is a fact that I have often observed, when a student, that the paroxysms seldom come on during the summer months, when the brain work and anxiety for the time being were stopped. The weather may also have had some influence here, as I have already remarked. I do not for a moment say that anti-rheumatic drugs will be successful in every case, but in a disease of this nature, which exhibits so many caprices, I think such drugs as salicylates or aspirin worth the trial especially in those cases in which a distinct rheumatic diathesis can be made out.
Another form of treatment which was greatly lauded some time ago by Whitehead of Manchester and reintroduced recently by a discussion in the British Medical Journal (Dec. 7th 1907, by Dr. Eldon Pratt, May 11th 1907, Wilson Parry, and Jan. 25th 1908) is that of the insertion of an ordinary tape seton, in the back of the neck and worn for three months on end. Cases have been recorded to have been cured by such treatment, but in others the condition has been aggravated.
Wilson Parry (B.M.J. May 11th 1907) applied the seton treatment in Menières symptoms and that he thought it acted by causing a counter irritation which had a direct action on the vaso-motor.system of the affected

side. He considered that Meniéres symptoms are due to hyperaemia of the vessels of the labyrinth and that the seton acting continuously for a prolonged time will by reflex action, influence the vaso-motor nerves of the affected part when in near proximity to it and thus correct a chronic vaso-dilatation of those vessels into one of normal tone." We have seen how the vaso-motor symptoms in megrim are secondary to some central stimulus, therefore this form of treatment does not appeal to me, as it aims at curing the result and not the cause of the condition. I will now bring together the cases of which I have kept a record. I regret that I allowed many patients whom I have seen and treated, especially those who only suffered from headache, without the other features of the paroxysm, to pass through my hands without comment. One or two of the cases have already been discussed, but a brief allusion will be made to them again here, in order to bring them together, side by side.

Case I. Mrs. S., age 42, suffered from severe headaches of an hemicranial character, all her life. She occasionally had eye symptoms consisting of luminous zigzags. There was a distinct history of rheumatism. She believed her headaches were worst during the catamenial periods. She had been treated by Bromides,

phenacetin, citrate of caffein, and other drugs without avail. Her condition was markedly improved by the administration of aspirin.

Case II. Miss Bridgford, aged 12, had had a fall two years ago with a resultant concussion and since that time has suffered from megrim. There was no history of heredity, but a history of rheumatism in the mother was elicited. Her attack started with ocular manifestations of the ordinary type in the right eye, but she had not noticed that any feeling of itchiness of the lids occurred prior to the onset of the attack. On the disappearance of the eye symptoms, tingling and numbness occurred in the right hand and arm. No speech disorders ensued. Her face was flushed, especially on the right side. I examined the fundus of the right eye and found the central artery and vein dilated. The left eye appeared normal. I did not notice any alteration in the size of the pupils. Vomiting subsequently occurred and sleep terminated the attack. I saw her too far on in the seizure to give her any drugs, but since then she has been put on a course of aspirin. It is just under two months since I saw her, so no conclusions can be drawn in this case. I have already referred to the fact of her

accident and its relation in respect of a cause of the malady. It would be interesting to learn if any other cases could be put down to accident as the starting point of the seizures - comparable to Jacksonian epilepsy. I examined her fundus after the attack was over and found it to be no longer congested.

Case III. Mr. B., a clergymen, had suffered from megrim for a long time. There was a history of heredity and he also at times complained of lumbago. His paroxysm usually occurred on a Monday after the strain of the Sunday's work. The attack started with luminous zig-zags in the right eye, which lasted about twenty minutes and then severe headache set in on the right side. He had noticed that sometimes he would rub his eye before the onset of the seizure. He found that by taking "salts" on the Monday mornings, he was often prevented from having an attack. He tried aspirin and found it very beneficial.

Case IV. Mrs. Smith, age 45, had suffered from megrim all her life. In many instances tingling in the right hand commenced the paroxysm and this spread up the forearm and arm. About fifteen minutes later eye symptoms developed and persisted for about twenty minutes, then nausea came on, which was followed by

vomiting and sleep terminated the attack. Her seizures usually came on during the menses. In other cases, her attack was ushered in by eye symptoms followed by tingling and disorders of sensation. She told me that if she were knitting which she could quite easily do in the dark or with her eyes shut, when the paroxysm came on with eye symptoms, she could no longer knit owing to failure of the right hand and the tingling and numbness subsequently developed. She could not remember noticing an itchiness of the lids previous to the onset of the paroxysm. Here, then is an instance which I consider comparable to the loss of movements in the fingers in my own case, when playing the piano. There was a distinct history of heredity but a rheumatic element was doubtful. I prescribed aspirin which she was to take before her next attack had fully developed and she said the drug had greatly lessened the severity of the attack. I have ordered her to use aspirin on other occasions and note the result.

Case V. Mrs. T., age 55. Fish Curer. I have already described this case fully. She had noticed that sometimes an itchiness of the lids occurred before the onset of the eye symptoms. She had a distinct rheumatic history. She had also noticed that the smell of certain substances was very prone to bring on

a paroxysm, just as I have observed in my own case regarding lobster. Her attacks have been much less frequent since her change of life. I regret that time has not allowed for a fair trial of the treatment in this case.

Case VI. In Mr. A., age 60, already discussed, there was a distinct history of heredity, he having suffered from attacks of what he called biliousness for many years. Some months ago he was suddenly seized with an attack of diplopia, accompanied by confusion of mind and disordered speech. The attack passed rapidly away and next day he was markedly improved. I believe that in this instance, this was simply an attack of megrim from which the other symptoms of the paroxysm were omitted. He had never suffered from disorders of speech in any previous attacks. I think that such cases as these, in which only one, and that a late one, of the symptoms of a paroxysm occur can be readily explained by the action of a toxine circulating in the blood. This patient too had lived abroad for many years and at one time had suffered very badly from malaria, although for many years he has been free from the disease, although the blind sick headaches continued. I think this also in favour of a toxic theory of production in this instance. I again regret that the occurrence

of this paroxysm has been of too recent a date to allow of an opinion as to results of treatment.

Case VII. Miss P., age 32, had a history of rheumatism, but no history of heredity. She periodically suffered from attacks which were ushered in by the development of eye symptoms. She describes the luminous zig-zags occurring in the lower part of the field of vision, where also occurred the area of inhibition. These eye symptoms lasted for twenty minutes and then the headache developed. Here aspirin made a marked improvement, and if taken early prevented the onset of the headache.

Case VIII. Miss Mac W. age 27, was a lady missionary, whom I attended when in the capacity of Ship's surgeon. She was invalided home on account of severe attacks of malaria accompanied at times by headache and often by nausea and vomiting. She was carried on board at Calcutta harbour, and when I saw her, her temperature was 103.4. Her tonsils were red and congested, there was a fine tremor noticed in the hands, but no evidence of thyroid enlargement. She told me that she had been the subject of relaxed throats for many years. She also suffered from fainting turns from time to time. On enquiry, I found that she had suffered from typical

blind headache accompanied sometimes by disorders of sensation followed by nausea and vomiting, long before she went to India, and that these attacks used to come on, usually about the time of occurrence of the menses. On the theory of malaria, I continued her treatment by Quinine, which she had been getting previous to her embarkation. This did not seem to do much good, her temperature continuing high and swinging a good deal. Next day she had a typical migrainous headache, with eye symptoms and I discontinued the Quinine and put her on X gr. doses of aspirin thrice daily. Her throat was still painful and I gave her a gargle of alum. I saw her in the evening when I found that the temperature had fallen to 98.6° and her headache had disappeared. Next day, November 14th 1905, I was called to see the patient at 6.30 a.m. She had had an attack of epitaxis accompanied by a fainting turn. Her pulse was rapid, weak and intermittent. No enlargement of the heart was found and the heart sounds were closed in all areas, with a slight tendency to reduplication in the mitral area. Her temperature was 101.2 I ordered sips of brandy every half hour until further notice. In the afternoon she developed another turn of sick headache and I gave her more aspirin which

seemed to lift the headache in the course of an hour or two. In the evening she was very much better. Next day, November 16th, her temperature was normal and three days later, she was on deck and rapidly gained strength during the rest of the voyage. She left the ship in London quite a different person. I may say that the voyage was remarkable for calmness and no one suffered from sea-sickness. Miss Mac W. further told me that she was a good sailor and had never been sea-sick. I believe in this case that the malaria superadded to by an hereditary tendancy to megrim and also the rheumatic diathesis, which the Quinine of itself was unable to disperse was the cause of the onset of the paroxysms. The aspirin did her a great deal more good than the Quinine which she had been taking for a considerable time previous to her coming on board ship.

Case IX. This is my own case which I have already fully described in its seven different forms. I will not again relate here, but would like to conclude by finally drawing attention to the many peculiarities observed therein. It would be interesting to note if many others are sufferers like myself, in that the paroxysm may be ushered in by speech disorders at one time and at another by visual disturbances. The early premoni-

tions and the after prodromes, I have now observed so frequently that I have felt compelled to mention them and also the frequent occurrence of a rheumatic element in many cases. At the final examinations which I sat for four years ago, one of the questions set was Megrim. In my paper on that day I mentioned, I think, the disturbances observed in my fingers on playing the piano. It is not once but many times that I have noticed such occurrences. Again regarding treatment I think the success of aspirin in my own case has warranted my drawing attention to the drug in this paper. It would be interesting to find out if others have found success in treating the paroxysm on these lines. It is on the principle, that little may lead to much, that has induced me to mention such otherwise insignificant points as I have related, occurring in a disease from which so many able men have suffered and who have left behind them such brilliant records of their paroxysms. Moreover, it is a disease which frequently attacks members of our own profession and in whom sound health is of the utmost importance. Even for such to be off duty for one day, especially without warning, is a serious consideration, and, therefore, any remedy which may even modify the seizure will be welcomed by such.
