OBSERVATIONS ON PERSISTENT AND RECURRENT HEADACHE WITH SPECIAL REFERENCE TO MIGRAINE.*

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PART II. MIGRAINE.

Migraine may be defined as a periodical and paroxysmal affection consisting of headache—unilateral, localised or general—which is frequently preceded or accompanied by characteristic visual symptoms and may be associated with nausea and vomiting. This definition applies to the average case but there are many mild and incomplete forms as well as others which are more profound and severe. An attack may last from an hour to several days and the actual paroxysm may differ in degree and complexity in the same person. It may also be said that any one of the classical symptoms—headache, visual phenomena and vomiting—may be entirely absent.

Areteus of Cappodocia, who flourished during the second century A.D., appears to have given the first description of migraine. The condition was also commented on by Celsus (*circa* A.D. 180), while Cælius Aurelianus—a Nubian who lived during the 5th century—noted that the Greeks termed the disorder “hemis-*crania*.” Almost ever since this time, the literature of the subject has been rich in descriptions of the symptoms by medical writers who have suffered from the malady.

Statistics indicate that in 75 per cent. of cases of migraine the first attack appears before the age of 20. After this age the incidence of onset progressively diminishes in each subsequent decade and it is rarely one meets with cases in which the onset occurs over the age of 40. As regards sex, females are more often affected than males; statistics vary from a proportion of 5:4 to 7:3.

As a rule, a family history is readily obtainable and quite frequently the condition shows direct transmission. C. K. Mills, for instance, reported a family in which migraine could be traced through five generations. Others have considered that the extreme prevalence of migraine renders conceptions regarding its hereditary nature a little doubtful. Nevertheless, in any considerable experience of the disorder, one cannot fail to be impressed with the hereditary tendency.

Etiology.

Migraine is not a disease *sui generis* but a symptom-complex. Various theories have from time to time been put forward to account for its phenomena. Migraine certainly appears to be a condition in which there are two main factors (1) An inherited constitutional predisposition—the *migrainous diathesis*—and (2) The actual disturbance (the attack) which is produced by one or more of a *variety of stimuli* acting through the sympathetic nervous system. These stimuli include (a) *Physical*—changes of altitude, temperature, fast movements, diving, trauma, etc. (b) *Chemical*—alcohol, nicotine, and toxic substances. (c) *Allergic states*—protein and other idiosyncrasies. (d) *Endocrine*—thyroid, pituitary and gonadal (especially ovarian) disturbances. (e) *Somatic reflex causes*—fatigue, eyestrain, alimentary disorder. (f) *Emotional or psychical*—excitement, fear, anger, etc. In some emotional states, the stimulus may be of endocrine or chemical origin through excess of adrenalin in the circulation or by the produc-

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tion of an acidosis. Thus we may account for the different forms of migraine described in the literature under the headings of "endocrine" or "ovarian" migraine, "bilious migraine," etc.

The stimuli acting through the sympathetic nervous system—probably hypothalamic centres^(*)—produce a vaso-motor disturbance (views as to whether vaso-constriction or vaso-dilatation is the initial factor have varied with different authors) with subsequent localised cerebral hyperæmia and possibly transient œdema (resembling angio-neurotic œdema) which result in the visual symptoms and, at times, such phenomena as ophthalmoplegia, hemianopia, aphasia and more rarely transient hemiplegia.

It must be admitted that the headache itself is referable to the meninges as the actual brain substance is insensitive. Also, it seems probable that the initial occurrence is a vascular spasm—intense vaso-constriction of both meningeal and cerebral arteries. Following the vaso-constriction there occurs an equally intense reactionary vaso-dilatation. In the meningeal arteries such vaso-dilatation would account for the headache with which E. Bramwell (1936) has compared the throbbing pain which comes on gradually and may persist for some time after the gum has been frozen or in a hand benumbed with cold when it is plunged in warm water. Vaso-dilatation and engorgement of the cortical vessels might well be followed by a transient œdema which would account for the visual phenomena (in the occipital cortex or chiasmal region), the transient aphasias and even the occasional pareses. This view is further supported by the observation that a distinct period of time often elapses between the disappearance of the prodromal symptoms (stage of vaso-constriction) and the beginning of the subsequent headache (usually throbbing in character) and other symptoms. The migraine attack can thus be accounted for by a localised vaso-motor spasm and subsequent vaso-dilatation (relaxation) of the cerebral or meningeal arteries (or of both). The prodromal symptoms result from spasm of the arteries while the headache and other symptoms are due to the re-establishment of the circulation in and engorgement of the meningeal arteries. In this connection, it is of interest to note that physiologists have at last admitted what clinicians have long contended—the existence of a vaso-motor mechanism in relation to the cerebral arteries.^(*)

Whatever the primary stimulus, it would certainly appear that the actual attack is associated with distension of the cranial arteries as cessation of the headache frequently results from the injection of ergotamine tartrate which acts as a vaso-constrictor.

According to some observers e.g. G. Holmes, the symptoms of migraine are more easily explained by the assumption of a localised swelling of the brain due to a local and temporary alteration in the osmotic relation of its substance with that of the cerebrospinal fluid in which it is bathed; the result is an imbibition of fluid and consequently a localised swelling. That such swellings do occur is known from the work of Reichter and others. If the occipital lobe were affected, the positive and negative visual symptoms of migraine would appear—teichopsia and scotomata—while the swollen brain pressing on the meninges would cause the headache.

^(*) The hypothalamic control of the cerebral blood-vessels is described by STAVRAKY, G. W., with numerous references in the *Archives of Neurology & Psychiatry* (1936), 35, 1002, "Response of Cerebral Vessels to Electrical Stimulation of the Thalamic and Hypothalamic Region."

^(*) See PENFIELD, W., *Archives of Neurology & Psychiatry* (1936), 36, 449; also CHAROBSKI, J. and PENFIELD, W., "Cerebral Vaso-dilatation Nerves and their Pathway from the Medulla Oblongata" (1932), *ibid.*, 28, 1257, and STAVRAKY, G. W. (1936) *ibid.*, 35, 1002.

The "pituitary hypothesis" has its attractions. The proximity of the pituitary body to the cavernous sinus with its adjacent sympathetic plexuses; the periodicity of the affection; the occasional changes detected in the sella turcica; the frequency of ophthalmic symptoms and the occasional response of pituitary therapy are factors cited in favour of this view.

Timme (1926) postulated a special type of migraine—"pituitary migraine"—which he considered the result of temporary swelling of the pituitary body. The headache was temporal or bi-temporal in distribution and "bursting" in character. The veins of the fundus oculi might be found distended during the attack and frequently vision was disturbed; ptosis, hemianopia, diplopia, strabismus, and even trigeminal neuralgia might occur. Polyuria was also sometimes present. Patients suffering from this type of migraine frequently showed other signs of dyspituitarism—erosion of the clinoid processes or of the sella turcica on X-ray examination, as well as occasional acromegalic features. The probable explanation is that given the "migraine diathesis," pituitary disorder may well act as a stimulus.

The frequent coincidence of migraine attacks with menstruation and their undoubted remission during pregnancy and lactation are mentioned in support of an "ovarian theory." Here again, ovarian influences can undoubtedly supply the stimulus.

Spitzer's hypotheses is that of congenital stenosis or narrowing of the foramen of Monro as a basis of the migraine constitution. During an attack it is assumed that the foramen becomes plugged by a tag of congested choroid plexus. In this way the intracranial tension is raised, secondary ventricular dilatation with temporary hydrocephalus occurs, and the cerebral cortex is pressed against the unyielding dura mater causing at first the premonitory symptoms and then headache.

Symptoms.

Few pathological conditions are more protean as regards symptoms and course, and there is often an extreme variability of individual attacks even in the same patient. Quite frequently, there is a prodromal stage of chilliness, pressure sensation in the head, moodiness, somnolence, dizziness, inability to concentrate, anorexia or occasionally nausea and pallor of the face.

The headache may be unilateral, supra-orbital, vertical or general. Visual symptoms that may occur are the common "floating spots" (*muscae volitantes*), fortification figures, flashing or revolving lights (*teichopsia*), scotomata, hemianopia and diplopia. Scotomata may occur without the headache and vomiting. Among the more general symptoms of an attack are chilliness, *paræsthesia*—either general or unilateral—vertigo, tinnitus, transient aphasia and amnesias, flushings, depression, neuralgia, nausea and vomiting and even transient monoplegias and hemiplegias. I have also seen a case with nystagmus, facial palsy and fifth nerve involvement—in fact all the signs of a cerebello-pontine angle tumour—which recovered within two days of the cessation of the attack.

Definite ocular physical signs, apart from or in addition to hemianopia, are sometimes present. For instance, the pupils may become unequal, one being widely dilated. Such unilateral dilatation may persist for some days after the attack has passed and I have known such unilateral pupillary dilatation be the only manifestation of an attack. The retinal appearances during an attack have been variable. Blanching has been observed by some and pulsation of the retinal arteries with

dilatation by others. I have at times seen similar dilatation in some cases but as a rule have found the fundus normal. Definite strabismus may occasionally occur (usually a transient external rectus palsy); in such cases every care should be taken to exclude organic disease.

One fact is not generally appreciated, that is, that in those subjects showing pupillary inequality during an attack, such inequality will in the course of time, with repeated attacks, become permanent and the patient present a moderately small pupil on one side and a dilated one on the other.

Course.

The course varies considerably in different cases. As a rule the patient is quite free from headache during the intervals. Occasionally a dull headache may persist for a few days after the attack and merge into the next attack if these are very frequent. Although the attacks often cease towards the end of the fourth or fifth decade, this is by no means the rule as migraine may continue throughout life. As stated previously, during pregnancy there is often a complete remission or at least a lessening of intensity, some symptoms disappearing while others remain.

I have already referred to a type of chronic and continued headache with exacerbations of severity (Part I); these latter may or may not be accompanied by visual and other symptoms. This condition I have come to regard as a variety of migraine. It has already been stated that in the more usual type of case, a dull headache may continue for a few days after an attack of migraine. In the cases to which I now refer, the dull headache appears to continue throughout the interval between attacks. The following three cases are examples.

Mrs. D. H., aged 23, was first seen in March, 1936 with a complaint of headaches occurring almost daily, varying in intensity and of bi-temporal distribution. Occasionally the pain was more intense over the left eye and would then be accompanied by giddiness and tinnitus but at no time had she had any visual symptoms.

The onset occurred about a year previously. Six months later the tonsils were enucleated, following which the headache was completely absent for a period of one month. There were no previous illnesses of importance.

Physical examination was entirely negative; the cerebrospinal fluid was normal and X-ray examination of the skull showed no abnormality. Considerable improvement, but not relief, followed the taking of Gardenal grs. $\frac{1}{2}$ morning and night for the next three months. A course of peptone injections produced little, if any, further improvement. She was then put on Ergometrine (0.5 mg.) twice daily in addition to the Gardenal with the result that the headache, although still present, was rather less intense. By September, 1937, she was sometimes free from headache for as long as 14 days and exacerbations were less frequent, but in November they recurred with their usual intensity. Nevertheless, the same line of treatment was continued and she has been much better during the whole of this year.

G. M. S., male, aged 28, was first seen in April, 1933, on account of headache, more or less constant although worse during the evening and reaching an intense exacerbation usually once a week after the week-end.

The headache had been present for about eight years—since the age of 20. A refractive error had been detected at the age of 12 since which time he had worn glasses; these had been altered from time to time. He led a very open-air life which did not seem to affect his headache but exacerbations would occur following the taking of any

alcohol. Three years previously he had had his tonsils enucleated and had experienced no headache for as long as six months; following this, however, the headache returned with its usual intensity. A characteristic feature was that following the week-end—living in the country he is more in the open air on Saturday and Sunday—the headache on Mondays was much more intense and of fronto-occipital distribution. All manner of treatments had been tried, including various sedatives, ergotamine preparations, peptone injections, autogenous vaccines and even a course of psychotherapy, all without producing any appreciable result. Finally, in October, 1936, he reverted to Bellergal taken regularly morning and evening, and the present position is that the headache troubles him very little during the week but is still more intense on Mondays.

Mrs. J. H., aged 25, had complained of headache since the age of 15. The headache is stated to be almost constantly present, even in the morning, and usually increases in severity during the day. It occasionally becomes worse and is then associated with visual symptoms consisting of fine lines or floating spots, also nausea but no vomiting. These exacerbations may result from excitement, smoking, alcohol or sudden rotation of the head.

The only points of importance in the previous history were (1) At the age of 12 she fell, striking the occipital region of the head. She was amnesic for about an hour but showed a normal recovery without either persistent headache or giddiness. (2) A long history of extremely obstinate constipation.

Physical examination showed no abnormality beyond a few nystagmoid jerks of the eyes on lateral deviation. The eyes had been examined four to five times and she showed no refractive error. Cerebrospinal fluid was normal in all respects, including total protein of 0.025 per cent.

In the first two of these cases one is very tempted to consider a "toxic" (probably streptococcal) stimulus, in that the removal of infected tonsils in each case was followed by a comparatively long period of freedom. In the third case obstinate constipation was a feature and it is hoped that improvement in this factor may afford some relief from the headache.

Relation to Epilepsy.

Various attempts have been made to connect migraine with epilepsy. Each condition is characterised by periodicity, by a paroxysm and often by an aura and by drowsiness after an attack. One meets with instances in which after some years of migraine, epilepsy has developed; similarly epilepsy is sometimes replaced by migraine, while in other cases attacks of migraine are interspersed—acting as "epileptic equivalents"—between attacks of true epilepsy. Also, epilepsy may occur among the relatives of a migrainous subject. Both disorders, it must be remembered, are comparatively frequent. Consequently when migraine and epilepsy occur in the same family due allowance must be made for the possibility of coincidence.

The term migraine-epilepsy has been applied to that form in which unconsciousness—with or without convulsions—occurs at the height of the migraine attack.

Diagnosis.

The diagnosis of migraine rests upon (1) an accurate history of periodical attacks of the symptom-complex already described, bearing in mind that any one or more of the classical symptoms—headache, visual phenomena and vomiting—may be absent and (2) the exclusion of organic disease. As regards differential

diagnosis I have already referred to the necessity for excluding cerebral tumour and sub-arachnoid hæmorrhage from a leaking cerebral aneurism (Part I). In addition, I have met with several examples of a condition closely resembling "migraine ophthalmoplégique," and which one can explain only by assuming the presence of an intermittent form of arachnoiditis or endymenitis. As examples, the two following cases may be mentioned.

Mrs. J. D., aged 49, was first seen in August, 1929, when she complained of an illness which began in April of the same year with headache and vomiting. Headache appeared first and was paroxysmal and generalised, the attacks lasting an hour or two, and the pain being described as being "bursting" in character. During this illness she had several attacks of transient weakness affecting the left arm and leg. These symptoms lasted acutely for three weeks and then passed off, but she continued to suffer from paroxysmal headaches. In August, 1929, the acute symptoms recurred with great severity. Headache and vomiting lasted for one month. The left arm and leg became weak and sensation was impaired on the left side for short periods of thirty minutes at a time. Similarly, transient attacks of loss of speech occurred frequently. Left-sided weakness diminished and was succeeded by weakness and impairment of sensation in the right arm and leg. During this month the patient suffered for a fortnight from diplopia but without definite strabismus.

She had suffered at intervals, since the age of 14, from acute attacks similar to those described above, being perfectly well in the intervals. These previous attacks were of varying severity lasting from one to three weeks and all were accompanied by short attacks of left hemiparesis and aphasia. They occurred at intervals of two or three years but had become more frequent during the last seven years, during which period, most of the attacks, when at their height, have been associated with loss of consciousness lasting up to twenty-four hours. Visual hallucinations have been present during all the acute attacks, but chiefly when the patient was in the dark or when her eyes were closed. A carpet would unroll itself towards her and smother her, or someone appeared standing beside her, etc. Vertigo was marked during all phases. Since April, 1929, mistiness of vision had been present in the left eye.

Physical examination (January, 1930). Cranial nerves: Pupils moderate in size, equal and regular. Pupillary reactions normal. Extra-ocular movements normal. (On September, 3rd, 1929, there had been difficulty in conjugate deviation to the left.) Vision: Right 6/5, left 6/6 with mistiness. Discs: in August, 1929, there were three dioptries of papilloedema in the right eye with hæmorrhage into the optic disc. This papilloedema has now diminished but the disc is reddened to the colour of the retina and the edges are blurred. The left disc is pale. Fields: Left, normal; Right markedly contracted, particularly in inferior nasal quadrant. Corneal reflexes, present and equal. No facial paresis. Hearing and speech normal. Other cranial nerves normal. Sensory system normal. Motor system: trunk muscles normal. Power slightly diminished in right arm and slightly impaired in both legs. Tone, range of movement and co-ordination normal. Reflexes: abdominals absent. Tendon reflexes present, not exaggerated and equal. (A few months ago the knee and ankle jerks were exaggerated on the left side.) Plantars flexor. No Rombergism. No sphincter incontinence. Gait: a little uncertain.

She was not seen again until 1934, when she was re-admitted to hospital with severe frontal headache, pain through both eyes and vomiting. On examination, she showed definite swelling of both optic discs amounting to about 1 dioptre each. In addition, there was partial paresis of both external rectus muscles and the right plantar reflex was extensor.

The cerebrospinal fluid was normal with total protein 36 mg. per cent. Following lumbar puncture she made a good recovery. A further attack occurred in August of the same year with severe headache, vomiting, blurred vision and diplopia towards the right. On this occasion there was no papilledema, the only abnormal physical sign being slight weakness of the right external rectus. Again relief occurred following lumbar puncture. I have seen her from time to time during the past four years and no further attacks have occurred.

Clearly the patient has suffered from an intermittent rise in intracranial tension at intervals for many years. When first seen the diagnosis of cerebral tumour was naturally entertained. None of the symptoms and signs, however, suggested definite localization of such a lesion. A tumour could scarcely produce recurrent attacks of raised intracranial tension, with perfect health in between over so long a period. The explanation that best seems to meet the facts is that of recurrent attacks of acute hydrocephalus.

The diagnosis that suggests itself is that of a chronic remittent form of arachnoiditis—a condition which may closely simulate cerebral tumour. The cause of this condition has been ascribed to a chronic ependymitis—either causing serous effusion into the ventricles or obstructing the iter or medullary foramina. Hypertrophy of the choroid plexuses is another possible cause.

F. B., male, aged 40, for the last five years has suffered from recurrent and severe headaches. The first headache began suddenly one evening and was so severe that he was afraid to move his head; it lasted three hours and then passed off leaving a duller type of headache which continued for four or five hours. Headache of similar type has recurred every few months; with some he has collapsed and vomited and has also complained of diplopia.

In September, 1938, he was seen during an attack. He complained of severe occipito-vertical headache which was greatly aggravated by any movement of the head. Fine lateral nystagmus was present, both to the right and to the left; this nystagmus is persistent and is also seen between the attacks. The edges of each optic disc were indistinct and blurred (between the attacks they are normal) and he showed slight right-sided facial weakness, diminution of sensation to pin-prick over the area of distribution of the right fifth nerve and right-sided palatal weakness. The deep reflexes on the left side were somewhat brisker than those on the right and the left plantar reflex was indefinite, the right being flexor. The blood pressure was 135/85 and other systems were normal. The cerebrospinal fluid was under slightly increased pressure and was obtainable in excess; on examination the only abnormality found was slight excess of protein (0.06 per cent.).

On the day following lumbar puncture, the headache had disappeared and also the abnormal physical signs with the exception of nystagmus.

Occasionally slight difficulty is experienced in distinguishing migraine from a Menière syndrome, especially if vertigo is one of the migrainous symptoms and the headache comparatively slight. The co-existence of vertigo with tinnitus and a definite degree of deafness (usually of nerve type) in one or the other ear, will differentiate the case as one of Menière syndrome.

Treatment.

The treatment of migraine taxes to the utmost the ingenuity of the physician. As one would expect from the etiological factors enumerated previously (stimuli), no one remedy offers relief in all cases and different cases may show improvement

on different forms of treatment. The "migrainous diathesis" exists in very varying degrees; in some cases a very slight disturbance is sufficient to precipitate an attack while, in others, a much greater stimulus is required. If the vasomotor hypothesis is accepted, it can be readily understood that the removal of various forms of irritation may result in eliminating some of the causes (stimuli) which activate the migrainous reaction. Thus, the relief of eyestrain, adenoids, constipation, dysmenorrhoea, etc. may benefit a certain number of patients. This explains why some are relieved by such widely differing measures; for instance, one woman who was treated by various methods over a number of years without benefit was practically cured by the removal of her appendix, while many other migrainous subjects I have known suffer an intercurrent attack of appendicitis and undergo appendectomy were in no way benefited.

Treatment of the attack. In many cases an injection of ergotamine tartrate (Fermegin) given early in the attack will relieve the headache and shorten the paroxysm.

M. E. Sullivan (1936) investigated the relative effectiveness of several non-sedative remedies in relieving the actual migraine attack, including caffeine, histamine, adrenalin, ephedrine, mecholol, amniotin, tissue extract, pitressin, amyl nitrate, calcium gluconate intravenously and ergotamine tartrate. Of all these, the latter was the only one which gave definite and constant results. It completely checked 34 headaches in 14 patients and failed to relieve only five headaches in four patients. In a further series of 97 patients, all but eight were benefited by this treatment—it completely checked 1,042 attacks in 89 patients. The initial dosage should be 0.25 mg. subcutaneously, this amount often relieving the attack within two hours. If after two or three hours the attack persists, this dose should be repeated and also if the attack returns. If repetition has been necessary an initial dose of 0.5 mg. should be given for future attacks.

Graham and Wolff (1938) have also analysed the results in 32 attacks of migraine in 16 subjects. They concluded that relief from headache depended upon vaso-constriction of the cerebral arteries. One direct observation in man, on the middle meningeal artery carried out during a craniotomy, showed a decrease in the diameter of the artery of approximately 20 per cent. following the intravenous injection of 0.5 mg. ergotamine tartrate.

Any untoward effects—nausea, weakness of the legs, joint stiffness, heaviness in the chest, uterine colic or tingling of the extremities—may be relieved by the injection of gr. 1/100 atropine. In those patients showing any of these symptoms, it is as well to give atropine at the same time as the ergotamine in subsequent attacks.

I have also found a severe attack of migraine greatly relieved by the intravenous injection of sodium luminal (grs. 3 in 10-15 c.cms. of distilled water). Injections of adrenalin (5 to 8 minims of a 1 in 1,000 solution) and of Impletol (Bayer)—a mixture of caffeine and novocain—have occasionally proved successful in aborting an attack.

Owing to the frequent presence of vomiting and especially as ordinary gastric peristalsis is inhibited during an attack, it is very seldom that any benefit results from the administration of remedies by the mouth. In cases in which there is no vomiting, however, it is worth while administering any of the following: Compral (Bayer) two tablets every three to four hours; Veramon (Schering) grs. 6 repeated in one hour; Dialacetin (Ciba) tablet repeated in three or four hours.

Preventive treatment (Treatment between the attacks). As regards attempts to prevent the occurrence of attacks, phenobarbitone in half-grain doses, taken regularly each morning and evening or in more severe cases three times a day, is a line of treatment which reduces the frequency and the intensity of the attacks in most cases. If a favourable result is not obtained with $1\frac{1}{2}$ grains of phenobarbitone per day, increasing the dose beyond this level is seldom helpful. Other cases may respond to the addition of one of the forms of ergotamine to the phenobarbitone e.g. Ergometrine^(*) Fermegin.

Bellergal (Sandoz) is a combination of ergotamine tartrate with phenobarbitol and bellafolin (parasympathetic sedative) in the same tablet. One tablet can be taken each morning and one or two at night.

When a pituitary factor is proved or suspected, the addition of whole gland desiccated extract (grains 2 morning and night) is often helpful.

In migraine that appears to have an alimentary-disorder stimulus the gall bladder is frequently blamed. The existence of migraine with gall bladder disease (gall-stones or cholecystitis) is by no means infrequent (it should be borne in mind that both are common disorders) but the results of removal of the gall bladder as regards benefit to the migraine have not been encouraging. Others (Hunt) invoke "subacute hepatitis."

In order to increase the threshold of resistance to sympathetic irritability and to minimise the liability to the migraine reaction, Dandy removed the first thoracic sympathetic and the inferior cervical ganglia. At first the results were said to be encouraging but observation on a larger number of cases has not justified the earlier optimism.

Finally, in very severe cases resort has been made to subtemporal decompression (by a small trephine opening). G. Holmes has said that he has never seen migraine persist in a patient who has had a decompression. I have known three cases in which this operation was carried out—not on my direct advice but rather with my passive acquiescence. In one case the result was excellent. The decompression was performed over 20 years ago and she has had no attacks since. A second case—a man aged 36 who suffered from severe attacks associated with ophthalmoplegia—has been free from attacks since the operation of six years ago. The third case was a woman, aged 26, with whom I lost touch about six months after the operation. During this interval (following the operation) she was known to have had three severe attacks.

In conclusion, I would say that in the treatment of migraine the general practitioner can often do more for an individual patient than the consultant. The latter can merely confirm the diagnosis and indicate certain lines of treatment on which trials can be made. The general practitioner will have more opportunity of contact with the patient and consequently of eliminating various stimuli that may be provoking the symptom-complex.

^{*} An oxytocic alkaloid isolated from ergot and given various names, including Ergometrine, Ergonovine, etc. According to the observations of LENNOX, W. G. (1938), *Amer. Journ. of Med. Sciences*, **195**, 458, ergotamine tartrate, when given by injection, is far more effective than ergometrine (ergonovine) in relieving the headache of migraine, while, taken by the mouth, ergometrine is relatively more efficacious than ergotamine tartrate, although both are less effective than when injected.

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