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Ronald F. Kirk
University of Nebraska Medical Center

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MIGRAINE HEADACHE

BY

RONALD F. KIRK

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COLLEGE OF MEDICINE

1933
Headache in itself is one of the commonest symptoms presented to the practitioner. Whether he be in general practice or in one of the many specialties of medicine a goodly percent of the patients coming to him for treatment will give in their anamneses the complaint of headache either as a minor or major symptom.

Auerbach (4) has stated that of the numerous patients who came to him complaining of headache, he was able to make a diagnosis of migraine in a large percent of these cases. He goes so far as to state that migraine headache is the commonest of all types of headache regardless of origin.

I have chosen for my topic this commonest type of headache. During my short medical contacts I have developed considerably more than a passing interest in the study of headache, and specifically headache of the migrainous type.

In the reading I have done on this topic I have been greatly surprised at the relatively small amount written upon this subject.

I have found in the current literature very few new plausible theories explaining the etiology of this condition and in fact have found many that have been proven purely fantastic.
The allergists at present are very enthusiastic over their results in certain cases. For some time this particular group of medical men have attempted to prove that migraine can be explained upon a basis of sensitization. Except for the new allergic possibilities of migraine there have been practically no new theories presented for a number of years. The writings I have covered in preparation of this work embrace rather generally the theories and hypotheses of some years past.

For example the views of Wright (26), Day (12) and Corning (10), whose writings on this topic appeared from 1871 to 1888, vary only slightly from those of our present writers.

It therefore seems evident that if the allergists fail to establish a definite basis for the etiology of migraine we will be in the same state of ignorance concerning the etiology of this condition as medical men were some fifty years ago.

By a rather complete canvas of past and current literature available to me I am attempting to present this subject in such a manner as to point out all possible angles.

I will give credit to the proper sources for any borrowed statements, ideas or cases quoted here in.

The Writer.
Migraine, sick headache, hemicrania and bilious headache are all synonymous terms used to describe a certain symptom complex in which headache is the commonest and most pronounced symptom.

This symptom complex may be said to be a recurring, paroxysmal, sensory disturbance conditioned on an inherent instability of the nervous system which reacts to various types of stress in a specific manner i.e. the migraine attack. Those afflicted have a definite migrainous hereditary background.

Migraine has long been recognized by medical men as a definite clinical entity. These paroxysmal attacks were first described by Galen, and as in many of his writings so with this condition he was far advanced beyond the medical knowledge of his age. His description of the paroxysm is remarkably correct and vivid. His explanation of the etiology being based upon swelling of the brain substance with subsequent pressure upon the cranial vault. It is from Galen we have received the name hemicrania.

For the occurrence and incidence of this condition we must rely entirely upon the diagnostic ability of men who have seen fit to maintain and report statistics. I have read several of such reports and have found that they vary only a few percent.
I am sincerely convinced that migraine is a far more common symptom complex than the average practitioner realizes.

In a report of Balyleat and Rinkel (5) they have stated that in the United States according to their records, two percent of all children under the age of twelve years suffer from migraine. In other words one million children present this symptom complex.

Considering the occurrence in all of the population of the United States the total is said to be seven percent.

By the result of comparative reports about thirty percent of these sufferers manifest their symptoms before the age of ten years. The vast majority of cases occur before the age of twenty five years and a very small percent of cases occur after that age.

A definite etiology of this condition is still disputed. There are however several known etiological factors which have definite bearing upon its incidence. The hereditary factor has long been recognized in the transmission of migraine. What might be expected from an affection that fulfils the law of segregation of characteristics is described by Buchanan (8) in an elucidation of the Mendelian phenomenon.

He shows that if a tall pea is crossed with the
dwarf pea the hybrid is tall. By breeding two tall hybrids, three tall and one dwarf are obtained. Of the tall, one always breeds true to tall, and two tall always breed three tall to one dwarf. The dwarf always breeds true to dwarf. This process is kept up indefinitely so that from each generation one pure dwarf and one pure tall, and two talls which have concealed within them the ability to produce both tall and dwarf are always obtained. The ratio of three to one is only approximate as Mendel in his observations found in a series of 1064 plants that there were 787 tall plants and 277 short or a ratio of 284 to 1. In a study of color of seed coats, Mendel found that among 929 plants, 705 bore violet red flowers and grey brown seed coats; 224 bore white flowers and white seed coats. This gave a ratio of 3.15 to 1. The exact ratio of 3 to 1 is rarely seen in plants or animals.

With the above examples in mind we proceed to a study of migraine as undertaken at the Mayo Clinic.

Study 1: Mother and father with migraine. This group comprised the offspring of 100 families made up on 143 children with migraine and 488 without migraine or a ratio of 3.13 to 1.

Study 2: Crossing of persons with dormant migraine.
Seventeen families were tabulated in which the migraine was not present in the father or mother of the family studied, but was present in brother, sister, father or mother of the parents of the family studied. In this group 30 children had migraine and 85 did not have migraine, or a ratio of 2.83 to 1.

Study 3: Parents with migraine. Three families were studied in which the presence of migraine in both parents was investigated. The total number of children born was fifteen, and as might be expected from the crossing of two pure types, all had migraine.

The total number of children in this study was one hundred ninety eight with migraine and 610 without migraine giving a ratio of 3.08 to 1. Thus this tabulation places migraine in the Mendelian ratio.

Smith's evidence (as quoted by Allan (21) is for the dominance of the trait. But his assertion that direct parental inheritance as a dominant trait, is disputed by Allan, who finds there will be a parental migraine in 100 percent of the instances if it is a dominant trait, or if headache is recessive, accepting the incidence as sixty percent, there will still be parental headache in 95 percent of the instances.

Allan's statistics are adducted from a review of
500 cases of migraine. By his equations it is shown:

(A) When both parents had migraine, the percentage of migraine in children was 83.3. (B) When only one parent had migraine the figure was 61 percent. (C) When neither parent had migraine, the percentage was 3.7. (D) With a "history" of migraine in one or both parents the figure was 91.4 percent. (E) When both parents should have migraine his figure is 26 percent. (F) With only one parent, 60 percent. (G) A history of migraine in neither parent is given as 8.6 percent. (H) In families of 376 migrainous patients containing 2105 children, 658 children had migraine, 504 did not have it, and in 567 the history was not known or the children were too young. This gives an incidence of 67.2 percent of the 1538 children studied. (I) In 163 family histories given by children who did not have headache themselves, 90 or 118 percent of a total of 911 children are said to have migraine, 722 to be without migraine, and in 99 the history was not known. The summation of statistical evidence points strongly to the following conclusions:

(A) That migraine in a child comes directly from migraine in one or both parents in 91.7 percent of all cases. (B) When both parents are migrainous 83.5 percent of the children are migrainous. (C) When one parent is migrainous 57 percent of the children are
migrainous. (D) It is also shown that without a history of migraine, the incidence is about eight percent. (E) Migraine is not a dominant characteristic.

As to the comparison of migraine in the sexes, on the whole, more women than men are attacked by the complaint but the difference according to Auerbach (4) is less than many authors have assumed. The proportion is about five or six to four, taking an average from the more carefully compiled statistics.

Position and occupation probably exert little influence, it is certainly not true that the so-called upper classes are notably more affected by the disorder. On the other hand, it is probable that brain-workers, in consequence of their mode of life in confined spaces and through lack of bodily exercise, are more frequently and severely visited by the attacks than are the members of the motor types whose business is in the open air.

Of the few cases I have personally contacted the individuals are of the more serious type. They seem to be abnormally active mentally and are invariably prone to allow themselves to worry over trifles and are easily excited and upset. A few cases I have known personally are the moody type. Their whole existence apparently runs in cycles of euphoria or depression to a degree.
I have found nothing written pertaining to any particular mental type which is liable to migraine paroxysms. My own observations as to this, being very limited, are probably in error.

Considerable has been written by Grimes (13) on the physiological and pathological instability of migrainous types. The instability, unexplained and undefined, on which migraine is conditioned is a constant factor, varying only in degree. It is this variation in degree of instability that perhaps accounts for its diversified manifestations. It is a graduation with the widest possible difference in the two extremes. At the upper limit it is as great as is compatible with life, at the lower limit it is the slightest possible degree. In all instances this instability is inherited. There is no acquired migraine.

It is evident that if the instability is great a slight stress is sufficient to initiate the attack, and when the instability is almost inconsiderable the stress must be profound in order to produce a reaction. It follows then, that a stress of the physiological kind is sufficient to cause a paroxysm in patients with a high degree of instability, while in those of low susceptibility the stress must be pathological. There are those who show a migraine response to ordinary physio-
logical stress of daily life, and those who do not respond except to the onset of disease. It follows that the greater the susceptibility, the earlier in life is the condition manifest. It may come on in infancy, at early school age, at the beginning of puberty, at any time when duties become more varied, on beginning of marriage responsibilities, or during the trials of a business career. The time of onset is determined by the degree of instability. Again, it may be occasioned by physiological stress, but come only as the result of pathological accident, or make its first appearance with the decrescence of age. In all cases the time and cause are conditioned on the degree of instability.

A great deal has been written upon the possibility of the part played by the endocrine system in the cause of migraine. As in many other unexplainable conditions we have looked to the endocrine system as a causitive factor. This is a possibility which must be considered but in my estimation a rather obscure one.

Those who support the endocrine theory have several excellent points in their favor.

Dr. Miel Steven's (24) in his studies at the Cornell clinic drew the conclusions that migraine lies deep seated in the germ-plasm, that it is an hereditary, constitutional disease and that it seems to be closely as-
associated with dysfunction of the endocrine glands.

His support of the endocrine theory lies based upon the facts, first in the age of onset, usually with the establishment of menstruation in girls and just before or at the beginning of adolescence in boys. It ceases, or changes its character in women at the menopause and it is rare to see it in men past fifty years of age.

Menstrual migraine according to Dr. Stevens is identical symptomatically with true migraine, except that it occurs only once a month and undoubtedly has some relation to the menstrual period. It is invariably associated with a scanty menstrual flow.

In a series of sixty cases of migraine in men and women, the basal metabolic rate was estimated. In the male group, half the cases studied (numbering ten) were below normal, ranging from minus 11 to minus 24. The rest of the males were within normal limits.

In the women the basal metabolic rate ranged from minus 3 to minus 16. The majority of these cases gave other evidences of hypothyroid condition, such as sub-normal temperature, sensitiveness to cold, fatigue, etc.

Dr. Stevens also claims favorable responses to treatment with glandular extracts. He says, assuming that hypofunction of the thyroid gland is responsible for
many cases of migraine, it might be permissible to suggest an explanation of the cause of an attack by outlining what occurs in the body during and following an attack of migrainous headache.

The thyroxin furnished by the thyroid gland regulates the metabolism of the body and is intimately connected with the nervous, circulatory and digestive systems. If there is an insufficient amount of hormone, in the body, processes are slowed, the mental processes are sluggish, the pulse becomes slow, the desire for food is diminished.

Is it not possible that the food consumed is incompletely burned and that the poisonous products of incomplete combustion accumulate in the system and when the limit of tolerance is reached, an attack occurs? An explosion occurs with its expression in the symptom complex of migraine, the thyroid undergoes a temporary stimulation under which, for a short time it puts out more than its ordinary amount of hormone, the bodily processes are stimulated (a temperature of 102-103 often occurs in a migraine attack), the toxemia is eliminated and the organism quickly returns to its normal state. (Patients often assert that they never feel so well as they do immediately following an attack). The thyroid relapses to its former condition of low output and the
cycle begins again, to end in a week or two in another explosion.

It may be asked why this is not the old story of toxemia restated? It is, but it also explains the periodicity of the attacks and the quick recovery.

The theory of intestinal intoxication or autointoxication as a cause of migraine, enjoyed for a few years a considerable degree of popularity. This popularity has waned in the past five years.

According to this theory, certain proteins when ingested were only partially broken down during the process of intestinal digestion. These incompletely digested proteins were said to be toxic and being soluble were absorbed into the blood producing a toxemia which brought about the migraine paroxysm.

Supporters of this theory attempted to prove their point with the fact that the toxic indican could be isolated in urine from patients during a paroxysm.

Many truths however have since been pointed out which have permanently put this theory into the background.

For example a low protein diet is helpful only in a very small percent of the cases and paroxysms can occur during a fasting or protein free period. Would this autointoxication occur only during the menstrual period?
Why should such autointoxication cease at the menopause or appear in younger life without other evidences of ill-health?

There is probably no phase of medicine that is subject to more criticism than the allergic phenomenon. Allergic conditions certainly do exist but as to the extent of their symptom producing ability great argumentation has occurred.

The allergists have for some time attempted to explain migraine on an allergic basis. Some are very enthusiastic in this respect, others rather passive.

In a recent conversation with an allergist I spoke of migraine and he informed me that he had had poor results in these cases and would have to be more thoroughly convinced of its allergic connection before he could place any faith in the allergic theory as applied to this condition.

Nevertheless the allergists have their points and must be considered.

Balyeat and Rinkel (5 and 6) have done a great deal of work in this field especially with migraine in children and have reported many favorable results.

They have proven to their own satisfaction that migraine paroxysms are the manifestation of the organism to protein sensitization and have reported several cases of
of cure based on this belief.

In their consideration of the hereditary factor they have pointed out a linkage of migraine with asthma, hayfever, urticaria and many forms of eczema. They also state that patients subject to migraine commonly show some other form of sensitivity.

The belief supported by them is that the sensitization in the greatest number of cases is of dietary origin. The food most commonly found to be the offenders were milk, wheat, eggs, nuts, beans and fish.

Dr. Albert H. Rowe (20) is probably more positive in his belief of the allergic etiology. He has stated that it is very possible that all cases of migraine are due to sensitization of the individual to a definite food or a definite type of foods.

He points out that physiological and pathological stress and strain are practically nil in children under the age of ten years, evidently pointing at the work of Dr. Grimes. I am personally in sympathy with the latter.

The variability of migraine headache is one of its outstanding characteristics. Small wonder at the variety of theories of etiology presented.

Regardless of etiology we know that these paroxysms have a number of exciting causes varying greatly in each individual. On account of this fact it is extremely di-
fficult to make theories of cause fit individual cases.

Probably the most common of all exciting causes is dietary indiscretion. Any sufferer of migraine realizes that he must pay the price for overindulgence in food. Especially foods of the richer types such as pastries, condiments, fried foods, etc. A high carbohydrate diet commonly will tend to increase the frequency of the paroxysm as will the more bulky vegetable food which are high in cellulose.

Fasting also tends to bring on an attack. In many cases missing or delaying a meal two or three hours will excite the chain of symptoms which do not subside as soon as food is taken. Breakfast is apparently the most important of all meals. The few cases I have contacted have stated that if they miss the first meal of the day they are certain to develop a headache before noon.

Constipation is a well known exciting factor. This symptom is present in most cases either in a marked or mild degree but the relief of the constipation alone is never sufficient to abort an attack.

Nervous and mental strain are also well known exciting causes of the migraine attack, as are eye strain, overheating, over fatigue, menstrual periods, and excessive indulgence in tobacco or alcohol.

The pathology of migraine has long been debated.
In a disease which leaves no pathological changes to be found on the autopsy table we must rely upon hypothesis for our knowledge.

It appears pertinent first to discuss the question: in which portions of the central and peripheral nervous systems can headache originate? It will be agreed that it is at the present time impossible to give an accurately scientific answer to this question. We can, however, on the strength of observations upon cerebral diseases of most diverse localization, as well as from the results of normal and morbid cerebral anatomy and physiology, establish one or two guiding facts which will help us to furnish an answer to the question raised.

We know that all headaches are perceived in the innervation areas of the fifth cranial or trigeminus nerves and of the sensory branches of the upper cervical nerves, and that very severe headaches are associated with all intra-cranial diseases leading to irritation of the meninges, particularly of the dura mater. (Cerebral tumors, cerebral abscesses, meningitis etc.)

The arachnoid has no nervous supply, the pia mater probably receives only sympathetic twigs accompanying the blood vessels.

The dura itself is supplied by five main nerve branches, (Cunningham (9)). The tentorial nerve, a
small recurrent branch of the ophthalmic division of the trigeminus is given off in the wall of the cavernous sinus. It runs posteriorly supplying the tentorium cerebellae and also sends branches to the dura of the post fossa of the skull.

The middle meningeal nerve, a small branch of the maxillary division of the trigeminus is given off in the cavity of the cranium. It is mainly distributed to the dura of the middle fossa of the skull.

The spinous nerve is a very minute branch of the mandibular division of the fifth cranial arises just outside the skull and accompanies the middle meningeal artery thru the foramen spinosum supplying the dura mater of the mastoid.

The vagus nerve also adds to the dural innervation. It gives off a meningeal branch from the ganglion jugular which passes posteriorly to supply the dura of the posterior fossa along with the tentorial nerve.

A recurrent meningeal branch of the hypoglossal nerve passes from this nerve at or near its origin to also supply the dura of the posterior fossa. This nerve communicates with the first and second cervical nerves from which it derives some sensory fibers.

The dura of the base of the skull is more richly supplied than that of the vault. This deduction can be
made clinically from the fact that headaches arising from tumors of the base of the brain are more severe and lasting than those caused by tumors situated on the convexity of the brain.

In trephining the brain substance can be needled without causing the patient discomfort but any pull or dissection of the dura causes the pain to become agonizing. The pain from irritation of the dura increases as the base of the skull is approached.

From the above knowledge we can then draw the conclusions that all headaches are produced by either a direct or reflex irritation of the nerves of the dura. What then causes this irritation and the multiplicity of nervous symptoms which often accompany migraine?

Muns (19) is of the opinion that migrainous symptoms are due to a congenital derangement of cerebral circulation plus a congenital defect in the chemistry of the body. After making this statement he gives no definite reasons for his beliefs. I am wondering just how he would explain the relation of defective circulation in relation to migraine.

Timme as quoted by Kennedy (17) advanced the theory that the migrainous attacks were caused by edema of the hypophysis impinging upon a small or closed sella turcica. X-ray however has not borne out this assertion. Cl-
In clinically, the pain caused by pituitary disease is much more deeply seated than that occurring in migraine.

Kennedy (17), Auerbach (4), Balyeat and Rinkel (5 and 6) are all in accord with the common conception that the headache in migraine is caused by localized vasodilatation of the arteries of the cerebrum. Such a dilatation causes multiple areas of active congestion in the brain substance, areas of edema result causing pressure on the dura producing headache and the accompanying nervous symptoms. Their theory is supported by the fact that ephedrine is helpful in relieving the pain in many cases of migraine.

This theory tends to support the allergic theory of migraine as all allergic phenomena are pathologically vasomotor dilatation in type.

It is the belief that the most common areas of the brain affected are the occipital lobe causing hemianopsia and scintillating scotoma, the sensory areas causing anesthesias and paresthesias, the motor areas causing motor losses, and Broca's area causing the different aphasias. (Starling (231)).

Along with the changes in the cranial space there are certain other changes which are known to be present during a paroxysm. It must be remembered that not only the nerves of the dura are affected but also an affect
may result through the ganglia of these nerves to other areas they supply.

The most commonly recognized activity of this reflex action is through the vagus nerve to the pyloric sphincter. It has been proven many times by use of the barium meal and X-ray that during the migraine paroxysm there is a marked pyloric stenosis. Food which is eaten just prior to, or during an attack, when vomited is returned pretty much in the same condition in which it was swallowed. This is especially the case in children, in whom the pyloric spasm is probably more marked.

As would be expected in the case of a cerebral edema which is mild in degree there is a mild rise in the pressure of the cerebro-spinal fluid. The increase is seldom over 5-8 m.m. of water. Removal of spinal fluid by lumbar puncture during a migraine paroxysm will give only momentary relief. The cell count in such a specimen is not increased.

I have a few times come upon the statement that an alkalinity accompanies the migraine phenomenon. I have been able to find nothing other than a casual statement to that effect. Who first originated this idea and on what he based his belief I have been unable to determine.

Menninger (18) has based his treatment of migraine with a ketogenic diet entirely upon this possibility.
In my own reasoning this possibility is obscure. I can see no reason why the reaction of the blood would shift to the alkaline side and can point out several reasons why it would be more liable to go in the opposite direction.

Why would alkalosis of the blood tend to cause cerebral edema and the resulting headache? If alkalosis is present why are good results often obtained through alkaline therapy?

In many cases fasting will infallibly precipitate an attack. This is certainly in opposition to the alkaline theory.

The symptomatology of migraine is extremely variable. In some cases headache is the only symptom in others there are many sensory disturbances and peculiarities. The classical triad is headache, visual symptoms and nausea with or without vomiting.

The onset of these symptoms most commonly occurs in the child below the age of ten years. Other patients will give a history of onset at the age of puberty. The largest percent of the cases manifest the symptoms before the age of 25 years and only a small percent after that age. When questioned as to the beginning of their trouble the patient frequently say, "I can not remember when I did not have headaches."
The migrainous attacks tend to follow a definite course throughout life. At their onset which as stated above, is usually in childhood the symptoms seem to be at their height and the attacks occur more frequently. This can probably be explained by the physiological instability which occurs in younger patients.

As life progresses the attacks become less frequent and the symptomatology tends to become less severe. This tendency of abatement continues until at about the age of fifty years, the patient is either entirely free from the attacks or is seldom subject to them. In women this cessation is common at the age of the menopause.

The outstanding feature of the symptomatology of migraine is its periodicity. In some it occurs but three of four times a year, in others as many times a month or week. In the pronounced types the time between attacks is constant. Although some incident may precipitate an attack earlier than usual, or habits of life postpone the paroxysm, the number of seizures suffered during a year is quite constant. The immediate exciting cause of one onset will as a rule, not again cause a paroxysm until a certain time has elapsed, and when an attack occurs earlier than usual there is a longer lapse of time before the succeeding one. Grimes (13) likens the attacks to a premature systole which produc-
es a temporary irregularity without changing the number of beats.

Typical paroxysms of migraine headache are often replaced by other types of sensory disturbance, thus giving rise to an apparent irregularity. When a case is critically analyzed, and paroxysms of migraine equivalent are taken into consideration, the attacks are found to be quite regular in time.

Hysterical seizures frequently take the place of the migraine attacks. These have been observed in women of the exhaustion type. In migraine coming on after the second decade of life the periodicity is found to be less constant.

In describing the symptoms of the individual attack of migraine it is best to divide the attack into phases. Namely: The prodromal symptoms, the aura, the attack itself and the post migrainous symptoms.

The prodromal stage is variable as to its length in different individuals. At the beginning of this stage the mind is more alert, there is an indescribable feeling of euphoria in which the person will attempt difficult mental and physical feats. The appetite is increased to a degree of almost entire loss of sense of repletion. Later the feeling of euphoria gives way to listlessness, and grogginess which prompts yawning and
the desire for food completely vanishes.

The Aura then appears and may manifest itself in various ways. Eye symptoms are frequently seen in which there is a scintillating scotoma, hemanopsia, ocular palsy or only a blurring of vision. There may be rather severe pelvic, abdominal or lumbar pain. Paresthesias of various distribution, with or without motor disturbances can occur. Vasomotor symptoms and vertigo are common and even psychic disturbances and aphasias are reported in some cases. A migraine subject does not always present the same type of aural symptoms thus giving rise to confusion in diagnosis.

With the beginning of the headache itself, the aural symptoms usually subside entirely or become less pronounced. The first pain denoting headache is usually dull in nature, constant and occurs over one eye within the cranium. It gradually increases in severity and seems to pass posteriorly to the occipital pole and still later the sensation of pain is throughout the whole cranial vault. At the height of an attack the headache is agonizing, the patient is prostrated and will lie quietly in a dark room if possible. These patients will usually refuse care as they wish to avoid disturbance.

The extremities are cold, the face is flushed, the eyes seem dim, the pain becomes throbbing and there is a
sensation that the cranium will split asunder.

At the height of the headache the sufferer becomes nauseated and finally vomiting occurs emptying the stomach completely. Vomiting may persist two to three hours in which the patient is able to raise only the bitter contents of the duodenum.

After the attack of vomiting, the headache usually subsides and rather suddenly. The patient sensing an almost pleasant sensation will drop into a deep and long sleep and arise apparently none the worse for the experience.

The duration of the paroxysm is of course variable. The average attack lasts from 12 to 16 hours.

Migraine in children like many other afflictions may vary as to symptomatology. In younger subjects dullness and languor are usually the first symptoms of an attack. There is no euphoria and abdominal discomfort is present instead of the increased appetite of adults. The child will sleep very soundly and may have a temperature as high as 104°. Headache is usually a minor symptom which seems to increase in its severity as the child grows older. The attacks of children usually become typical before the age of twelve.

The diagnosis of migraine is usually not difficult and looms large in general practice. The more often it
is looked for the more frequently it is found. The typical migraine sufferer usually comes to the physician with his own diagnosis. It is the atypical or the equivalent case that constitutes the main problem in diagnosis. Grimes (13) states that in 40 percent of the cases of migraine found in his practice the patients did not come to him primarily for relief of migrainous symptoms but their presence was elicited in the course of history and examination.

History is the all important factor in the diagnosis of migraine. The age of onset, the heredity, the periodicity are the outstanding points of differentiation. A history carefully taken will usually eliminate other diseases in which headache is the outstanding symptom.

The treatment of migraine, of course has as many possibilities as its etiology. Different medical men have obtained various results with different types of treatment. The results are always inconstant. Some cases are almost completely cured while others resist therapy entirely.

I recently heard one of our staff men make the remark that the only satisfactory treatment of migraine was fifty years of life. In certain cases this is apparently true.

27.
I am convinced that the sooner treatment is begun the better will be the prognosis. It is useless to treat each individual attack as it arises.

I know no other disease in which the care of the general hygiene is more important. Outdoor exercise is indispensible. Sufficient sleep is essential. The diet should be carefully regulated. Most cases seem to do best on a regime of low carbohydrate. Strict attention should be paid to prevent constipation. The removal of nervous and mental stress are very important. This is impossible in most cases but arrangements where by these factors can be reduced to a minimum can usually be made with good result. Psycho-therapy has produced surprising results in some cases.

The removal of focal infections of the tonsils, sinuses, teeth, and gall bladder or appendix have often aided in lessening the number of attacks. Dr. Shearer of our present staff is enthusiastic over results he has obtained in certain cases by the removal of impacted third molars.

Medication often accomplishes much. The treatment, par excellence in my estimation is alkalinization in conjunction with small doses of luminal. Citro-carbonate seems to be the best alkalinizing agent but many contend that sodium bicarbonate will give as good a re-
Citro-carbonate in half ounce doses with one pint of water two hours after meals or ten grains of sodium bicarbonate in the same amount of water given at the same time probably accomplishes the same result. In conjunction one half grain of luminal is taken on retiring. The treatment is continued for one year, four weeks at a time, with intervals of two weeks.

Under the treatment about twenty percent of all cases obtain complete relief and fifty percent are markedly improved.

The intravenous administration of the alkali, as introduced by Dr. Sicard of Paris has no advantage over the oral administration. Menninger (18) has claimed good results in the treatment of migraine by use of a ketogenic diet. In this diet he attempts to maintain a constant state of ketosis, using one gram of protein per kilogram of body weight with a fat carbohydrate ratio of approximately 1.5 to 1. His treatment has not been very heartily endorsed as most men claim a certain amount of danger in the ketogenic state.

The allergists base their treatment upon the elimination from the diet those foods which have proven by skin tests to be sensitizing to the individual. They claim as good therapeutic result as any other treatment.
Cannabis indica has long been used in attempt to abort migraine paroxysms. Good results are undeniable but this drug as is morphine must be used cautiously as there is danger of addiction.

Endocrine therapy is still in its infancy. Thyroxin, pituitrin, and ovarian extracts have been used in selected cases with fair results. They are certainly worth a try if indicated by the symptomatology.

The care of the paroxysm itself is important. First of all the patient should be given an enema and put to bed in a darkened quiet room. Water per mouth should be forced. An ice cap to the head and a hot water bottle at the feet are helpful. No food should be taken during an attack it only tends to exaggerate the symptoms.

Many analgesics have been recommended, aspirin in large doses is always helpful. Ephedrine and amytal compound is excellent to use in conjunction with the aspirin. I do not like to use caffeine as it is a cerebral stimulant. The barbiturates and analgesic coal tar products have their uses but must be given in large doses to be of any value. Bromides are slow in their activity and have no advantage.

Dr. W. Dandy (11) has written an article on "Treatment of Migraine by Removal of the Inferior Cervical
and First Thoracic Sympathetic Ganglia." I have gone over the article and cases reported carefully and have found that this operative treatment refers to cases of migraine neuralgia and not to cases of migraine headache.

The prognosis as to life is always good. Under treatment the patient is usually able to go about his occupation with a small degree of incapacitation.

Cases beginning earlier in life usually have a worse prognosis as to cure or relief than those coming on after the age of puberty. All sufferers of migraine like the congenital myopic have a degree of assurance that in old age their symptoms will have subsided.

Great interest attaches to the relationship between migraine and epilepsy. It is undeniable that there is a great clinical similarity between these diseases. In both there is incidence in attacks, frequently an introductory aura and a terminal sleep. In both there are complete and incomplete seizures; the determining factors are almost identical. The conjecture that underlying both diseases is a similar cerebral change differing only in a degree, is one which suggests itself to every one. This assumption is better grounded in that it can no longer be doubted that a transition may occur in the same individual from long-standing
hemicrania to epilepsy (Auerbach). Both diseases may be present simultaneously. These are only exceptional occurrences, but of theoretical interest. It would be going much too far to consider epilepsy and migraine as the same thing; one need only bear in mind the fact that in the latter case dementia never occurs, however long the duration.
Conclusions

1. Migraine is a definite clinical entity having a hereditary background which is Mendelian in character.
2. It usually occurs before the age of 25 years.
3. Pathological and physiological instability have a definite part in the cause of migraine paroxysms.
4. The exciting factors are variable.
5. Allergic and endocrine theories of cause are only applicable to selected cases.
6. There are probably multiple areas of edema in the cerebrum caused by vasodilation during an attack.
7. The classical symptom triad is headache, eye symptoms and nausea but the symptomatology is variable.
8. Diagnosis is based on a careful history. The disease is more common than generally supposed.
9. Probably the best treatment is good general hygiene plus alkaline and luminal therapy.
10. Prognosis is good as to life but poor as to complete cure. Migraine is definitely related to epilepsy but never goes over into dementia.
Case Reports
Grimes (13)

Case I.

A woman, single, age 48, a teacher, was the eldest of three children, all living and well. Her parents were living and in good health. Her menstrual life began at twelve and is still regular and normal. There had been no case of migraine in the family except the father who suffered severely from early youth until he was forty, when the paroxysms gradually disappeared.

The headaches made their appearance two years ago and have grown progressively worse. The attacks came every two weeks, initiated by hemiopia and vertigo. These symptoms persisted for an hour or two, when hemicrania came on, associated with nausea and most distressing vomiting. The attacks continued from twelve to twenty four hours.

Physical examination was negative.

Case II.

A married woman, age 33, was the mother of one child, age 6, living and in good health. The patient was the elder of two children. Her mother was a sufferer of migraine and her sister was like wise migrain-
At the age of 14, the patient began to have sick headache every three or four weeks, not in relation to the menstrual periods which also began at age fourteen. Since childbirth, six years before, the paroxysms have grown progressively worse. The attacks all had the classic symptoms.

Physical examination was negative throughout.

Case III.
A single woman age 28 and a teacher. Parents were living and in good health. She was the third of four children, all living and well. Medical and surgical history were negative. The maternal grandmother was the only known case of migraine in the family.

On entering college at 18 she began to suffer from typical migraine which came on every two or three weeks. The paroxysms were classical.

Physical examination was negative but her basal metabolic rate was found to be plus twenty.

Case IV.
A housewife, married, age 52, mother of three healthy children, the youngest 14. There was no history of miscarriages, and childbirth was uneventful. She had
never been ill. One year ago she began to have severe headache every ten days. The attacks were typical migraine, with scintillating scotoma, nausea, vomiting and hemicrania.

Two maternal aunts were migrainous the family history was otherwise negative.

Physical examination was negative except for two infected teeth.

Case V.

The patient was a bank clerk, age 38, with a wife and two children in good health. The family history revealed that his father was migrainous.

The onset of his attacks was in childhood at about the age of four. The attacks have occurred every two or three months and last twelve to twenty-four hours. They were predicted by euphoria and visual blurring. Headache nausea and vomiting followed.

Physical examination was negative.

Case VI.

The patient was a woman age 45, married, the mother of five children all living and well. Childbirth was uneventful. The family history showed migraine in a maternal grandmother, mother and sisters. The patient was

36.
the eldest of six children, four of whom had migraine. At the beginning of menses she suffered severe dysmenorrhea and backache never passing a painless menstrual period. At 36 she had a ventral fixation of the uterus with no improvement.

Later the dysmenorrhea was associated with epigastric pain and vomiting and a cholecystectomy was performed which gave no relief. The suffering continued until the menopause, when dysmenorrhea was replaced by typical migraine attacks which have continued since that time.
Note: I have entered here only the main references. I have not entered any subreferences found under the following writings.

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41.