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The Biological Treatment of Migraines, Based on Experience

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A classification of various types of migraines opens this lecture which Dr. Gerhard presented at the 3rd HEEL U.S. Symposium in San Francisco in March, 1988. Dr. Gerhard, a physician with much experience in the biological treatment of migraines, then discusses syndromes similar to migraines and concludes the lecture with his recommendations for migraine therapy.

The vast number of publications devoted to the topic of migraines confronts the reader with a considerable amount of contradictory information. Neither the etiology nor the pathogenesis of the affliction has ever been satisfactorily explained. At present, however, three factors have led me to make my own contribution to the discussion.

1. The large number of patients visiting doctors' offices for cure of "migraine headaches."
2. My own professional experience with the use of homeopathic preparations, which I have been using for the treatment of simple and classic migraines with considerable success. I hope to make this combination accessible to a wider range of therapists.
3. Establishment of a new nomenclature.

The nomenclature used with migraines in the German-speaking countries has for many decades made heavy use of French terms (for example, migraine ophthalmique, migraine accompagnée, et al.).

In recent decades, however, English terminology has found increasing use in the international literature. This development is completely desirable and should also be incorporated into the terminology used in this country and abroad to avoid difficulties of understanding. The most important variants are the simple and the classical migraine. A third, very rare, and thus more marginal variant is the complicated migraine.

In the case of the simple migraine, the pain develops without a clear progression through phases and without accompanying neurological dysfunction. The accompanying symptoms usually affect the autonomic nervous system with manifestations ranging from nausea to vomiting. In addition there are signs of a generally reduced stimulus threshold in the central nervous system with symptoms such as emotional instability, photophobia, and phonophobia. This is the most common form of migraine and affects almost 70-80% of all migraine patients.

The term "classical migraine" is used when the attacks occur along with accompanying transient neurological dysfunctions. These may appear in a phase prior to the actual headache pain or accompany it briefly. The problems involved here include sensory, visual, and motor dysfunctions as well as impairments of speech. A scintillating scotoma, appearing in a phase prior to the actual headache pain, is regarded as the aura in migraine diagnosis. The frequency of this variant is roughly 20%.

The complicated migraine, with a frequency of less than 1%, is much more seldom. Here the symptoms include attacks of headache pain with prolonged neurological dysfunctions which may outlast the actual headache pain by hours, sometimes even days.

Classification of migraines

1. Simple migraines:

– No development in phases

– No neurological dysfunctions

2. Classical migraines:

– Migraine attacks with development usually in phases and short-term neurological dysfunctions which precede headache pain or accompany it in stretches.

The following terms are included in this category:

– Ophthalmic migraines

– Migraine accompagnée

3. Complicated migraines:

– Migraine attacks with prolonged neurological dysfunctions which may outlast headache pain.

This includes terms such as:

– Hemiplegic migraines

– Ophthalmoplegic migraines

– Basilar migraines

The diagnosis of migraines

Because there are no pathognomonic bases, whether physical, apparative, or chemical in nature, for the certainty of migraine diagnoses, the anamnesis remains an important determining value. The classification of symptoms into categories of "hard" and "soft" seems especially helpful.

Basic diagnosis for migraine patients

– Organic headache genesis must be ruled out

– Recurrent headache pain attacks of at least several hours in duration

Hard symptoms:

– Development in phases

– Hemicrania

– Vomiting

– Scintillating scotoma

– Focal cerebral symptoms

– Throbbing, pulsating, hammering pain

– Occurrence within patient's family

Soft symptoms:

– Vertigo

– Nausea

– Photo-/phonophobia

– Attacks often begin upon awakening from night sleep

– Attack fades after sleep

In view of the special importance and of the diagnostic differentiation of migraines, I would like to briefly discuss trigeminal neuralgia, cluster headaches, temporal arteritis, and glaucoma (in part).

Differentiating among these diagnoses offers fewer difficulties. Confusion may occur, however, between trigeminal neuralgia and cluster headaches.

Trigeminal neuralgia

This affliction primarily affects the elderly. Women are more frequently affected than men. Apart from certain exceptions, trigeminal neuralgia generally occurs in strictly unilateral form. The pain spreads astorientationally. The second and third rami are affected most frequently, either individually or mutually. Less frequent is the common development of pain in the first and second rami; development of pain in all three rami at once or only in the first is extremely rare. According to patient descriptions, the attack begins with a sudden fulgurant pain of extreme intensity, lasting from a few seconds up to about half a minute. These attacks within the framework of a genuine trigeminal neuralgia are clearly among the most unbearable pains which can be experienced by human beings; suicidal tendencies are by no means uncommon. At the onset of pain, stiffening of the facial features or individual convulsions of the facial musculature can be observed (tic douloureux). The irritation of certain localized areas in the skin or in the mucous membrane of the mouth triggers the pain. In general one speaks of the triggering of the pain or of trigger zones.

Chart 1: The diagnostic criteria of trigeminal neuralgia

– Unilateral attacks of pain

– Sudden fulgurant pain of extreme intensity

– Commonly centered in the second and third rami

– Trigger mechanisms: tactile stimulations, chewing, speaking, shaving, and many others

– Tic douloureux

– Normal neurological findings

– First manifestation normally after age 50

Cluster headache pain (Big Horton's syndrome, histamine headache, erythroprosopalgia)

In stark contrast to the migraine, as well as to the genuine trigeminal neuralgia, males are affected much more frequently. The ratio of men to women is roughly 7:1. Diagnosis is made easier by the fact that the pain is often accompanied by a variety of symptoms, mainly in conjunction with the automatic nervous system. The individual symptoms, each attended by great intensity of pain, include substantial secretion of tear fluid (lacrimation) and nasal discharge (rhinorrhea) or the feeling of a congested nose and injection of

conjunctival vessels. The following may also occur on an irregular basis: a light reddening of skin around the eyes along with a feeling of warmth, hyperhidrosis, incomplete Horner's syndrome with ptosis and possibly also light miosis, swollen temporal artery, periorbital oedema, facial hyperalgesia, and vagal bradycardia. There are frequent complaints of phono- and photophobia as well as nausea. As is the case with migraines, however, vomiting is more seldom.

Chart 2: Diagnostic criteria for cluster headaches

- Strong, unilateral attacks of pain, duration of attacks 20 minutes to 2 hours, often several times a day, pain of extreme intensity
- Periods of pain lasting up to several weeks
- Pain-free intervals lasting up to several months
- Typical localization: area around eyes, temples
- Pronounced vegetative/vasomotory accompanying symptoms
- Redness of conjunctiva, also often of facial skin
- Lacrimation on the side affected
- Rhinorrea, or also congested nose on side affected
- No pathological neurological findings, attack sometimes contains incomplete Horner's syndrome
- Primarily affects men, usually around age of 20-25 years
- Ratio of men to women: 7:1

Arteritis temporalis (giant cell arteritis, auto-immune disease, Horton-Magath-Brown syndrome)

While diagnostic errors may occur in the case of trigeminal neuralgia and cluster headaches without causing harm to the patient, errors should definitely be avoided in the case of arteritis temporalis and glaucoma. Often associated in the preliminary stage with a polymyalgia rheumatica, the latter two cases often lead patients to experience fatigue and a strong feeling of sickness. Pain persists for the course of several days or weeks and, aside from a few exceptional cases, often responds quickly to therapy with corticosteroids. A relatively frequent danger is that of blindness due to iscaemic opticomalacia. Blindness can be prevented in any event if corticosteroid therapy is begun on time. With the majority of patients the location of the arteria temporalis superficialis is conspicuous at the temples, often prominent, pressure-sensitive, convoluted, and in many cases even pulseless. Chemical analysis in the laboratory shows a rise in the amount of alpha 2 globulin as well as a very high level of blood sedimentation.

Chart 3: Diagnostic criteria of arteritis temporalis

- Intense, dull continuous headache pain, mostly unilateral on one side in temple or nape, also bilateral
- Temporal artery usually pressure-sensitive, prominent with adjacent oedema
- Fatigue, general feeling of sickness, slight fever possible
- Brief or irreversible loss of vision (opticomalacia)
- Manifestations after age 55
- Erythrocyte sedimentation often drastically accelerated
- Increase of alpha 2 globulin in electrophoresis
- Rheumatoid factors negative

Certain diagnosis:

Biopsy from arteria temporalis superficialis: giant-cell arteritis.

Glaucoma

In many cases, of course, a family doctor will be consulted rather than an ophthalmologist, and information supplied by the patient may well be misleading or unhelpful.

I can recall an eye clinic lecture in which our professor presented the following case. A 65-year-old man complained about increasing discomfort, headaches, and uncontrollable vomiting until his family doctor finally admitted him, on the basis of acute pains in the upper stomach, to a surgical clinic, where a laparotomy was subsequently performed. Two days later the man consulted an ophthalmologist, who then diagnosed an acute glaucoma. In the interim the eye had gone blind.

This example should provide grounds for thoughtfulness.

For this reason the questions of eye pain and eye pressure should never be forgotten. An additional factor is the appearance of the cornea, which is often lustreless, dull, and may display a turbid epithelium with flattened anterior chamber. In addition there may be reduced visual acuity and congestive reddening of the eye (not to be confused with conjunctivitis or iritis). The most important sign, however, is the hardness of the bulbus. Descriptions of pressure increases of up to 100 mmHg (normally 15-20 mmHg) can be found in the literature.

In order to examine for this, the doctor asks the patient to close his eyes and look down. The doctor then feels the upper lid with his right and left index fingers; in the case of an "acute glaucoma" he will find a rock-hard bulbus. For comparison the doctor may then feel the patient's other eye, or one of his own, in order to arrive at a more certain diagnosis.

An acute case of glaucoma is an emergency situation.

Chart 4: Diagnostic criteria in the case of glaucoma

- Extremely intense pains in the head, face, and eyes
- Frequent vomiting
- Generally strong feeling of sickness
- Pronounced redness with congestion of the episcleral vessels
- Turbid cornea
- Flattened anterior chamber
- Dilated and reactionless pupil
- Increased intra-ocular pressure values of up to more than 80 mmHg (normal range from 15-20 mmHg)
- Deterioration of sight

Differential diagnosis:

Iritis, conjunctivitis, trigeminal neuralgia

The pathogenesis of migraines (hypotheses)

Unfortunately neither the etiology nor the pathogenesis of migraines has been fully explained even today. There is a wide variety of models and hypotheses which, however, in most cases do not hold up under closer scrutiny. Theories about heredity also failed to withstand closer observation, as shown by comparative studies involving twins. Many scientists additionally claim that it is not the sickness per se which is inheritable, but rather a special pattern of reaction. This basis leads to development of a reactivity which can be modified by various factors (endogenic, exogenic). The additional effect of various accidental factors then finally triggers the migraine attack.

One definite precondition for the occurrence of migraines is an abnormal vascular instability of the intra- and extracranial vessels. Wolfe's ideas, generally accepted today with some reservations, distinguish between 3 phases within the framework of the migraine attack:

a) vasoconstrictor phase

b) vasodilatator phase

c) oedemal phase

a): During the vasoconstrictor phase there occurs a narrowing of the intracranial vessels, leading as the result of ischaemia to visual symptoms (scotoma, diplopia, amaurosis, pupil dysfunction, and many others) as well as other neurological symptoms (hemiplegia, hemihypoaesthesia, opthalmoplegia, aphasia, etc.). These symptoms precede, as a rule, the phase of actual headache pain.

b): This is followed by the vasodilatatoric phase attended by a widening of primarily extracranial vessels. It leads to the typical migraine headache, characterized by intense, heavy, often semilateral localization and pulsating character. An external sign which sometimes occurs is a visible swelling of the arteria temporalis; the pain can be temporarily reduced by applying pressure to the vessel.

c): If the attacks last over a longer period of time, the oedemal phase begins, in which a transudation into the vascular walls or perivascular tissue takes place. It is characterized by a duller headache pain which may often last for several days. If the attacks are of longer duration and greater frequency, the result may be long-term disturbances incurred as a consequence of ischaemia or infarct. Long-term disturbances of this type, such as visual dysfunction, hemiplegia, hemihypaesthesia, aphasia, and basilar migraine symptoms fortunately occur only singly.

Because many migraine patients also suffer from normal headache pains, the question always arises of whether the first indications of headache pain are already preliminary indicates of a migraine attack. We thus distinguish between certain and uncertain indications:

Uncertain indications (feelings of ill health)

Head pains, pressure in head

Certain indications of an aura

Scintillating scotoma, polyuria

Certain biogenic amines, mediators, and thrombocytes play an extremely important role in the explanation of migraine attacks. A particularly key role is ascribed to serotonin. Its plasma concentration increases during the prodromal phase and declines in the later phases. Serotonin is thus assumed to have been initially subjected to increased decomposition and can finally be found as an end product in urine in the form of 5'hydroxyindolacetic acid. The individual steps of investigation have also been largely accounted for today. Research is thus being conducted to determine the initial triggering factor, discovery of which would undoubtedly provide the key to determining the ultimate cause of migraine attacks.

Program of examination

I have examined a total of 28 patients (several in cooperation with a neurologist and an ophthalmologist) who were suffering from simple and classical migraines. The average age was 32, of which 17 patients were female and 11 male. In only 5 cases were there insignificant improvements in the patients' condition. In 3 cases the therapy had no effect; here I must add, however, that the migraines stopped following dental examination and removal of the causes (granuloma, jaw cyst). 20 patients were treated successfully (over 70%), with some of them remaining symptom-free for more than 8 months; among other results, there was a clear decrease in the frequency, strength, and duration of attack. Amnestically, severe abuses of medication had to be dealt with in all cases. One patient had to be repeatedly admitted to a clinic due to gastric

bleeding and gastric ulcers after having taken all of the prevalent preparations over a course of many years:

- Analgesics
- Secale alkaloids
- Anti-emetics
- Antihistimines
- Tranquilizers
- Serotonin antagonists
- Beta-blockers
- Calcium antagonists
- Antipleptics
- Thrombocyte aggregation impeders

and many others.

This provides the basis for a compulsive therapy intended to be not only safe but also successful. This demand can be fulfilled with homeopathic preparations.

Migraine therapy

I now come to the most important part of my discussion, namely, the therapy of migraines.

It can be said the therapeutic success of migraine treatment is based essentially on 3 elements:

A) Avoidance of triggering factors

B) Treatment of acute migraines

C) Interval therapy

A): Avoidance of triggering factors
Triggering factors among normal and luxury foods and stimulants include, in the order of their frequency, alcohol, followed by chocolate, cheese, dairy products, fruit, and many others. Additionally, of course, hunger, exhaustion, disturbance of the sleeping-waking rhythm (sleep deficit, workplace disturbances, as well as stress and psychic factors and, last but not least, medication) may also be responsible for triggering migraine attacks. Here the doctor should make an effort to influence the patient's conduct of life.

B): Treatment of acute migraines
The following HEEL preparations are administered intravenously in mixed-injection form:

- Spigelon (ampules)
- Aconitum homaccord (ampules)
- Belladonna homaccord (ampules)
- Gelsemium homaccord (ampules)
- Ferrum phosphoricum-injeel (ampules).

If intense gastric disturbances are also prominent, some Nux vomica-homaccord (ampules) might also be added.

In the case of patients who develop migraines with particular frequency on weekends, Iris-injeel (ampules) has proved an especially effective additive.

C): Interval therapy

Interval therapy should be considered if an average of more than two migraines a month occurs or if individual attacks run with particular intensity for several days. A distinction is made between medicamentous and non-medicamentous treatment.

I would like to name several preparations for medicamentous interval therapy which I have put to successful use in my practice. They are:

- Colocynthis homaccord (drops)
- Gelsemium homaccord (drops)
- Psorinoheel (drops)

For standard therapy with, additionally:

- Cimicifuga homaccord (drops) for painful menstruation
- Iris injeel forte (in orally administered ampule) for weekend migraines

These preparations should be mixed well in amounts of 45 drops to one glass of water or tea and drunk over the course of the day, before meals. The patient should be instructed to hold the drinking water in the mouth for a period of time in order to ensure better absorption through the mucous membranes.

Furthermore:

- Spigelon (tablets)
- Hepeel (tablets)

1 tablet should be taken 3-4 times per day by letting it dissolve beneath the tongue (or sucking it).

Application of the abovementioned therapies will bring about reductions in the frequency, intensity, and duration of attacks and cures can be expected.

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