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MIGRAINE EQUIVALENTS*
LORNE D. PROCTOR, M.D.**

Migraine is an illness probably known to the Egyptians as early as 12 B.C., but first described adequately by the Greeks (Arateaus of Cappadocia)\(^1\) in the first century A.D. Classically it applies to a vascular type of headache involving only one half of the cranium, preceded by a visual aura and accompanied by nausea and vomiting. The illness is recurrent and has a familial diathesis.

Wolff and his associates\(^2\) have discussed vascular headaches extensively and have ample evidence supporting vasodilatation of blood vessels with an altered contractile state, probably following a phase of vasoconstriction, as the vascular dysfunction associated with migraine. The vessels involved are the internal and external carotid systems, the circle of Willis and the basilar systems. These phenomena may be seen at times in the conjunctival vessels and/or in the retinal vessels on the affected side.

There is little need to dwell on the so-called “migraine personality”. May it be sufficient to point out that hypertonicity and rigidity of thinking and behavior are outstanding components in this personality pattern. It may be comforting to point out to migraine sufferers that this illness has a predilection for the more mentally agile individual.

Migraine and its equivalents follow, rather than accompany unusual stress.

In considering migraine equivalents, we will deal with those conditions in which apparently the above vascular dysfunctions produce clinical manifestations that do not conform to the classical migraine syndrome. The equivalent may be present as a cranialgia followed by some other neurological signs or symptoms or it may consist of a purely transient episode of a localized neurological dysfunction e.g., neuralgia, palsy, etc. Migraine equivalents, in my experience, are as common, if not more common than the classical migraine “attack”.

In describing the various migraine equivalents it is easier to understand the variety observed if one considers the multitude of areas of the central nervous system that may be locally involved in this vascular dysfunction. Should only the occipital cortex be involved, quadrantic or hemiopic visual defects will occur without subsequent hemicranialgia or vomiting (ophthalmic migraine). If the parietal lobe is the site of this vascular dysfunction, paresthesiae, analgesia or various types of pain, usually limited to the face, neck and upper limbs on the contralateral side of the body, could be the predominant symptoms. Similarly if only the motor cortex is involved by the vascular dysfunction, facial weakness, monoplegia or hemiplegia could result. The sensory and/or motor dysfunctions may last for minutes or hours, and it is sometimes difficult to differentiate between such migraine equivalents lasting 12 to 48 hours and so-called “little strokes”. Personally I am not quite certain what many authors mean by this latter term. I assume they have in mind at least a permanent occlusion of one or more small vessels producing this neurological clinical picture.

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*Presented as part of Postgraduate Course No. 6 of the American College of Physicians at Henry Ford Hospital, Detroit, Michigan, January 25-29, 1960.
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If the migraine equivalent vascular dysfunction involves the temporal lobe (on the dominant side), particularly the posterior portion, episodes of expressive or receptive aphasia may occur. Extensive involvement of the temporal lobe or lobes could produce varying degrees of impaired consciousness. In this latter manifestation one must differentiate between a migraine equivalent and psychomotor epilepsy. This author collected a series of 130 cases having as a common denominator a psychomotor variant EEG pattern, and among these more than 30% complained of episodic headache. Three of the 30% with headaches had the classical migraine type. Ninety percent of all cranialgias in this series improved on sodium dilantin medication. This is further evidence of the close relationship between migraine and epilepsy as first pointed out by Lennox.

When the uncinate lobes are involved in this vascular process, gustatory and/or olfactory episodes may occur. A patient seen by the author had suffered classical migraine attacks for many years and these were replaced in her 40's by periods of smelling "strong gasoline". The most effective therapy for these attacks continued to be Cafergot, the same medication she had used for the previous migraine episodes. The usual gustatory experience is an episode of metallic or sour taste.

Continuing consideration of various cortical areas involved by the "migraine" type of vascular dysfunction, one of the more rare equivalents, manifesting an episode of spatial disorientation — the patient feels suspended above the floor, not necessarily in the vertical position and is confused as to right and left, etc. — is thought to be due to localization of the vascular dysfunction in the region of the angular gyrus.

Much has been written about abdominal migraine, a migraine equivalent. Headache may precede the abdominal cramps, nausea and possibly vomiting, or abdominal migraine may not include the "aura" headache. The insula area may be involved in these episodes. Some authors have described episodes of thoracic pain, pelvic pain or precordial pain, the latter particularly associated with tachycardia and dyspnea (precordial migraine). Nausea and vomiting may precede or accompany such episodes. I find it difficult to suggest the probable localization of the central nervous system dysfunction in these equivalents.

Another rare equivalent is episodes of cerebellar ataxia, no doubt the result of "migraine" vascular dysfunction involving the cerebellar arteries. Similarly, basilar artery involvement could produce episodes of vertigo, impairment of conjugate deviation of the eyes, ataxia and finally even impairment of consciousness, e.g., confusion, etc., through involvement of midbrain reticular formation.

Severe autonomic or hypothalamic migraine equivalents are also rare, but the milder forms are more frequently seen, in which periodic episodes of hypersalivation, rhinorrhea, dilation of pupils, hyperhidrosis and flushing preceding or attendant with headache occur.

The author has not observed ophthalmoplegic migraine, another equivalent, but Merritt and Friedman describe cases with episodes of recurrent unilateral headache accompanied by extraocular palsies. They suggest pressure upon the 3rd cranial nerve passing between oedematous superior cerebellar and posterior cerebral arteries,
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and similarly pressure on the 6th cranial nerve passing between oedematous auditory and inferior cerebellar arteries as the basis for these extraocular palsies. The 3rd cranial is the most commonly affected (10 times), and the pupil invariably is dilated. The palsies may last for days or weeks after the headache.

Recurrent episodes of a variety of neuralgias, with or without headache, could be included in this ill defined equivalent category. The most common involves the trigeminal area, probably through vascular embarrasment of the Gasserian ganglion. The remaining facial neuralgias in this group could be attributed to a "migraine" dysfunction of the external carotid artery, and would include Gardner's petrosal neuralgia, vidian neuralgia and appears to closely resemble Horton's histaminic cephalalgia and cluster headaches.

Finally, neuralgia over the distribution of the temporal artery is mentioned only to set out that transient changes in and about this vessel are associated with this complaint. Wolff has used the changes in tissues surrounding the temporal artery in such a case to illustrate that edema and change in pain sensitivity does occur in subjects with vascular headaches of the migraine type. Such a neuralgia should be differentiated from temporal arteritis. This latter "neuralgia" is progressively of longer duration, and the artery is found to be hard and nodular and exquisitely tender.

Bell's palsy has been noted as an accompaniment of headache, and may last for hours or days after the headache.

The differential diagnosis of migraine equivalents must include tension headaches, the most common of all headaches (30%), but this presents little difficulty as the occipital muscle mass is usually tense and tender to pressure, the headaches are not cyclic, but continuous (days or weeks) and nausea or vomiting seldom occurs; there is no aura.

It is most important to keep in mind that migraine equivalents may be symptomatic of organic rather than functional disease of the central nervous system, and should there be any question as to the cyclic nature of the dysfunction or if there occurs a progressive increase in severity of consecutive episodes, a most thorough neurological investigation, including angiography, pneumoencephalography and electroencephalography is indicated. The lack of response, of what is thought to be migraine equivalent, to early treatment by vasoconstrictor drugs should arouse a strong suspicion regarding this diagnosis.

In the case of precordial pain, the usual thorough cardiovascular investigation is indicated before the diagnosis of migraine equivalent is made as would be a thorough gastroenterological investigation in the case of abdominal migraine. Electroencephalography will assist in screening out those borderline cases of so-called epileptiform syndromes where the migraine syndrome is in reality an epileptic equivalent, and requires anti-convulsant medication, i.e., sodium dilantin and phenobarbital, rather than vasoconstrictor drugs.

The treatment of migraine equivalents is the treatment of migraine. Prophylaxis is important. We are usually dealing with hypertonic individuals that are experiencing
difficulty adapting to their stress. The need for psychotherapy is obvious, and many can be adequately treated by merely supportive psychotherapy with adjustment of habit pattern and a mild relaxant e.g., sodium amytal, grs 1, b.i.d. or t.i.d. Others may require interpretive psychotherapy or a lengthy formalized psychiatric program. Alcohol ingestion is one of the best ways to trigger a migraine equivalent.

During the attack ergotamine tartrate, 1 mgm, with caffeine, 100 mgms (Cafergot) has been found to be of the greatest value. It should be administered at the earliest possible stage of the attack, and if the patient cannot tolerate this medication orally (vomiting), suppositories, 2 mgms ergotamine tartrate with 100 mgms caffeine, may be used. The maximum dose of ergotamine should be 6 to 8 mgms per day, and this program not repeated in less than 10 days. If parenteral ergotamine is necessary, dihydroergotamine methanesulfonate (DHE 45) is indicated. It is less likely to produce side effects e.g., vomiting, cramps, tingling of hands, etc., but as it has less vasoconstriction action than ergotamine tartrate a 1 mgm dose intramuscularly could be repeated in 1 hour. Ergotamine compounds are contra-indicated in the presence of pregnancy, peripheral vascular disease, angina pectoris, impaired renal or hepatic function, etc.

Analgesics e.g., acetylsalicylic acid and its combinations including, if necessary, a minimum dose of codeine is the usual means of controlling a lengthy cranialgia.

There are many other pharmacological preparations for the treatment of migraine and the equivalents, e.g., wigraine, migral, histamine diphosphate, diamox, octin hydrochloride, etc. A very thorough consideration is given to this subject in Merritt & Friedman's recent text.1

The medical problem of migraine equivalents lies in the confusion these syndromes create if the internist does not consider the vascular nature of their aetiology. They may mimic, in part, intracranial disease of many types, i.e., neoplastic, vascular, the neuritides, meningitides, Meniere's disease, arteritis and epilepsy, etc. The cyclic nature of their occurrence, the usually episodic nature of this syndrome and the marked benefit from vasoconstrictor drugs will be of the greatest diagnostic assistance. If we are to diagnose migraine equivalents, we, of course, must think of them, remembering they may be symptomatic of organic disease of the central nervous system.

REFERENCES