

# Headache Toolbox

## What is Migraine?

For a disorder that afflicts a tenth of our population and is the subject of thousands of private conversations each day, surprisingly few people – even physicians – can provide a coherent answer if asked: What is migraine?

Migraine is relatively easy to define in clinical terms. One is said to have migraine if within his/her lifetime there have occurred 5 or more attacks of unprovoked headache lasting 4-72 hours, severe enough to markedly restrict or even prohibit routine daily activity and accompanied by nausea or light/sound sensitivity. Note that the clinical definition of migraine does not require the headache to be pulsatile (“throbbing”) or lateralized to one side of the head: although such features are common in migraine, they are far from invariable. Nor does the diagnosis of migraine require the occurrence of *aura* symptoms, visual or otherwise; only 20-25% of migraineurs ever experience aura, and in that minority who do there are relatively few who experience aura with each and every attack. Finally, the headache of migraine need not be intense or disabling on every occasion; some migraine attacks may involve no headache whatsoever (aura without headache), and many attacks may involve headache that is mild in intensity and more reminiscent of tension type headache than what one typically associates with migraine.

What causes migraine? For many years it was believed that migraine attacks arose consequent to changes in the blood vessels which supply the head and brain; aura (when it occurred) was attributed to constriction of arteries, with neurological symptoms resulting from impaired blood flow and the “throbbing, sickening” pain of migraine attributed a compensatory dilation of those and other vessels.

We now believe that migraine is probably genetic in origin and that the disorder reflects a genetically induced *hypersensitivity* involving neurons (brain cells) located within the central nervous system. If a genetically primed neuron is triggered by a change in the external environment (eg, drop in barometric pressure) or internal environment (eg, sudden drop in estrogen level), that neuron may activate and, triggering its neighboring neurons to join in, induce the pathways in the brain that normally conduct head pain to awaken and produce the familiar symptoms of a migraine attack.

As such, migraine is somewhat akin to epilepsy. Both conditions reflect brains containing neurons that are abnormally sensitive, and as with migraine, the source of this sensitivity in epilepsy may be genetic. Migraine and epilepsy are “bi-directionally co-morbid” (ie, if one has migraine, he/she is more likely to have epilepsy than normally would be expected and vice versa). Further cementing this relationship is the fact that not a few of our best medications for migraine prevention were first developed to treat epilepsy (eg, divalproex sodium [Depakote] and topiramate [Topamax]).

Both acute and chronic therapy for migraine thus is intended to stabilize a genetically primed brain that has become acutely or chronically unstable electrochemically. While blood vessels may play an important role in the sequence of physiologic events that generate migrainous symptomatology, it appears to be the brain – not blood vessels – that represents the ultimate origin of migraine.

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