

### Transcript Details

This is a transcript of an educational program. Details about the program and additional media formats for the program are accessible by visiting: <https://reachmd.com/programs/rethinkingmigraine/creating-dialogue-with-patients-on-the-pathophysiology-of-migraines/9966/>

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## Creating Dialogue with Patients on the Pathophysiology of Migraines

### Announcer:

This is ReachMD. Welcome to this special series, *Rethinking Migraine*, sponsored by Lilly. On this episode, titled *Answering the Question: Why Do I get Migraines?*, we hear from Dr. Nada Hindiyeh, Clinical Assistant Professor of Neurology & Neurological Sciences at Stanford School of Medicine, to answer the question.

### Dr. Hindiyeh

Explaining migraine pathophysiology to patients can seem like a challenging task, especially since the underlying mechanisms are complex and still not fully understood. Nevertheless, patients will still ask the question: "Why do I get migraines?" And how detailed you make your discussion will really depend on what your patient's looking for. Some patients just want to know the basics, so it's important to relay that people who suffer from migraines have a genetic predisposition to a hyperexcitable brain that is provoked by internal or environmental triggers. For those who want a more detailed explanation of what's happening in the brain during a migraine, it's important to consider what their individual experiences are during the course of a migraine, as this will resonate most with patients. The phases of a migraine attack include the premonitory or prodromal phase, the aura, the headache pain and the postdrome. Not all migraineurs will experience all phases, and they can vary in length. Migraine is thought to be generated centrally in the brain in areas like the hypothalamus and the cortex. The hypothalamus is thought to play a role in the prodromal phase of the migraine, and it contributes to regulation of homeostasis and circadian rhythms. It's reasonable to think that when altered, patients experience symptoms such as mood changes, fatigue, food cravings, repetitive yawning, and urination changes, among others. These changes happen anywhere from hours to days before the headache phase comes on. And the aura phase only occurs in about 25 to 30% of people with migraine and typically lasts from 5 minutes to an hour, and studies suggest that it is caused by cortical spreading depression, a slowly propagating wave of depolarization or excitation followed by hyperpolarization or inhibition in cortical neurons. These alterations are thought to activate the trigeminal vascular system leading to a cascade of events resulting in migraine pain. In this system, the distal trigeminal nerve terminals innervate pain-producing structures such as the meninges and the intracranial blood vessels. They then converge centrally in the trigeminal cervical complex of the brain stem where neuropeptides are released. The trigeminal cervical complex has key connections to other brain areas such as the hypothalamus, the thalamus, other brain stem nuclei and the cortex, accounting for the constellation of symptoms seen during a migraine attack. Some of the goals you want to think about when discussing migraine pathophysiology with your patients is that the exact cause of migraine is still not fully understood. We do know that it is a complex brain network disorder, and providing patients with an educational background on migraine pathophysiology gives them the tools to understand what's happening in their brain during a migraine, help them better communicate with you about questions they are having, and will also help them understand why as physicians we recommend certain treatment strategies for reducing migraine frequency and severity.

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