

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/the-evolution-of-migraine-theories/9964/>

ReachMD

www.reachmd.com
info@reachmd.com
(866) 423-7849

The Evolution of Migraine Theories

Announcer:

This is ReachMD. Welcome to this special series, Rethinking Migraines sponsored by Lilly. On this episode, titled A History of Migraine: Pathogenic Understandings we will hear from Dr. Mark Green, professor of neurology, Anesthesiology, and Rehabilitation Medicine and director of headache and pain medicine at the Icahn School of Medicine at Mount Sinai in New York, NY.

Dr. Green:

Historically, many of us, certainly including me, learned the theory of migraine from Harold Wolff, largely spoken about in 1948. Harold Wolff's theory was the vascular theory of migraine. And fundamentally, he said that migraine auras were due to constriction of arteries and migraine headaches were due to vasodilatation of the same arteries. So it was then described as a vascular headache. If you look at this a little bit more carefully in terms of perfusion, you'll see on this slide that there's hypoperfusion during an aura; however, the hypoperfusion continues as the attack transitions from an aura to the headache. And then largely during headache, there's hyperperfusion. But if you look at this carefully, you'll see as the headache resolved hyperperfusion continued. So, at best, you have to appreciate that the phenomena of hypoperfusion and hyperperfusion, and aura and headache, are clearly not very tightly coupled. About 40 years later things changed when the work of Michael Moskowitz came out, and he spoke about trigeminal vascular neurons, explaining that these arteries didn't do anything on their own. They only did what nerves told them. He also introduced the concept of neurogenic inflammation. The fact was that there was inflammation in the meninges, and this explains why there are so many similarities of an acute migraine attack to an acute attack of meningitis. The pain is often pulsatile in both, patients don't move their eyes around without pain; their neck becomes tight. And so we talk about vessels becoming involved in migraine, but there's also a meningeal inflammatory response that occurs during a migraine attack. So when these trigeminal nerves become activated they release a variety of chemicals. These chemicals are proinflammatory: neurokinin A, substance P, calcitonin gene-related peptide, for example. These fall onto the artery wall. They make them vasodilate and swell. About 70 years ago, a then-graduate student at Harvard, Leão, when he applied potassium and excitatory amino acids to the cortex of rabbits, they developed a wave which went from the back of the brain to the front at about 3 mm a minute. And this was called cortical spreading depression. Perhaps it should have been called cortical spreading activation, because the original wave is activated, but as it moved forward it left the brain in a state of depression. This is another way of explaining migraine. Perhaps people with migraine have a cortex that's very easily activated and when it becomes activated you see release of excitatory amino acids, nitric oxide, potassium, calcitonin gene-related peptide, all on the meninges, all as a result of cortical spreading depression. That information is transmitted through the trigeminal nerve, largely the first division of the trigeminal nerve - perhaps that's why you have pain in the eye - that then descends into this trigeminal nucleus caudalis region, exciting the parasympathetics through the superior salivatory nucleus, resulting in eye tearing and nasal congestion. This information then travels back up into the meninges, through the sphenopalatine ganglion, causing more inflammation and more vessel dilation. And this, perhaps, is the arc that occurs during a migraine attack.

Announcer:

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