Pain’s Labyrinth
By Katie Ransohoff

A bright, purple-pink light crept into the corners of her visual field, edging closer to the center. Then came the sensation of partial blindness, like having an extraordinarily bright photograph was taken, and its imprint would not leave. She was disoriented, scared. Within a few minutes, her vision to returned along with nausea and a throbbing headache.

This is how one student described her first migraine. More than just a simple headache, migraines can last from four hours to a few days and are often accompanied by nausea and increased sensitivity to light and sound (1). About 10-25% of the 28 million migraine sufferers also experience auras, which are aberrant visual sensations as described above (2). Author Oliver Sacks describes the auras of the migraines he has had since childhood as “tiny branching lines, like twigs, or geometrical structures covering the entire visual field: lattices, checkerb oards, cobwebs, and honeycombs” (3). The visual experiences of patients are similar to those of schizophrenics and seizure patients, sparking research interest into their similarities. Current treatment is limited to treating the symptoms of migraines, mostly pain relief, since the causes of migraines and auras are largely unknown.

Recent research has offered new insight into the possible causes of migraines. Until recently, it was generally thought that a migraine is a vascular, or blood vessel, disorder. The reasoning was that perhaps a blood flow abnormality triggers the “throbbing pain” associated with migraines (1). However, research now suggests that migraines are genetic and are caused by external triggers such as changes in barometric pressure or flickering lights. These environmental changes can then trigger neuronal misfirings that can lead to concurrent activation of neurons involved in cerebral pain (2).

One study used single fiber electromyography (SFEMG) to look at muscular function during task performance in migraine sufferers. Researchers had migraine patients perform a simple movement task in three-dimensional space that involved moving their arm from a resting position to a point in front of them. They found that in patients who had aura migraines, there was a correlation between the precision of the arm movements and the muscle fiber disturbance, which occurs when the fiber moves arrhythmically. This disturbance in normal smooth motion of the muscles suggests that aura migraines may be in part caused by a dysfunction in ion channels important for smooth transmittance of signals from neurons to muscle cells. In families suffering from migraines, genetic tests have revealed mutations in the CACNA1A gene, a gene coding voltage-dependent calcium channels, offering support to the hypothesis. This new finding could lead to a drug that targets these ion channels, thereby reducing the severity of migraines. These drugs could also have fewer side effects than conventional treatment, because of their potential for high specificity.

While much research still needs to be done on the exact causes, genetic testing can provide valuable insight into which mutations lead to migraine and aura formation. Such information can ultimately lead to creation of drugs which target the specific pathways which underlie the symptoms rather than simply treating pain. [11]

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