Space age treatment for seizures may offer hope for migraines that just won’t quit.

The vagus nerve is the longest nerve in the body, over 20 feet long. It starts in the base of the brain and travels along the gastro-intestinal tract to the mid-colon. Recently, researchers have found that stimulating the vagus nerve in the neck with a small electrical stimulator, similar to a cardiac pacemaker, can suppress seizures. In many of the initial studies of the vagal stimulator for the treatment of seizures, patients who also suffered from intractable migraines reported that their headaches diminished or were completely eliminated when they used their vagal nerve stimulator. 2-3 This led researchers to test vagus nerve stimulation for the treatment of migraine and other severe headaches. Early results have been promising.

In acupuncture, stimulation of the point known as ST-9, in the neck has been traditionally used to treat headaches and other problems in the head and neck. It belongs to a group of points known as "Windows to the Sky." This acupuncture point is associated with the carotid artery. The point is located in the anterior lateral region of the neck where the carotid pulse can be felt. The vagus nerve runs with the carotid artery in the neck. This region is known as the carotid triangle. Placement of a TENs electrode in this region can stimulate the vagus nerve through the skin. Some research shows that TENs stimulation of the vagus nerve can suppress seizures, much like the stimulators that are surgically implanted. 2
In previous newsletters we discussed the headache circuit. It consists of the pain sensitive structures of the brain, the collection of nerves in the brainstem called the trigeminal ganglion and connections with the nerves in the upper neck. Another structure known as the hypothalamus is emerging as an additional part of the circuit that causes cluster and chronic migraine headaches. Somatostatin receptor blockade in the posterior hypothalamus. Functional MRI studies show that repeated vagus nerve stimulation at 20 Hz produces the most significant changes in the central nervous system. 4

In migraine patients a chemical in the cerebrospinal fluid called somatostatin is abnormally low. When headaches occur this chemical drops even lower. 8 In the animal model of migraine, somatostatin suppresses activity of the trigeminal ganglion (see headache circuit) 12. This suggests that somatostatin is a headache suppressing agent. 10

Drugs known to antagonize CGRP appear to abort acute migraine attacks. Researchers are encouraged by the potential for CGRP blocking therapy as a new anti-migraine treatment. 10

Another chemical known as calcitonin gene related peptide (CGRP), appears to be related to the acute phase of migraine. 10

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When headaches occur this chemical somatostatin is abnormally low. 8 In the animal model of migraine, somatostatin drops even lower. 8 In the animal model of migraine, somatostatin suppresses activity of the trigeminal ganglion (see headache circuit) 12. This suggests that somatostatin is a headache suppressing agent. Ooctreotide, a synthetic form of somatostatin has been shown to abolish both cluster and migraine headaches. 11

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Thus it appears that the former has the potential to abort an acute migraine attack by blocking CGRP and the later, if taken over time may raise somatostatin levels in migraine patients. As mentioned above headache patients have lower somatostatin levels that actually drop during a headache attack compared with non headache control patients. Lastly the vegetable rhubarb appears to potentially increase levels of somatostatin. 18

References