Migraines- Headaches Condensed Version
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My postgraduate education is in Neurology the following information will help to explain why the treatment I render will be more specific to the brain and how it will have a greater impact on improving Mrs. Kohler's migraines and give her a better quality of life.

Basically in layman's terms brain (left and right hemispheres) projects to the sympathetic portion of the nervous system. This portion causes constriction of blood vessels. When the brain is **dampened on one side (deficit /hemisphericity)** the ability for the brain to keep the sympathetic nervous system in check become less, vasoconstriction occurs which creates the migraine /headache. The following paragraph explains.

In a vascular migraine there is commonly vasoconstriction of the blood vessels in the brain. This vasoconstriction is commonly seen in the Calcarine cortex of the brain. This is the area for visual integration. With vasoconstriction of cerebral vessels there is also a period of anoxic depolarization (loss of O2) whereby the neurons in the Calcarine cortex spontaneously fire resulting in visual disturbances such as auras or tunnel vision. These visual disturbances are not seen by others only the patient.

With long periods of vasoconstriction, there is subsequent fatigue of the sympathetic neurotransmitter system. When this occurs the sympathetic neurons that were controlling the tunica intima and media (layers of the blood vessel wall) the vascular walls lose their control and the vessel dilates. The beating of the heart will expand these vessels and stretch the perivascular pain plexus (which surrounds the brains major blood vessels).

This will depolarize nociceptors / pain receptors and result in a pounding migraine /headache.

Understanding how this physiological shift happened is just as important especially when considering the best treatment for this condition. The frontal cortex projects fibers through the anterior limb of the internal capsule to the pontomedullary reticular formation to activate the descending inhibitory reticulospinal projections. These pathways are responsible for the inhibition of the Intermediolateral cell nucleus (IML) or the sympathetic column.

To treat this, I manipulated the upper cervical supine on the opposite side of the hemisphericity; I activated powerful ascending pathways to the cerebellum. The cerebellum increased the activity of the opposite frontal cortex through thalamic projections. This leads to increased frontal cortical activation. The frontal cortex in turn activates the pontomedullary reticular formation, which inhibits the sympathetic outflow and the patient's clinical profile changed in nanoseconds resulting in keeping the blood vessels in check or open for normal blood flow and the migraine or headache is reduced or never presents itself.