Background: In appropriately selected patients, intranasal surgery can relieve migraine headaches. The objective of this study is to explore the pathophysiology of migraines and its relation to intranasal contact points.

Methods: We reviewed the current literature discussing the pathophysiology of migraine and intranasal contact point headaches, as well as the anatomy of the sinonasal cavity. We propose a theory to explain this relationship between migraines and contact points.

Pathophysiology: C-fibers of the trigeminal nervous system are pseudounipolar and are responsible for carrying dull pain from the head and face along with the parasympathetic system. In migraine patients, when the Pain Stimulation Level (PSL) reaches and surpasses a certain threshold level, Calcitonin Gene Related Peptide (CGRP) is secreted at the neural endings. We identified this threshold level as “Pain Activation Threshold Level” or PATL. Pain control modulation system (suprareperial excitatory/inhibitory, ON cells/OFF cells) dictates the PATL according to the genetic makeup of the individual which is different in each person. Multiple other factors (stress, hormonal imbalance, drugs overuse, etc) have an impact on PATL as well. In a migraine attack, neurogenic inflammation, plasma extravasation and swelling of the surrounding tissue will occur in all the head and neck areas (including area of intranasal contact point), which in turn leads to more pressure between the two intranasal opposing surfaces. This translates to pain between the eyes, behind the eyes, over the forehead and temples. Increased pain will cause the release of more CGRP and leading to more swelling and pain stimulation. In actuality this is a vicious cycle.