Mechanism of Efficacy of Microvascular Decompression for Trigeminal Neuralgia

Letter:

In an article published in the August 2015 issue of WORLD NEUROSURGERY,[1] Tenser argues that the occasional reactivation of Herpes simplex virus (HSV) after surgical treatment of trigeminal neuralgia provides insights into the mechanism for efficacy of these operations. Specifically, he argues that because HSV reactivation has been observed in ablative treatments (radiofrequency lesions, glycerol injections, balloon compressions) and in nonablative treatments (microvascular decompression), then these treatments must share a common mechanism related to an alteration in trigeminal ganglion neuronal function.

The proposal that microvascular decompression works by means of injury to trigeminal neurons (similar to ablative treatments) is not new, although this is the first time it has been framed in the context of HSV reactivation. However, any neurosurgeon that frequently performs microvascular decompression would attest to the fact that pain relief commonly occurs even in cases where the nerve has been barely manipulated as the offending vessel has been mobilized. Similarly, microvascular decompression for hemifacial spasm is highly effective, although no clinical or electrophysiologic injury to the facial nerve is detectable after a successful operation.

An alternative explanation is that microvascular decompression works neither by relieving pressure on the nerve, nor by injuring the nerve, but by eliminating the pulsatility that acts as an ignition stimulus on a chronically injured and demyelinated nerve.[2] In trigeminal neuralgia, demyelination of sensory axons compressed by arterial loops has been well documented.[3] The ignition hypothesis explains how demyelination can result in paroxysmal pain and why such pain is relieved immediately after microvascular decompression.[4] Injured neurons are susceptible to spontaneous or stimulus-induced afterdischarge. Demyelination results in axon-to-axon cross-excitation.[5] Ephaptic cross-talk and crossed afterdischarges result in the synchronization of afterdischarges in a large group of neurons.[6] Recruitments of increasingly large groups of axons in this process produce a chain reaction that terminates in an explosion of pain.[3] Importantly, although chronic compression of the nerve is responsible for demyelination, it is the pulsation of the artery against the demyelinated nerve that is thought to produce the ignition stimulus for the pain paroxysms. The fact that not every pulsation produces a paroxysm has to do with the time constant for the refractory period after massive axonal depolarization.[3] It is, therefore, possible that the elimination of pulsatility and not decompression accounts for immediate pain relief after microvascular decompression (long before any healing of the injured axons can occur).[7,8]

The ignition hypothesis would explain why HSV reactivation may occur after microvascular decompression. Elimination of the pulsatile ignition stimulus would cause immediate disruption of the chain reaction of synchronized afterdischarges and axonal recruitment. Such sudden alteration of the axonal electrochemistry may be sufficient to trigger HSV reactivation in the ganglion neurons. This hypothesis would also explain other apparent paradoxes raised by Tenser, including why microvascular decompression may be effective in some cases of multiple sclerosis, where demyelination of trigeminal axons is caused by multiple sclerosis but the ignition stimulus is still the pulsatile vessel.

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Published online 11 September, 2015; http://dx.doi.org/10.1016/j.wneu.2015.09.005.

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