THE ROLE OF THE SPHENOPALATINE GANGLION ON THE CEREBRAL HEMODYNAMICS

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The Sphenopalatine ganglion (SPG) and its involvement in the regulation of cerebral blood flow has been the subject of debate during the last century. The ganglion is a complex neural center composed of sensory, motor, and autonomic nerves, which makes it difficult to determine its pathophysiology. Several studies have shown that stimulation of the SPG in anesthetized animals induces vasodilation of cerebral arteries and elevations in cerebral blood flow (CBF) as assessed by laser Doppler flow studies, mass spectrometry, or angiography. Stimulation of the SPG had shown to elevate cerebral blood flow (CBF) and to be neuroprotective after permanent, middle cerebral artery occlusion. SPG-stimulation had shown to increase the CBF bilaterally within the normal brain, and reduces the infarct size in the rat permanent suture model. Sphenopalatine ganglion stimulation had helped to lessen and reverse the vasospasm that results from subarachnoid hemorrhage, which was associated with significant vasospasm of both middle cerebral arteries in dogs. The increases cortical blood flow as a result of Stimulation of cerebrovascular parasympathetic nerves is believed to be due Nitric oxide (NO) or a NO containing compound is present in these nerves, and its release may therefore be partly responsible for the flow increase. In addition, transmitters released from the nerves may cause synthesis and release of this compound from the endothelium.

Recent findings have suggested that acetylcholine, which is co-stored with NO in cerebral perivascular nerves, plays a role in modulating NO release, presumably by acting on muscarinic (M2) receptors on nitric oxidergic nerve terminals, that presynaptic M2 receptors on porcine cerebral perivascular nitric oxidergic nerves mediate inhibition of NO release. The inhibition is due primarily to a decreased Ca$^{2+}$ influx through N-type Ca$^{2+}$ channels. Since this method carries a potential for human application, additional studies are warranted to determine the effects on more severe vasospasm and during period of cerebral hypoperfusion.