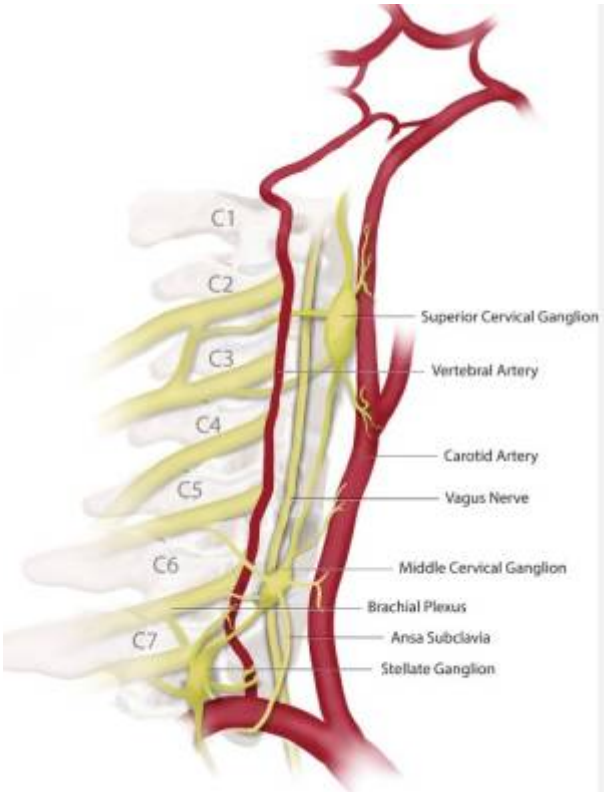
Cervical Sympathetic Nerve Block for cerebral vasospasm



Sympathetic perivascular nerve fibers originate from the superior cervical ganglion (SCG) to innervate the cerebral vasculature, with activation resulting in vasoconstriction. Sympathetic pathways are thought to be a significant contributor to cerebral vasospasm [1].

A simple treatment such as a cervical sympathetic nerve block may be an effective therapy but is not routinely performed as cerebral vasospasm treatment/DCI. cervical sympathetic nerve block consists of injecting local anesthetic at the level of the cervical sympathetic trunk, which temporarily blocks the innervation of the cerebral arteries to cause arterial vasodilatation. cervical sympathetic nerve block is a local, minimally invasive, low cost and safe technique that can be performed at the bedside and may offer significant advantages as a complementary treatment in combination with more conventional neurointerventional surgery interventions. Bombardieri et al. reviewed the literature that describes cervical sympathetic nerve block for vasospasm/DCI prevention or treatment in humans after aSAH. The studies outlined in this review show promising results for a cervical sympathetic nerve block as a treatment for vasospasm/DCI. Further research is required to standardize the technique, explore how to integrate a cervical sympathetic nerve block with conventional neurointerventional surgery treatments of vasospasm and DCI, and study its long-term effect on neurological outcomes [2].

SCG was surgically identified in 15 swine and were electrically stimulated to achieve sympathetic activation. CT perfusion scans were performed to assess for changes in cerebral blood flow (CBF),

cerebral blood volume (CBV), mean transit time (MTT), and time-to-maximum (TMax). Syngo. via software was used to determine regions of interest and quantify perfusion measures.

Results: SCG stimulation resulted in 20-30% reduction in mean ipsilateral CBF compared to its contralateral unaffected side ($p < 0.001$). Similar results of hypoperfusion were seen with CBV, MTT and TMax with SCG stimulation. Prior injection of lidocaine to SCG inhibited the effects of SCG stimulation and restored perfusion comparable to baseline ($p > 0.05$).

Conclusion: In swine, SCG stimulation resulted in significant cerebral perfusion deficit, and this was inhibited by prior local anesthetic injection into the SCG. Inhibiting sympathetic activation by targeting the SCG may be an effective treatment for sympathetic-mediated cerebral hypoperfusion [3].

Hu et al. investigated the therapeutic effects of SGB in a rat model of subarachnoid hemorrhage (SAH) complicated by delayed CVS and explore the underlying mechanisms. The SAH model was established by the double injection of autologous arterial blood into the cisterna magna. They simulated SGB by transection of the cervical sympathetic trunk (TCST), and measured changes in the diameter, perimeter, and cross-sectional area of the basilar artery (BA) and middle cerebral artery (MCA) to evaluate its vasodilatory effect. To investigate the underlying mechanisms, we determined the expression level of vasoactive molecules endothelin-1 (ET-1) and calcitonin gene-related peptide (CGRP) in the plasma, and apoptotic modulators Bcl-2 and Bax in the hippocampus. We found a significant increase in the diameter, perimeter, and cross-sectional area of the BA and right MCA in SAH rats subjected to TCST. Application of SGB significantly reduced the expression of ET-1 while increasing that of CGRP in SAH rats. We also found a significant increase in the expression of Bcl-2 and a decrease in the expression of Bax in the hippocampus of SAH rats subjected to TCST, when compared to untreated SAH rats. The mechanism of action of SGB is likely mediated through alterations in the ratio of ET-1 and CGRP, and Bax and Bcl-2. These results suggest that SGB can alleviate the severity of delayed CVS by inducing dilation of intracerebral blood vessels, and promoting anti-apoptotic signaling. Our findings provide evidence supporting the use of SGB as an effective and well-tolerated approach to the treatment of CVS in various clinical settings [4]

After successful modeling of cervical sympathetic block, 18 healthy male white rabbits were randomly divided into three groups (n=6), ie, sham operation group (Group A), SAH group (Group B) and SAH with cervical sympathetic block group (Group C). Models of delayed CVS were established by puncturing cisterna magna twice with an injection of autologous arterial blood in Groups B and C. A sham injection of blood through cisterna magna was made in Group A. 0.5 ml saline was injected each time through a catheter for cervical sympathetic block after the first injection of blood three times a day for 3 d in Group B (bilateral alternating). 0.5 ml of 0.25% bupivacaine was injected each time through a catheter for cervical sympathetic block after the first injection of blood three times a day for 7 d in Group B. 2 ml venous blood and cerebrospinal fluid were obtained before (T1), 30 min (T2) and 7 d (T3) after the first injection of blood, respectively, and conserved in a low temperature refrigerator. Basilar artery value at T1, T2 and T3 was measured via cerebral angiography. The degree of damage to nervous system at T1 and T3 was recorded.

Results: There was no significant difference in diameter of basilar artery at T1 among three groups. The diameters of basilar artery at T2 and T3 of Groups B and C were all smaller than that in Group A, which was smaller than Group C, with a significant difference. There was no significant difference in

NO and NOS in plasma and cerebrospinal fluid among three groups. The NO and NOS contents at T2 and T3 of Groups B and C were all lower than Group A; Group C was higher than Group B, with a significant difference. The nerve function at T3 of Groups B and C were all lower than Group A and that of Group C higher than Group B, with a significant difference.

Cervical sympathetic block can relieve cerebral vasospasm after subarachnoid hemorrhage and increase NO content and NOS activity in plasma and cerebrospinal fluid to promote neural functional recovery [5)]

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