

Continuous stellate ganglion block in delayed cerebral ischemia: A possible supplementary approach to traditional therapy?

Andrea Bortolato¹, Davide Simonato², Paolo Feltracco¹, Marina Munari³

¹Institute of Anesthesia and Intensive Care Medicine, ²Institute of Radiology, ³Neuroanesthesia and Intensive Care Unit, University Hospital of Padova, Padova, Italy

Abstract

Delayed Cerebral Ischemia (DCI) is a major contributor to morbidity and mortality after SAH. Currently the prevention of vasospasm and DCI relies on nimodipine administration and on maintaining an adequate cerebral perfusion pressure. We report a patient with initial DCI after SAH in which stellate ganglion block (SGB) was performed after nimodipine administration. Firstly the procedure was characterized by a iv and intra-arterial nimodipine administration which did not result into a normal perfusion pattern. Therefore a single-shot stellate ganglion block was performed, as suggested in literature. Because of the not sufficient but promising perfusion improvement, we decided to deliver a continuous ganglion block (cSGB) for 5 days. Consequently a further improvement of the cerebral perfusion on CTPerfusion and Real Time Angiographic Perfusion Assessment was registered. In order to treat cerebral vasospasm, SGB is known to be a further valuable treatment, despite its temporary effect. However the continuous use of SGB during initial DCI has never been described before.

Keywords: DCI, stellate ganglion block, Vasospasm

Delayed cerebral ischemia (DCI) is the major cause of morbidity and mortality in a patient with subarachnoid hemorrhage (SAH). The stellate ganglion block (SGB) has been proposed as an effective rescue treatment of this unfavorable complication. The rationale of the application of SGB relies on the fact that pial vessels are densely supplied with noradrenergic sympathetic nerve fibers, which originate from the superior cervical ganglion, accompanying the carotid artery, and project to the ipsilateral hemisphere.^[1,2] Consequently, cerebral arteries constrict in response to cervical sympathetic stimulation and dilate when sympathetic nerve fibers are damaged or blocked.^[3,4]

Herein, we report a patient with SAH-related DCI treated successfully, first with intravenous (IV) and intra-arterial

nimodipine aided by a single shot SGB, and then with a continuous SGB (cSGB).

A 60-year-old woman was admitted to the emergency department for tachypnea, headache, and impaired level of consciousness (Glasgow Coma Scale [GCS]) = 11; Hunt and Hess Scale = 3, blood pressure = 150/80, heart rate [HR] in the range of 60–70 bpm). A computed tomography (CT) scan of the head and CT angiography revealed SAH (modified Fisher-scale IV) because of a berry-like ruptured anterior-communicating artery aneurysm.

First, the patient underwent endovascular aneurysm repair with coils, and then a left fronto-parietal decompressive

Address for correspondence: Dr. Andrea Bortolato, Anesthesia and Intensive Care University-Hospital of Padua, Via Nicolò Giustiniani n. 2, 35121 Padova, Italy. E-mail: freeandrea.b@libero.it

This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 4.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.

For reprints contact: reprints@medknow.com

How to cite this article: Bortolato A, Simonato D, Feltracco P, Munari M. Continuous stellate ganglion block in delayed cerebral ischemia: A possible supplementary approach to traditional therapy? *J Anaesthesiol Clin Pharmacol* 2020;36:265-7.

Submitted: 04-Aug-2019 **Revised:** 08-Oct-2019 **Accepted:** 24-Oct-2019
Published: 15-Jun-2020

Access this article online	
Quick Response Code:	Website: www.joacp.org
	DOI: 10.4103/joacp.JOACP_251_19

craniotomy to decrease the intracranial pressure (ICP). IV nimodipine therapy (2 mg/hi.v.) was administered under the strict control of hemodynamic monitoring for the prevention of cerebral vasospasm.

On day 13, the patient's symptoms worsened, with fever, tachycardia, a reduction from 9 to 4 of GCS, a severe increase in serum C reactive protein (240 mg/L), and no changes of procalcitonin level. Marked changes of C Reactive Protein are considered by some authors as a sensitive marker of cerebral vasospasm,^[5] and are evaluated by our center in this setting. A diagnostic suspicion of cerebral vasospasm required an emergent CT perfusion that showed under-perfusion of some areas in frontal lobes. There was a distal narrowing of the right anterior and middle cerebral artery branches on CT-angiography (CTA), and these findings were also confirmed by digital subtraction angiography. Therefore, an intra-arterial bolus of 3 mg nimodipine was injected into the right internal carotid artery. A two-dimensional (2D)-perfusion-analysis (Allura Clarity, Philips Healthcare, Best, Netherlands) was also performed to evaluate the possible changes of the cerebral blood flow parameters after the bolus. Despite nimodipine bolus, the mean transit time was found to be still high (4.5 sec) in the watershed territories between the right anterior and middle cerebral arteries.^[6] Hence to manage vasospasm and to improve the cerebral perfusion a right-sided SGB was administered using 20 ml bolus of ropivacaine 0.5% injected at C7- T1 level through a 24-gauge fluoroscopically guided needle via anterior paratracheal approach. The injection of a relatively high volume (20 ml) of a highly concentrated anesthetic solution (0.5%) was preferred to both improve the spread of anesthetic solution to cervical and thoracic ganglia and prolong the effect of single shot block.

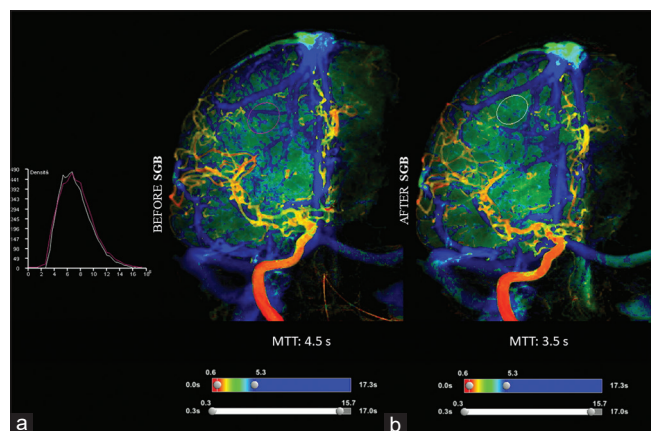


Figure 1: Two-dimensional perfusion angiography after nimodipine (a) and after 20 min (b) from the single cervicothoracic ganglion block (b). The decrease of the mean transition time parameters found in the sample region of interests demonstrates a further significant increase of peripheral perfusion despite the good results obtained by the only intra-arterial administration of nimodipine

Fifteen minutes after the block, the 2D-perfusion-analysis revealed an improvement of the mean transit time from 4.5 sec to 3.0 sec [Figure 1]. Considering that the SGB was supposed to last a maximum of 20–24 h, we decided to implement a continuous SGB, with the purpose to prevent the continuous activation of sympathetic nerve fibers. A right perineural catheter was placed with the aid of ultrasound and fluoroscopy (Pajunk Medizintechnologie, Geisingen, Germany), maintaining the same site of access to the SGB [Figure 2]. The 48-h CT-perfusion imaging revealed a normal perfusion pattern with no further ischemic areas and a CTA showed normal cerebral blood flow. Continuous infusion of ropivacaine 0,2% at 5 ml/h was released for five days, till the time inflammatory markers normalized.

The patient was therefore discharged, with GCS of 10 and right hemiplegia. At a 12-months follow-up visit, only moderate disability was assessed (modified Rankin Scale = 3).

SGB decreases cerebral blood flow velocity, which in turn improves cerebral blood flow and perfusion thereby improving the clinical neurological signs. Moreover, SGB may produce a significant decrease in zero flow pressure, which is an indirect marker of cerebral vascular tone,^[7] as well as providing an improvement of regional cerebral oxygenation.^[8]

As a result, encouraging effect on perfusion by SGB prompted us to try to guarantee this effect over the 24 hours with a continuous peri nervous stellate block, thus avoiding the complications of repeated angiography or the positioning of an intra-arterial microcatheter for continuous nimodipine infusion.^[9] Complications are rare but can be life-threatening. The incidence of severe complications was 1.7 in 1000

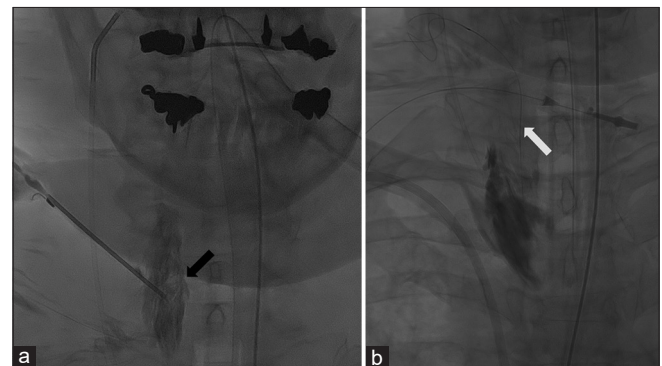


Figure 2: (a) Fluoroscopically guided procedure of continuous stellate ganglion block—the needle is accurately placed anterolaterally to the C7 vertebral body, near the right-sided cervicothoracic ganglion, using the anterior tubercle of the C6 vertebral as a landmark to find the ganglion. Contrast injection has been useful for the tracking of the local anesthetic down the prevertebral fascia to the stellate ganglion below (black arrow). (b) The microcatheter (white arrow) has been correctly placed near the ganglion to guarantee the continuous stellate ganglion block through the infusion of local anesthetic for at least 5 days

blockades after performance of 45,000 SGBs, and most of these were neurological complications (i.e., convulsions). A high subarachnoid block was reported in six cases, high epidural blockade in three, pneumothorax in nine, and allergic reactions in two.^[10]

Eventually, whenever invasive medical approaches fail or are temporarily inapplicable SGB may be considered as a rescue modality in the setting of DCI.^[3]

Informed consent

Informed consent was obtained from the individual participant included in the study.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient has given her consent for her images and other clinical information to be reported in the journal. The patient understands that her name and initials will not be published and due efforts will be made to conceal her identity, but anonymity cannot be guaranteed.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

References

1. Edvinsson L. Neurogenic mechanisms in the cerebrovascular bed. Autonomic nerves, amine receptors and their effects on cerebral blood flow. *Acta Physiol Scand Suppl* 1975;427:1-35.
2. Tuor UI. Local distribution of the effects of sympathetic stimulation on cerebral blood flow in the rat. *Brain Res* 1990;529:224-31.
3. Treggiari MM, Romand J-A, Martin J-B, Reverdin A, Rüfenacht DA, de Tribolet N. Cervical sympathetic block to reverse delayed ischemic neurological deficits after aneurysmal subarachnoid hemorrhage. *Stroke* 2003;34:961-7.
4. Hu N, Wu Y, Chen BZ, Han JF, Zhou MT. Protective effect of stellate ganglion block on delayed cerebral vasospasm in an experimental rat model of subarachnoid hemorrhage. *Brain Res* 2014;1585:63-71.
5. Romero FR, Cataneo DC, Cataneo AJM. C-reactive protein and vasospasm after aneurysmal subarachnoid hemorrhage. *Acta Cir Bras* 2014;29:340-5.
6. Jain V, Rath GP, Dash HH, Bithal PK, Chouhan RS, Suri A. Stellate ganglion block for treatment of cerebral vasospasm in patients with aneurysmal subarachnoid hemorrhage-A preliminary study. *J Anaesthesiol Clin Pharmacol* 2011;27:516-21.
7. Gupta MM, Bithal PK, Dash HH, Chaturvedi A, Mahajan RP. Effects of stellate ganglion block on cerebral haemodynamics as assessed by transcranial Doppler ultrasonography. *Br J Anaesth* 2005;95:669-73.
8. Zhang Y, Qian Y, Bao H, Shi H, Zhou J. [Effect of stellate ganglion block on bilateral regional cerebral oxygen saturation and postoperative cognitive function]. *Sheng Wu Yi Xue Gong Cheng Xue Za Zhi* 2016;33:132-5.
9. Biondi A, Ricciardi GK, Puybasset L, Abdenour L, Longo M, Chiras J, *et al.* Intra-arterial nimodipine for the treatment of symptomatic cerebral vasospasm after aneurysmal subarachnoid hemorrhage: preliminary results. *AJNR Am J Neuroradiol* 2004;25:1067-76.
10. Wulf H, Maier C. [Complications and side effects of stellate ganglion blockade. Results of a questionnaire survey]. *Anaesthesist* 1992;41:146-51.