

Stellate ganglion block for treatment of cerebral vasospasm in patients with aneurysmal subarachnoid hemorrhage – A preliminary study

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Abstract

Background: Stellate ganglion block improves cerebral perfusion by decreasing the cerebral vascular tone. Its effects on cerebral vasospasm to relieve neurological deficits have not been evaluated. This prospective observational study was carried out to evaluate the effect of stellate ganglion block on cerebral hemodynamics in patients with symptomatic cerebral vasospasm following aneurysmal subarachnoid hemorrhage.

Materials and Methods: Fifteen patients of either sex, aged 18-75 years, who underwent surgical clipping of aneurysm and developed refractory cerebral vasospasm were included. Stellate ganglion block was performed using 10 ml of bupivacaine 0.5% on the side with maximum cerebral blood flow velocity. Neurological status, cerebral blood flow velocity and pulsatility index were assessed before and 10 minutes, 30 minutes, 2 hours, 6 hours, 12 hours and 24 hours after stellate ganglion block.

Results: Improved Glasgow coma score was observed 30 minutes after stellate ganglion block. Neurological deficits reduced in 11 patients. Ipsilateral middle cerebral artery mean flow velocity decreased from 133.66 cm/sec before stellate ganglion block to 110.53 cm/sec at 6 hours ($P<0.001$) and 121.62 cm/sec at 24 hours ($P<0.001$) after stellate ganglion block. There was a decrease in ipsilateral anterior cerebral artery mean flow velocity after stellate ganglion block ($P<0.001$), which persisted for 12 hours. A decline in flow velocities was observed in contralateral middle cerebral artery ($P=0.008$) and anterior cerebral artery ($P=0.041$) for 12 hours.

Conclusion: This study suggests stellate ganglion block to be an effective modality of treatment for refractory cerebral vasospasm after aneurysmal subarachnoid hemorrhage.

Key words: Cerebral blood flow velocity, cerebral vasospasm, stellate ganglion block, subarachnoid hemorrhage, transcranial Doppler

Introduction

Cerebral vasospasm is a recognized complication of subarachnoid hemorrhage (SAH). Angiographically detected vasospasm is more common, occurring in two third of patients, than symptomatic vasospasm (clinical evidence of cerebral ischemia). Symptomatic vasospasm may occur in 46% of

patients.^[1] Mortality is increased by 1.5-3 fold in the first 2 weeks after SAH due to cerebral vasospasm.^[2] It is also a major cause of morbidity in aneurysmal SAH patients.^[3] Management modality of vasospasm includes nimodipine,^[4] hypervolemia, hypertension, hemodilution (HHH) therapy,^[5] interventional neuro-radiological procedures like transluminal angioplasty^[6] or intra-arterial administration of vasodilators.^[7] These measures are associated with medical complications or may have procedural limitations. Newer options like magnesium, statins, endothelin antagonists, and fibrinolytic therapy are under investigation but large trials are awaited to prove their efficacy.^[8] The effect of stellate ganglion block (SGB) on cerebral hemodynamics was recently assessed using transcranial Doppler (TCD). There was a significant increase in cerebral perfusion pressure due to decrease in cerebral vascular tone.^[9] It is not yet known whether these effects of SGB would help relieve delayed ischemic neurological deficits in symptomatic cerebral vasospasm. A prospective study was carried out to evaluate the effect of SGB in surgically clipped aneurysmal SAH patients with symptomatic cerebral

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vasospasm. The primary aim was to assess any change in clinical status, and the secondary objective was to assess the changes of cerebral blood flow velocity (CBFV) by TCD.

Materials and Methods

After institutional ethics committee approval and informed written consent from next of kin, patients of either sex, aged 18-75 years suffering from aneurysmal SAH, who underwent surgical clipping, and developed symptoms of cerebral vasospasm were studied over a period of 2 years. Symptomatic vasospasm was defined as new onset of focal neurological deficit or deterioration in the level of consciousness after excluding other possible causes such as re-bleeding, hydrocephalus, surgical complication, cerebral edema, electrolyte disorder, infection and seizure. TCD confirmation of cerebral vasospasm (mean CBFV > 120 cm/sec) in any vessel, and clinical correlation was done before enrolling patients into the study. Exclusion criteria were asymptomatic patients with vasospasm, patients with more than one aneurysm, refusal of consent, history of allergy to local anesthetic agents, disturbed coagulation profile, and patients with pre-existing pupillary changes where assessment of effectiveness of SGB would be difficult.

All patients underwent surgical clipping of aneurysm and were treated in the postoperative period with a standard treatment protocol that included intensive care monitoring, maintaining normotension, fluid therapy to maintain normovolemia (positive fluid balance > 500 ml/day), and spontaneous hemodilution to maintain a hematocrit of 30%. Normothermia (35.5°C-36.5°C) was maintained during the study period. All patients received oral nimodipine 60 mg every 4 hours. TCD study was performed daily by an experienced neuro-intensivist. On clinical suspicion of vasospasm, a diagnostic brain computed tomographic (CT) scan was done to rule out ventricular dilatation or focal brain lesion explaining the clinical symptoms. Then, HHH therapy was instituted. Hypervolemia was instituted with administration of colloids and crystalloids with volume infusion up to 3-4 L/day with a targeted central venous pressure of 10-12 mmHg. The systolic blood pressure was targeted to 160-200 mmHg. This was achieved with infusion of vasopressors (dopamine or noradrenaline). The lowest hematocrit allowed was 30%. If symptoms of cerebral vasospasm did not improve after 24 hours of HHH therapy, decision was taken to enroll the patient into the study.

Five-lead electrocardiogram, invasive arterial blood pressure, end-tidal carbon dioxide and arterial oxygen saturation were monitored continuously for 24 hours. Neurological status was assessed by an independent observer (neurosurgeon) who was

unaware of the study. Middle cerebral artery (MCA) and anterior cerebral artery (ACA) on both sides were insonated through the temporal acoustic window using two 2 MHz transcranial Doppler ultrasound probes (Four-View™, Rimed Ltd, Israel) with separate TCD machines. The TCD probes were fixed at a constant angle using a headband, and a stable continuous tracing of waveform of blood flow velocities in vessels was established. Vessels were identified and confirmed using standard criteria. Baseline observations including neurological condition [Glasgow coma scale (GCS) score, motor deficits], hemodynamic parameters (heart rate, mean blood pressure), and TCD values [peak, mean, diastolic blood flow velocity, pulsatility index (PI)] in MCA and ACA on both sides were recorded. Under aseptic precaution, SGB was performed at C₆ level, using anterior paratracheal approach, on side showing maximum mean CBFV. Bupivacaine (0.5%) 10 ml was administered. After 10-15 minutes, onset of SGB was confirmed by the presence of an ipsilateral Horner's syndrome, ipsilateral increase in skin temperature.^[10]

Baseline hemodynamic and TCD parameters were reassessed by the same observer at 10 minutes, 30 minutes, 2 hours, 6 hours, 12 hours and 24 hours after SGB. Simultaneously neurological status was assessed by the independent observer. Sedative medications were avoided during the study period. However, in mechanically ventilated patients, propofol 2-3 mg/kg/hr was administered whenever deemed necessary. It was ensured that propofol administration discontinued at least 1-2 hr prior to the observation so that no residual sedative effects were observed. In mechanically ventilated patients, ventilator settings were not changed during the study period. A hematocrit of 30 ± 5% and temperature (35.5°C-36.5°C) was maintained throughout the study period.

Survivors were followed after discharge during their subsequent hospital visits. Outcome was assessed according to the Glasgow outcome scale (GOS) at 6 months after SAH. The proportions of patients achieving good recovery or moderate disability were considered having favorable outcome.

Data was analyzed using STATA 9.1 (College Station, Tx, USA). Results were expressed as mean (standard error). Generalized estimating equation (GEE) was applied to compare the difference in mean values over different time intervals since the observations were correlated. A *P* value < 0.05 was considered statistically significant.

Results

A total of 102 patients with diagnosis of aneurysmal SAH undergoing surgical clipping were admitted to our neuro-intensive care unit during the study period. Out of 38 patients

with clinical suspicion of cerebral vasospasm, 23 did not meet the inclusion criteria. The remaining 15 patients with TCD-confirmed vasospasm were included in the study [Table 1]. The mean age was 45.5 ± 13.6 years, and mean weight was 60.5 ± 7.9 kg. SGB was successful in all patients. Signs of sympathetic blockade (Horner's syndrome, increase in ipsilateral skin temperature) were evident on the side of block after 11.2 ± 0.2 minutes of local anesthetic administration. The average duration of sympathetic block was 6.8 ± 5.0 hours. No change in heart rate and blood pressure were observed during the study period.

An improvement in GCS score was observed after 30 minutes of SGB ($P=0.002$) that persisted for 24 hours. There was a decrease in CBFV on ipsilateral side ($P<0.001$). The flow velocity in ipsilateral MCA decreased from $133.67(7.65)$ cm/sec prior to SGB to $110.53(5.78)$ cm/sec at 6 hours after block ($P<0.001$). Though, a slight increase in CBFV, $121.62(5.91)$, was observed at 24 hours as compared to 6 hours after SGB, still the CBFV were significantly lower in comparison to the baseline value ($P<0.001$) prior to SGB. There was a decrease in ACA flow velocity from $106.07(6.42)$ before SGB to $95.76(4.81)$ 12 hours after SGB ($P<0.001$) [Table 2]. An increase in ipsilateral PI of MCA was observed after 30 minutes of SGB ($P=0.001$), which remained elevated for 24 hours [Table 2]. On the contralateral side of block, a significant decrease was observed in mean MCA

($P=0.008$) and ACA flow velocity ($P=0.041$) for 12 hours [Table 3]. No significant change was observed in PI of contralateral MCA and ACA except increase in PI of MCA at 24 hours [Table 3]. No complication was observed during study period.

Favorable outcome (GOS) at six months after SAH was seen in 11 (73%) patients. Three patients died and one remained vegetative. The cause of death in two patients was sepsis whereas another patient had severe vasospasm.

Discussion

Our results demonstrate a temporal correlation between the effect of SGB and decrease in CBFV with concomitant improvement of neurological status. The use of SGB to improve cerebral blood flow for treatment of cerebrovascular events is not new.^[11] In our study, using TCD, we have more objectively demonstrated the efficacy of SGB in increasing cerebral blood flow, as evident from the decrease in CBFV and clinical improvement in neurological status. Gupta *et al.* demonstrated that SGB produced a significant decrease in zero flow pressure, which is a surrogate marker of cerebral vascular tone, leading to improved cerebral perfusion pressure.^[9] This is the logical explanation for clinical improvement in our patients. These findings also support the suggested use of superior cervical sympathetic

Table 1: Clinical details of the patients

Gender/ Age	Hunt and Hess grade	WFNS grade	Fisher grade	Aneurysm location	GCS score on admission	Days after SAH for onset of vasospasm	Symptoms	Outcome
M/60	3	1	3	MCA	15	9	Decreased GCS	Moderately disabled
F/55	5	4	4	ACom A	10	6	Decreased GCS	Good recovery
M/40	3	2	3	ACom A	14	4	Lt arm weakness	Good recovery
F/44	3	4	3	ACom A	12	8	Decreased GCS, Rt arm weakness	Moderately disabled
F/17	2	1	3	ACom A	15	5	Decreased GCS, Rt leg weakness	Good recovery
F/35	5	4	4	Right ICA	9	4	Decreased GCS, Lt hemiparesis	Moderately disabled
F/65	5	4	3	ACom A	11	4	Decreased GCS	Moderately disabled
F/34	3	1	3	Left ICA	15	4	Decreased GCS, Rt hemiparesis	Good recovery
M/58	4	1	3	ACom A	15	8	Decreased GCS, Lt arm weakness	Moderately disabled
M/40	3	2	3	Anterior choroidal A	14	6	Decreased GCS	Good recovery
F/55	5	4	4	Lt MCA	11	4	Decreased GCS	Death
M/36	2	1	3	Rt MCA	15	3	Lt hemiparesis	Death
M/38	3	1	3	ACom A	15	5	Decreased GCS, Rt hemiparesis	Death
F/60	5	4	4	ACom A	10	5	Decreased GCS	Vegetative
M/28	2	1	3	ACom A	15	3	Lt leg weakness	Good recovery

MCA = Middle cerebral artery; Acom A = Anterior communicating artery; ICA = Internal carotid artery; GCS = Glasgow coma score; SAH = Subarachnoid hemorrhage; WFNS = World Federation of Neurosurgeon; Lt. = Left; Rt. = Right; M = Male; F = Female

Table 2: Observed transcranial Doppler parameters ipsilateral to Stellate Ganglion Block (Data are presented as mean ± standard error)

n=15	Baseline	10 minutes	30 minutes	2 hours	6 hours	12 hours	24 hours
MCA BFV							
Mean(SE)	133.66 (7.65)	126.53 (6.82)	120.00 (6.07)	112.60 (5.70)	110.53 (5.78)	113.33 (5.86)	121.62 (5.91)
Difference [§]		7.13	13.66	21.06	23.13	20.33	12.04
95 % CI		(4.81,9.45)	(9.54,17.78)	(16.34, 25.79)	(18.19, 28.07)	(14.37, 26.29)	(6.10, 17.99)
P value		< 0.001*	< 0.001*	< 0.001*	< 0.001*	< 0.001*	< 0.001*
ACA BFV							
Mean(SE)	106.07 (6.42)	102.84 (5.70)	98.92 (5.47)	95.53 (4.61)	94.38 (4.46)	95.76 (4.81)	104.76 (5.27)
Difference [§]		3.23	7.15	10.53	11.69	10.30	1.30
95 % CI		(1.38,5.07)	(4.72,9.58)	(6.31,14.76)	(6.58,16.79)	(4.89,15.71)	(3.26,5.88)
P value		0.001*	< 0.001*	< 0.001*	< 0.001*	< 0.001*	0.576
MCA PI							
Mean(SE)	0.47 (0.02)	0.48 (0.02)	0.52 (0.03)	0.52 (0.03)	0.53 (.03)	0.54 (0.02)	0.55 (0.02)
Difference [§]		0.01	0.047	0.043	0.061	0.066	0.075
95 % CI		(0.01,0.03)	(0.03, 0.06)	(0.00, 0.06)	(0.02,0.10)	(0.02,0.10)	(0.03,0.11)
P value*		0.339	< 0.001*	< 0.001*	0.003*	0.001*	0.001*
ACA PI							
Mean(SE)	0.52 (0.047)	0.53 (0.04)	0.55 (0.03)	0.54 (0.03)	0.53 (0.03)	0.58 (0.02)	0.51 (0.02)
Difference [§]		0.004	0.025	0.019	0.008	0.058	-0.013
95 % CI		(0.02,-0.01)	(0.06,-0.01)	(0.07,-0.03)	(0.07,-0.05)	(0.14,-0.02)	(0.06,-.09)
P value		0.648	0.199	0.460	0.793	0.189	0.744

n = Number of patients [§]Difference in mean values between baseline and at respective time interval; *P value <0.05; Statistically significant; MCA BFV = Middle cerebral artery blood flow velocity; ACA BFV = Anterior cerebral artery blood flow velocity; MCA PI = Middle cerebral artery pulsatility index; ACA PI = Anterior cerebral artery pulsatility index

Table 3: Observed transcranial Doppler parameters contralateral to Stellate Ganglion Block (Data are presented as mean ± standard error)

n=15	Baseline	10 minutes	30 minutes	2 hours	6 hours	12 hours	24 hours
MCA BFV							
Mean(SE)	95.93(3.88)	91.86(4.01)	87.20(4.27)	86.20(4.19)	84.8(4.34)	89.4(4.80)	91.25(4.96)
Difference [§]		4.06	8.73	9.73	11.13	6.53	4.68
95 % CI		(2.88,5.25)	(6.89,10.57)	(7.03,12.42)	(8.20,14.05)	(1.72,11.33)	(1.59,10.96)
P value		< 0.001*	< 0.001*	< 0.001*	< 0.001*	0.008*	0.144
ACA BFV							
Mean(SE)	73.7(5.34)	72.4(5.09)	70.1(4.97)	69.4(5.09)	68.7(4.77)	72.2(5.35)	72.9
Difference [§]		1.30	3.60	4.30	5.00	1.50	0.80
95 % CI		(0.64,1.95)	(2.04,5.15)	(2.80,5.79)	(3.34,6.65)	(0.06,2.93)	(1.36,2.96)
p value		< 0.001*	< 0.001*	< 0.001*	< 0.001*	0.041*	0.468
MCA PI							
Mean(SE)	0.58(0.041)	0.58(0.042)	0.56(0.03)	0.55(0.03)	0.52(0.40)	0.54(0.02)	0.63(0.04)
Difference [§]		0.006	-0.012	-0.024	-0.055	-0.034	0.05
95 % CI		(0.01,0.02)	(0.03, 0.05)	(0.03, 0.08)	(0.01,0.16)	(0.01,0.08)	(0.01,0.08)
P value*		0.512	0.578	0.413	0.133	0.156	0.002*
ACA PI							
Mean(SE)	0.51(0.068)	0.49(0.06)	0.52(0.06)	0.52(0.07)	0.51(0.03)	0.52(0.07)	0.55(0.07)
Difference [§]		-0.012	0.014	0.017	0.008	0.014	0.04
95 % CI		(0.08,-0.03)	(0.02,-0.05)	(0.02,-0.05)	(0.02,-0.03)	(0.02,-0.04)	(0.00,-.08)
P value		0.254	0.477	0.389	0.610	0.442	0.099

n = Number of patients [§]Difference in mean values between baseline and at respective time interval; *P value <0.05; statistically significant; MCA BFV = Middle cerebral artery blood flow velocity; ACA BFV = Anterior cerebral artery blood low velocity MCA PI = Middle cerebral artery pulsatility index; ACA PI = Anterior cerebral artery pulsatility index

block to relieve cerebral vasospasm in patients with SAH. Treggiari *et al.* demonstrated improved cerebral perfusion without change in vessel diameter at cerebral angiography.^[12] The observations of our study are more significant as the patients studied had delayed ischemic neurologic deficits owing to refractory cerebral vasospasm. We have earlier demonstrated the efficacy of SGB in two patient's refractory to papaverine administration.^[13]

Cervical sympathetic nerve activity blockade by SGB improves cerebral blood flow. The stellate ganglion contains the cell bodies of inferior cervical and first thoracic sympathetic ganglion. The cerebral vasculature receives a noradrenergic sympathetic nerve supply mainly through the fibers that originate in cervical ganglion, accompany the carotid artery, and project into ipsilateral cerebral hemisphere.^[14-16] Cerebral vasculature, especially pial vessels, is densely innervated with

noradrenergic sympathetic nerve fibers. Intracerebral vessels constrict in response to cervical sympathetic stimulation and dilate when these fibers are interrupted.^[14,16] Blockade of sympathetic nerve activity or reversal of over-activity may dilate intracerebral vessels improving cerebral blood flow.

There are conflicting reports on the changes in cerebral blood flow following SGB.^[17,18] Theoretically, there should be an ipsilateral increase in cerebral blood flow due to the effect of SGB, but in our study, a significant decrease in CBFV was noted on both sides. The decrease in flow velocity on the contralateral side may be due to improved global cerebral perfusion as a result of increased cerebral blood flow. Cerebral vasospasm, involves cerebral vasculature as a whole. A slight narrowing of all basal arteries takes place leading to increased flow velocities.^[19] Moreover, the stellate ganglion may give projections to contralateral sympathetic pathways.^[20] We chose to give SGB on the side of higher CBFV presuming the affected side to be more at risk of ischemic deficits than the other.

TCD is widely used as a monitoring tool to assess cerebral hemodynamics in patients with cerebral vasospasm. It is non-invasive and can be used repeatedly when required. It has been proved to be an ideal aid to monitor the effectiveness of various therapies instituted for vasospasm. Though cerebral angiography is gold standard for assessing cerebral vasospasm, it cannot be performed at frequent intervals as it is invasive and there is a need of a neuro-radiological suite. We used TCD as a modality for monitoring cerebral vasospasm. Physiological variables like hemodynamic parameters, carbon dioxide concentration, temperature and hematocrit may affect cerebral blood flow, thereby affecting TCD interpretation. All these parameters were maintained in the range recorded prior to SGB in all our patients.

In patients with neurological disorders, disturbances in cerebral vascular reactivity or cerebral autoregulation are associated with poor outcome. It is important that any intervention which is intended to use in patient with neurological disorders should not impair autoregulation. It has been demonstrated that SGB does not impair the cerebral vascular reactivity or cerebral autoregulation.^[9] The present study shows that a drug induced chemical sympathectomy can improve cerebral perfusion in patients with symptomatic cerebral vasospasm. The potential benefits of cervical sympathectomy can be decreased morbidity and mortality attributed to vasospasm and decreased radiation exposure in an interventional neuro-radiology suite. It may help reduce the need for extreme hemodynamic end points in HHH therapy to obtain reversal of symptoms, which are of concern particularly in elderly patients.^[12] Since the morbidity related to vasospasm may be associated with only mild symptoms, an effort should be made to correct even mild

deficits with therapy of minimal adverse effects. The effect of chemical sympathectomy lasts much longer than the action of intra-arterial papaverine and it is thus helpful in dealing with distal and diffuse vasospasm. SGB can be used for the treatment of cerebral vasospasm as an adjunct to standard procedures, or when standard procedures are not applicable or have failed. It should ideally be carried out under fluoroscopic guidance or using ultrasound, to ensure correct placement of needle. The procedure needs to be performed by an expert in patients with compromised condition, as the margin of error is minimal. SGB offers an advantage over superior cervical block being technically relatively easier to administer and as a bedside procedure of low-cost, and familiarity with the technique among the pain physicians, intensivists and anesthesiologists. However, subjective nature of TCD is a limitation to this study.

To conclude, SGB seems to be a simple and minimally invasive technique, effective in improving cerebral perfusion by relieving symptomatic cerebral vasospasm. A large randomized controlled trial is needed to compare its efficacy with other treatment modalities for cerebral vasospasm and to evaluate the potential role of SGB as a single mode of therapy.

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