Prospective Evaluation

# The Effect of Oxygen Administration on Regional Cerebral Oxygen Saturation after Stellate Ganglion Block on the Non-Blocked Side

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**Background:** Stellate ganglion block (SGB) causes sympathetic denervation of the head, neck, and upper extremities. In some studies, it has been reported that cerebral blood flow on the non-blocked side decreases after SGB, so when performing an SGB for pain management of the head, neck, and arm, the increased risk of cerebral ischemia should be considered.

**Objectives:** To examine the influence of administration of oxygen via nasal cannula after SGB on regional cerebral oxygen saturation ($rSO_2$) of the non-blocked and blocked sides using near-infrared spectroscopy (NIRS).

**Study Design:** Prospective observational study.

**Setting:** Outpatient department for interventional pain management at Yonsei University College of Medicine, Seoul, Korea

**Methods:** Thirty-eight patients with disease entities in the head, neck, and upper extremity and 3 volunteers were studied. SGB was performed with 10 mL of 1% lidocaine using an anterior paratracheal approach at the C6 transverse process level. A successful block was determined based on the appearance of Horner syndrome at 15 minutes after SGB. Oxygen was supplied at a rate of 5 L/min via nasal cannula starting 15 minutes after SGB. $rSO_2$, blood pressure (BP), and heart rate (HR) were obtained at 5-minute intervals for 30 minutes using NIRS, a non-invasive blood pressure manometer, an electrocardiogram, and a pulse oximetry.

**Results:** On the non-blocked side, when compared to the baseline values, there were significant decreases in the $rSO_2$ ($P < 0.001$) and after administration of oxygen, there were significant increases of the $rSO_2$ compared to the $rSO_2$ at 15 minutes ($P < 0.001$). The lowest $rSO_2$ at 15 minutes on the non-blocked side recovered to greater than the baseline value 5 minutes after starting oxygen administration. On the blocked side, when compared to the baseline values, there were significant increases at all time points ($P < 0.001$) and after administration of oxygen there were significant increases compared to the $rSO_2$ at 15 minutes ($P < 0.001$). The $rSO_2$ on the blocked side and the non-blocked side were significantly different at 15 minutes ($P = 0.015$). After oxygen administration, there were no significant differences of $rSO_2$ between the 2 sides.

**Limitations:** This study is limited by its sample size and observational design. It is difficult to precisely define the importance of the effect of SGB and oxygen administration on $rSO_2$ change as we did not examine how the intensity of the nerve block changed with the passage of time.

**Conclusion:** SGB leads to decreased cerebral blood flow of the non-blocked hemisphere, and oxygen administration seems to be a simple method to compensate for this response.

**Clinical Trial:** NCT01532713. IRB No.: 4-2011-0358.

**Key words:** Brain ischemia, cerebrovascular circulation, nerve block, oximetry, oxygen, regional blood flow, spectroscopy, near-infrared, stellate ganglion

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**S**tellate ganglion block (SGB), a type of sympathetic block, is a commonly performed procedure for the treatment of patients with vascular insufficiency and pain syndromes of the face, neck, and upper extremities (1,2). The stellate ganglion is formed by the fusion of the inferior cervical ganglia and first thoracic ganglia. Sympathetic preganglionic fibers synapsing with the upper and middle cervical ganglion pass through this stellate ganglion. Sympathetic postganglionic axons generated from the stellate ganglion move along the internal and external carotid and vertebral arteries, and then are distributed over structures within the cranium. These axons are also connected with gray rami communicantes, which join with the cervical nerve to supply the neck and upper extremity (1,2). Because all the sympathetic nerves distributed to the head and neck and a greater part of those in the upper extremity traverse the stellate ganglion, SGB causes sympathetic denervation of the head, neck, and upper extremity, and as a result, it causes a decrease in peripheral vascular resistance and an increase in blood flow to the areas innervated by the stellate ganglion (3).

However, the physiological significance of sympathetic innervation on the cerebral vasculature remains controversial and the effect of sympathetic denervation by SGB on cerebral blood flow has not yet been clarified (3-10). After SGB, while some researchers have reported an increase in cerebral blood flow of the blocked side (3), others have reported there is only increased blood flow in extracranial vessels and there is no significant change in cerebral blood flow of the non-blocked side (5,9). Several studies have shown that cerebral blood flow on the contralateral side decreases (4,10), which may increase the risk of cerebral ischemia on the non-blocked side, and advise that when performing SGB for pain management of the head, neck, and arm, this increased risk of cerebral ischemia should be considered.

The reference method for measurement of cerebral blood flow is the Kety-Schmidt method (11). Other techniques that have been used to measure cerebral blood flow include xenon-enhanced computed tomography (CT), single photon emission computed tomography (SPECT), positron emission tomography (PET), CT perfusion, magnetic resonance imaging (MRI) with perfusion-weighted imaging, and MR spectroscopy (12). In the Kety-Schmidt method, the patient inhales a mixture of $O_2$ and $N_2O$, and blood is sampled using invasive catheters. In imaging techniques, there are several risks including ionizing radiation exposure, anaphylac-

tic reactions to contrast material, and contrast-induced nephropathy. Moreover, the high cost and impracticality of continuous monitoring make it difficult to apply these techniques as a part of routine clinical practice. Transcranial doppler ultrasonography, the main alternative to these methods, is limited to the analysis of flow velocities in large vessels (4).

Cerebral oximetry is the measurement of brain tissue oxygen saturation using near-infrared spectroscopy (NIRS) (13). Regional cerebral oxygen saturation ($rSO_2$) measured by NIRS in the frontal lobe has been used previously to evaluate overall cerebral blood flow (13-15).

In the present study, we examined the effect of oxygen administration via nasal cannula after SGB on $rSO_2$ of the non-blocked and blocked sides using NIRS, which may be a surrogate for overall cerebral blood flow.

## METHODS

This study was approved by the institutional review board, and written informed consent was obtained from all patients. Forty-one patients who were scheduled to receive SGB at our pain management clinic were recruited between January and February 2012. Inclusion criteria were age between 20 and 70 years old; symptoms in the head, neck, and upper extremity; and American Society of Anesthesiologists (ASA) physical status I or II. Three volunteers were included in this study. Patients were excluded if they had previously undergone intracranial surgery or had any history of neurologic, cardiac, or pulmonary disease. The patients with coagulation disorders were also excluded.

Patients were positioned supine with a shallow pillow under their shoulders. $rSO_2$, blood pressure (BP), and heart rate (HR) were obtained at 5-minute intervals after monitoring with NIRS (INVOS 5100; Somanetics Corp., Troy, MI, USA), a non-invasive blood pressure manometer, an electrocardiogram, and a pulse oximetry. SGB was performed with an anterior paratracheal approach at the C6 transverse process level and 10 mL of 1% lidocaine were injected. Oxygen was supplied via nasal cannula at a flow rate of 5 L/min starting 15 minutes after SGB.

$rSO_2$, BP, and HR were measured at baseline and at 5-minute intervals for 30 minutes (5, 10, 15, 20, 25, and 30 minutes) after SGB. A successful SGB was defined based on the appearance of Horner syndrome (ptosis, miosis, and hemifacial anhidrosis) 15 minutes after SGB. If any complications arose during or after performing

SGB, or in the event of an unsuccessful block, the study protocol was terminated.

Based on the results of our previous study (10) in which $rSO_2$ of the non-blocked side significantly decreased 15 minutes after SGB, we hypothesized that after oxygen administration starting at 15 minutes after SGB, the $rSO_2$ on the non-blocked side might increase to greater than the baseline $rSO_2$. To prove this hypothesis, we calculated that 41 patients would be required with 80% power at a significance level of $\alpha < 0.05$.

Statistical analysis was performed using SAS 9.2 (SAS Institute Inc., Cary, NC, USA). Data were shown as mean ± standard error. Comparisons of the parameters with the baseline values at each time point were analyzed using one-way repeated measures ANOVA and Dunnet's test. The changes after oxygen administration compared with the $rSO_2$ at 15 minutes were also analyzed. Comparisons of the blocked side and the non-blocked side at each time point were performed using a paired t-test with Bonferroni post hoc comparisons. $P < 0.05$ was considered statistically significant.
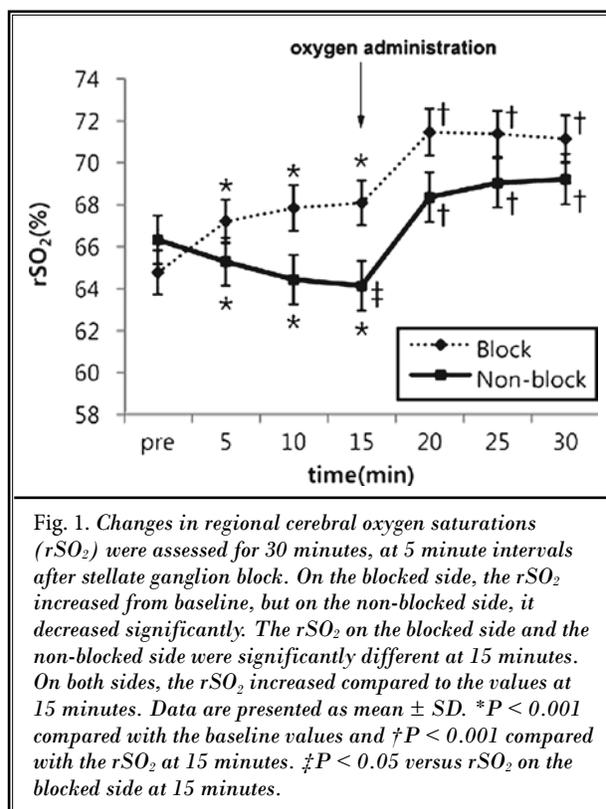
## RESULTS

Forty-one subjects, 14 men and 27 women, with a mean age of 53.5 ± 15.7 years, completed this study. Nineteen subjects underwent right-sided SGB and 22 subjects left-sided SGB. Presentation of the patients' symptoms were facial pain in 12, neck pain in one, shoulder pain in 13, arm pain in 10, hand pain in one, and sudden onset sensorineural hearing loss in one patient. Three people were healthy volunteers. All subjects showed signs of a successful block (Table 1).

On the non-blocked side, when compared to the baseline values, there were significant decreases of the $rSO_2$ at 5, 10, and 15 minutes ($P < 0.001$) and after oxygen administration there were significant increases in the $rSO_2$ at 20, 25, and 30 minutes compared to the $rSO_2$ at 15 minutes ($P < 0.001$). The lowest $rSO_2$ at 15 minutes on the non-blocked side recovered to greater than the baseline value at 5 minutes after starting oxygen administration (Fig. 1).

On the blocked side, when compared to the baseline values, there were significant increases at all time points (5, 10, 15, 20, 25, and 30 minutes, $P < 0.001$, respectively) and after administration of oxygen, there were significant increases at 20, 25, and 30 minutes compared to the $rSO_2$ at 15 minutes ($P < 0.001$).

The $rSO_2$ on the blocked side and the non-blocked side were significantly different at 15 minutes ($P = 0.015$) and the difference between the 2 groups was



Fig. 1. *Changes in regional cerebral oxygen saturations ($rSO_2$) were assessed for 30 minutes, at 5 minute intervals after stellate ganglion block. On the blocked side, the $rSO_2$ increased from baseline, but on the non-blocked side, it decreased significantly. The $rSO_2$ on the blocked side and the non-blocked side were significantly different at 15 minutes. On both sides, the $rSO_2$ increased compared to the values at 15 minutes. Data are presented as mean ± SD. *$P < 0.001$ compared with the baseline values and †$P < 0.001$ compared with the $rSO_2$ at 15 minutes. ‡$P < 0.05$ versus $rSO_2$ on the blocked side at 15 minutes.*

3.95 ± 1.28 (mean ± standard error). At 20, 25, and 30 minutes after administration of oxygen, there were no significant differences of $rSO_2$ of the blocked side and the non-blocked side (Fig. 1). The adjustment for age did not affect the results. After SGB, significant hypotension or bradycardia did not occur in any of the subjects.

## DISCUSSION

This study is the first to examine the effect of oxygen administration on $rSO_2$ after SGB. We found that on the non-blocked side, immediately after SGB, the $rSO_2$ decreased but it recovered within 5 minutes of oxygen administration. Improvements in $rSO_2$ were found on both the block and non-blocked sides.

NIRS is a simple and noninvasive method measuring the $rSO_2$ via an emitted near-infrared light that detects the absorption of oxygenated hemoglobin compared with deoxygenated hemoglobin (16,17). Once the infrared light penetrates living tissue, the relative absorption of the infrared light at different wavelengths is dependent on the concentration of the various hemoglobin species. Based on the relative absorption of the infrared light at various wavelengths,

Table 1. *Demographic data.*

| | |
|---|---|
| Age (yr) | 53.5 ± 15.7 |
| Sex (Male/Female) | 14/27 |
| Weight (kg) | 60.7 ± 10.8 |
| Height (cm) | 160.0 ± 8.1 |
| Location of SGB | |
| Right-sided | 19 |
| Left-sided | 22 |
| ASA physical status | |
| I | 22 |
| II | 19 |
| Patients' symptoms | |
| Facial pain | 12 |
| Neck pain | 1 |
| Shoulder pain | 13 |
| Arm pain | 10 |
| Hand pain | 1 |
| SSNHL | 1 |

SGB: stellate ganglion block, SSNHL: sudden onset sensorineural hearing loss
Data are presented as mean (± SD) or numbers.

the specific concentration of the hemoglobin species can be determined using a modification of the Beer-Lambert law (18).

Jugular venous bulb oxygen saturation ($SjO_2$) reflects the overall balance of cerebral oxygen supply and demand and serves as an indicator of global cerebral oxygenation (19,20). It has been previously demonstrated that $rSO_2$ is proportional to $SjO_2$ and $rSO_2$ has been used to measure the relative change in cerebral blood flow (13,14,21). In this way, although $rSO_2$ measured by NIRS in the frontal lobe does not give the absolute value of cerebral blood flow, it can be used to track the changes in cerebral blood flow indirectly and may be used as a surrogate marker.

There have been several studies on the change of cerebral blood flow after SGB, but it has not yet been clarified, and what is more, the change in cerebral blood flow on the side contralateral to the SGB is unknown. In an experimental study in rats, the intracerebral vessels constrict in response to cervical sympathetic stimulation and dilate when these fibers are interrupted (6). In a study using SPECT, Umeyama et al (3) reported that ipsilateral cerebral blood flow increased after SGB. Ohinata et al (4) measured cerebral blood flow using the ultrasonic doppler method and found that the blood flow of the ipsilateral common carotid artery and vertebral artery increased by 134% and that the blood flow of the contralateral side decreased by 87%, with no change in cardiac output in patients with sudden sensorineural hearing loss. In the present study, while ipsilateral $rSO_2$ increased significantly 15 minutes after SGB, the contralateral $rSO_2$ decreased significantly. These results are consistent with results of the previous studies including the study by Park et al (10).

Conversely, in a study measuring the flow velocity of the carotid and vertebral arteries by MRI using a direct bolus tracking method after SGB, Nitahara et al (5), reported that the blood flow velocity of the ipsilateral common carotid artery increased, that of the vertebral artery did not change, and both the contralateral common carotid artery and vertebral artery were unchanged. Considering this result and the fact that the common carotid artery supplies both cerebral and extracranial vessels, they concluded that SGB mainly causes vasodilation of the ipsilateral extracranial vessel. Furthermore in a study of the change in signal intensity measured by magnetic resonance angiography (MRA) of major cerebral vasculature before and after SGB, Kang et al (9) reported that, after SGB, the blood flow of the extracranial vessels increased, that of the intracranial vessels slightly decreased, and on the contralateral side, both intracranial and extracranial blood flow decreased slightly or remained unchanged.

Our inconsistent results may be due to contamination of the $rSO_2$ value with the signal from blood in the extracerebral tissues, but the possibility is low considering the reliability of the $rSO_2$ values measured by NIRS demonstrated in prior studies (22,23).

With reference to the neurophysiological studies of cerebrovascular tone control, generally cerebral vasculature tone is derived from the cervical ganglion and receives a noradrenergic sympathetic nerve supply which projects into the ipsilateral cerebral hemisphere along the carotid artery (3,6,8). Intracranial extracerebral blood vessels including the pial vessels are innervated by noradrenergic sympathetic nerves that derive from the cervical ganglion and nerves that originate from the sphenopalatine and otic ganglia and the trigeminal ganglion. Therefore, they are under mainly neurogenic control (24). Intraparenchymal blood vessels, however, are controlled by activity of perivascular nerves, which are afferents from the cortical interneuron and the subcortical neuron, and by chemical signals (3,24). The density of adrenergic innervation of the large arteries decreases as their size decreases. Thus, the vessels responding to sympathetic stimulation with constriction

are likely large arteries and arterioles which supply rather sizeable cerebral territories (6). Gotoh et al (25) suggested that cerebral vessels with a diameter greater than 50 μm are under neural control and smaller vessels are controlled chemically in cerebral circulation.

Even though we considered the neurophysiological context, it is difficult to explain why the blood flow of the major intracranial vessels did not change in the study by Kang et al (9). However these results indicate the change in a major vessel, not of the overall flow of the cerebral hemisphere. Therefore, we cannot conclude that the results of our study, which suggest that overall blood flow increases in the ipsilateral cerebral hemisphere and decreases in the contralateral side, are different from that of Kang et al (9).

This point has been discussed previously in the studies by Treggiari et al (7) and Gupta et al (8). In a study using cerebral angiography, Treggiari et al (7) reported that in patients with cerebral vasospasm after subarachnoid hemorrhage, SGB augments cerebral perfusion, which may reduce delayed ischemic neurologic deficits, making it a potential component of the treatment of cerebral vasospasm, a major complication of subarachnoid hemorrhage. As they could not observe the change in the caliber of the major vessels when they performed SGB in patients with vasospasm, they assume that an increased distal cerebral perfusion is the result of peripheral resistance decrease. In a study examining the change of cerebral hemodynamics after SGB using transcranial doppler ultrasound, Gupta et al (8) reported that performing SGB augments cerebral blood flow by decreasing cerebral vascular tone and increasing cerebral perfusion pressure without influencing the capacity of cerebral blood vessels, which produces a response by $CO_2$ change via autoregulation, which could be helpful in preventing and curing cerebral vasospasm. They also observed a slight decrease in flow velocity and considered the possibility of a change in diameter of the middle cerebral artery. They concluded that cerebral vascular tone decreases using the change of zero flow pressure value, a surrogate measure.

If we synthesize the various outcomes, we find that in the current literature, it has been shown that blood flow in the major cerebral vessels does not change significantly but cerebral vascular tone does seem to decrease after SGB. However, the subjects of these studies were limited to patients with cerebral vasospasm or only a unilateral change was observed. Our study demonstrates that the cerebral blood flow of the ipsilateral hemisphere overall increases while that

of the contralateral side decreases after SGB in patients breathing room air without cerebrovascular illness, cardiac disease, and lung disease.

The most impressive finding in the present study is that at 5 minutes after starting oxygen administration, the decreased $rSO_2$ on the non-blocked side recovered to greater than the baseline value, and that the differences in $rSO_2$ values between the 2 groups are no longer significant at 5 minutes after oxygen administration.

In a study using NIRS on patients undergoing carotid surgery, Stoneham et al (26) reported that ipsilateral $rSO_2$ which had decreased during carotid cross-clamping increased to normal after 100% oxygen administration. The etiology of cerebral oxygenation improvement was not clarified, but they hypothesized that it was associated with the increase of $O_2$ content or an increase in cerebral blood flow induced by hyperoxia. In a study of non-hypoxemic patients with chronic obstructive pulmonary disease who desaturated during exercise, Oliveira et al (27) demonstrated that impaired $rSO_2$ corrected with supplemental oxygen, and the result was not due to improved central hemodynamics, but due to enhanced arterial $O_2$ content ($CaO_2$). In the present study, after 5 L/min oxygen administration, the increase of $rSO_2$ in both the ipsilateral hemisphere and the contralateral side was considered to be the result of increased oxygen delivery to the brain, based on a recent study that hyperoxia did not influence the cerebral metabolic rate of oxygen ($CMRO_2$); however, the population studied was patients with acute severe head injury (28). As total oxygen delivery is the product of $CaO_2$ and blood flow, this increased oxygen delivery might be caused by the cerebral blood flow increase due to hyperoxia, or by the $CaO_2$ increase.

But, there is controversy over the effect of hyperoxia on cerebral blood flow. Even though there is experimental evidence that hyperoxia increases cerebral blood flow (29), the opinion that hyperoxia decreases cerebral flow seems to be prevalent (29); however, recent studies have shown that any decrease is likely to be mild (30). Therefore, we conclude that an increase in $CaO_2$ causes an increase in the $rSO_2$ of both the ipsilateral hemisphere and the contralateral side after oxygen administration, compared to the levels measured at baseline.

In a study by Markovitz et al (31), 97% oxygen administered via nasal cannula effectively increased $FiO_2$ by 2.5% per liter of increased flow. In the present study, which utilized 5 L/min oxygen administered via nasal cannula, the effective inspired oxygen concen-

tration is estimated at around 32.7%. Slight hyperoxia increases the $rSO_2$ of both sides after only 5 minutes. In particular, on the non-blocked side, the $rSO_2$ recovered to greater than the baseline values, and became as high as the level measured on the blocked side. This result can be attributed to the features of the vascular beds measured by NIRS. $rSO_2$ determined by NIRS involves measuring a field containing capillaries, arteries, and veins and estimates a combined saturation of arterial and venous blood calculated in proportion to their contribution to cerebral blood volume beneath the NIRS probe. This ratio is approximately 25% arterial blood and 75% venous blood (23,32). According to the sigmoid oxygen dissociation curve of adult hemoglobin, oxygen saturation on the steep part of the curve may vary considerably with a small change in the partial pressure of oxygen. This may explain the rapid increase of $rSO_2$ values with only 5 L/min oxygen administration via nasal cannula. This is also the reason that NIRS can be a sensitive monitor for cerebral oxygenation at a given point.

Determination of the threshold of $rSO_2$, which warrants investigation and potential intervention is needed because the possibility of severe cerebral ischemia is defined not by absolute values, but by the degree of change of $rSO_2$ values. A review of the clinical efficacy of NIRS in cardiac surgery based on 48 studies that used a threshold equivalent to a decrease of 20% from the baseline pre-intervention value to reverse desaturation noted a lower incidence of neurological complications, lower incidence of renal failure, shorter length of intensive care unit stay, shorter total hospital stay, and lower surgical cost (33). In older patients undergoing prolonged major abdominal surgery, cerebral oxygen desaturation of more than 20% from baseline was equally associated with a higher incidence of postoperative cognitive dysfunction and major cerebral dysfunction (34). In addition, some studies have suggested a threshold of 75% of the baseline value or less for severe cerebral ischemia (35), while others have suggested a decrease of 13% or more as a threshold (36). However, Tobias (32) advised that if $rSO_2$ decreases by more than 20% from baseline or reaches 75% or less of the baseline value, investigation and intervention is needed.

## LIMITATIONS

The main limitation of this study is that we did not examine the duration and strength of the effect of SGB, or how the intensity of the nerve block changed with the passage of time. Therefore, it is difficult to precisely define the importance of the effect of SGB and oxygen administration on $rSO_2$ change. However, our aim was to examine the recovery of cerebral oxygenation, so the limitation stated above may be irrelevant. This should be studied further. Relatively small alterations in the partial pressure of arterial carbon dioxide ($PaCO_2$) may cause significant alterations in cerebral blood flow and volume, and therefore, cerebral oximetry value. We did not investigate the effect of $PaCO_2$ on the present study, as we did not perform any arterial blood gas testing. However, we thought that the $PaCO_2$ change was not significant enough to influence the result, as there might be the same effect of the $PaCO_2$ change on both the non-blocked and blocked sides.

## CONCLUSION

In the present study, we confirmed that cerebral blood flow of the hemisphere ipsilateral to the block increases and that of the contralateral side decreases simultaneously after SGB in patients under normoxia without cerebrovascular illness, cardiac disease, and lung disease. In only 5 minutes after providing 5 L/min oxygen via nasal cannula, the decreased $rSO_2$ value of the non-blocked side not only recovered to greater than the baseline value but almost increased to the level of the blocked side. Based on these findings, the authors believe that administration of oxygen could be used as a simple strategy to reduce the possible risk of transient cerebral ischemia caused by a decrease of cerebral blood flow of the non-blocked side after SGB.

## ACKNOWLEDGMENTS

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