

# Effect of Stellate Ganglion Block on the Cerebrovascular System

## Magnetic Resonance Angiography Study

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### ABSTRACT

**Background:** Several studies have shown that stellate ganglion block (SGB) is an effective treatment for certain cerebrovascular related diseases; however, the direct effect of SGB on the cerebral vasculature is still unknown. The present study investigated the effect of SGB on the cerebral vascular system using magnetic resonance angiography.

**Methods:** Time-of-flight magnetic resonance angiography images of 19 healthy female volunteers (mean ages of  $46.4 \pm 8.9$  yr) were obtained before and after SGB with 1.5-T magnetic resonance imaging. The authors determined successful interruption of sympathetic innervation to the head with the appearance of Horner syndrome and conjunctival injection. We measured changes in the average signal intensity and diameter of the major intracranial and extracranial arteries and their branches, which were presented with mean ( $\pm$ SE).

**Results:** The signal intensity changes were observed mainly in the ipsilateral extracranial vessels; the external carotid ar-

tery (11.2%,  $P < 0.001$ ) and its downstream branches, such as the occipital artery (9.5%,  $P < 0.001$ ) and superficial temporal artery (14.1%,  $P < 0.001$ ). In contrast, the intensities of the intracranial arteries did not change with the exception of the ipsilateral ophthalmic artery, which increased significantly (10.0%,  $P = 0.008$ ). After SGB, only the diameter of the ipsilateral external carotid artery was significantly increased (26.5%,  $P < 0.001$ ).

**Conclusions:** We were able to observe significant changes in the extracranial vessels, whereas the intracranial vessels were relatively unaffected (except for the ophthalmic artery), demonstrating that both perivascular nerve control and sympathetic nerve control mechanisms may contribute to the control of intracranial and extracranial blood vessels, respectively, after SGB.

### What We Already Know about This Topic

- ❖ Stellate ganglion block (SGB) has been applied to treat diseases affecting the intra- and extracranial circulation, but its effect on these vascular beds remains controversial

### What This Article Tells Us That Is New

- ❖ In 19 healthy volunteers, SGB increased the diameter of extracranial vessels by time-of-flight magnetic resonance angiography
- ❖ Except for the ophthalmic artery, intracranial vessels were unaffected by SGB

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**S**TELLATE ganglion block (SGB) is a type of sympathetic blockade technique frequently used for a variety of therapeutic, diagnostic, and prognostic purposes, including vascular insufficiency and pain syndromes of the face, neck, and upper extremities.<sup>1–6</sup> It has also been shown to be efficacious for reducing delayed ischemic neurologic deficits caused by the contraction of brain vessels after an aneurysmal subarachnoid hemorrhage<sup>7</sup>; for ocular diseases caused by the acute reduction of blood flow to the eye; and for hot flashes caused by paraneoplastic syndrome and other hormonal imbalances.<sup>8–10</sup>

Many of the diseases affecting the head and face for which SGB is clinically indicated occur mainly because of a reduc-

tion of blood flow in particular cranial vessels.<sup>9</sup> Although there is much interest in the effects of autonomic control on the cerebral vasculature, controversy still exists regarding the physiologic outcome of sympathetic blockade on these vessels. Some studies found an increase in blood flow velocity of the carotid and vertebral arteries after SGB using Doppler ultrasound,<sup>11</sup> whereas other studies reported that sympatholytic substances had little or no effect on cerebral blood flow (CBF).<sup>12–15</sup> Various animal studies have failed to show that sympathetic nerve activity modulates CBF,<sup>16–19</sup> yet others have reported changes in CBF as a result of sympathetic stimulation.<sup>7,20,21</sup> The inconsistencies observed in previous reports may be due to the methods used to investigate the effects of SGB on the cerebral vasculature.

A number of methods have been used to measure changes in blood supply, including single photon emission computed tomography, digital subtraction angiography, transcranial Doppler ultrasound (TCD), and magnetic resonance imaging (MRI). Studies have shown the flow changes in the brain after SGB by single photon emission computed tomography<sup>22</sup>; however, the detection of changes in individual vessels is impossible because of its low spatial resolution. Changes in small blood vessels could be observed with digital subtraction angiography after SGB in patients with aneurismal subarachnoid hemorrhage, providing evidence that SGB have an effect on delayed ischemic neurologic deficits.<sup>7</sup> Single photon emission computed tomography and digital subtraction angiography, however, require the use of contrast agents, such as Tc 99m-labeled perfusion agents or iodinated contrast material. On the other hand, TCD and MRI can measure changes in CBF noninvasively without contrast agents.

Researchers have traditionally employed TCD for SGB studies because of its ability to detect vessel diameter changes and its high temporal resolution, allowing it to easily measure blood flow. However, TCD is limited by its narrow field of view and its inability to visualize smaller cerebral vessels located deep within the brain.<sup>23,24</sup> MRI, on the other hand, can detect CBF and the vessel diameter changes with a reasonable acquisition time (resolution at a time) and is not limited by imaging area or vessel size or location, depending on the imaging resolution. MRI images, however, are sensitive to subject head motion, and because SGB cannot be performed within the MRI scanner, two separate MRI scans are needed for evaluation of the effects of SGB. Therefore, precise positioning is also important to obtain accurate results regarding the changes observed in the small vessels induced by SGB. Previous studies reported the effects of SGB on the cerebrovasculature using MRI, but the results were limited to the large cerebral arteries, and comprehensive changes of the entire blood vessels in human brain were not presented. In this study, we used MRI to examine changes in signal intensity and vessel diameter of the cerebral blood vessels induced by SGB with the aid of fiducial markers and

**Table 1.** MRI Parameters

Parameters	MRA (1 Slab)	MRA (3 Slabs)	MPRAGE
Pixel Size, mm	0.7 × 0.7	0.9 × 0.9	2.0 × 2.0
Slice Thickness, mm	0.7	0.7	2.0
TR/TE/TI, ms	24/7	24/7	1,160/3.88 (600)
Bandwidth, Hz/pixel	130	130	180
Flip Angle, °	22	22	15
FOV, mm	224 × 196	224 × 205	256 × 224
Matrix	320 × 280	256 × 240	128 × 112
Slices Per Slab	48	44	80
TA, min:s	3:46	5:37	1:39

FOV = field of view; MPRAGE = magnetization-prepared rapid gradient echo; MRA = magnetic resonance angiography; MRI = magnetic resonance imaging; TA = acquisition time; TE, echo time; TI, inversion time, TR, repetition time.

other correction techniques to overcome the positioning issue of the subject.

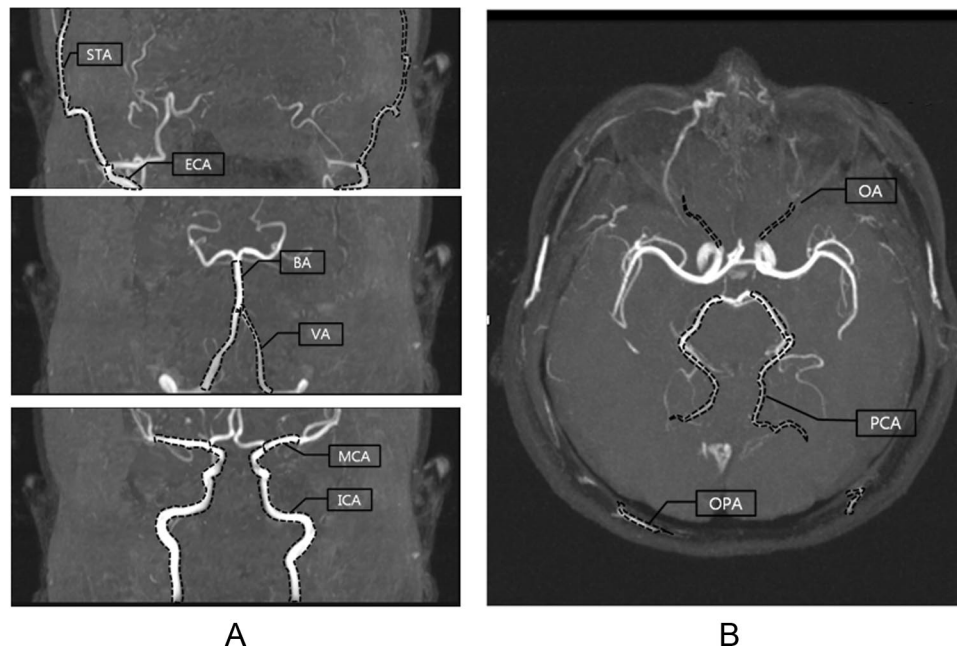
## Materials and Methods

### Subjects and MRI System

Nineteen healthy female volunteers (mean age ±SD, 46.4 ± 8.9 yr) from the local community participated in the study, which was approved by the Gachon University of Medicine and Science institutional review board (Inchon, Republic of Korea). SGB shows great potential as a means of reducing the number of hot flashes and night awakenings suffered by breast cancer survivors and women experiencing extreme menopause.<sup>10</sup> For this reason, this study was to investigate healthy control subjects before the patients were examined. Subjects who had any history of neurologic diseases, intracranial surgery, allergies to local anesthetic agents, cardiovascular problems, or metabolic disorders were excluded. After obtaining informed consent regarding the study's purpose, procedures and risks, MRI was performed before and after SGB using a 1.5-T MRI scanner (Avanto; Siemens, Erlangen, Germany). Subjects were positioned supine inside the bore of the magnet and asked to avoid head movement during the entire experiment. We excluded 4 of 19 subjects for the analysis: one did not show the appearance of Horner syndrome and three had severe image distortion and/or they did not show suitable blood vessel images because of motion artifact and/or MRI system error.

### Imaging Protocols

Time-of-flight magnetic resonance angiography (MRA) and magnetization-prepared rapid acquisition gradient echo were used to investigate the vascular changes induced by SGB, to identify abnormal anatomic brain structures, and to determine the accuracy of the subject's position in the scanner (table 1). The time-of-flight technique is also referred to as "entry slice phenomenon" or "inflow (flow related) enhance-



**Fig. 1.** Vessel regions of interest used for intensity measurements. (A) Coronal maximum intensity projection (MIP) images of a representative subject showing the regions of interest for the internal carotid artery (ICA), external carotid artery (ECA), basilar artery (BA), vertebral artery (VA), middle cerebral artery (MCA), and superficial temporal artery (STA). (B) The regions of interest for the ophthalmic artery (OA), posterior cerebral artery (PCA), and occipital artery (OPA) shown in the axial MIP image.

ment.” Therefore, time-of-flight signal intensity indicates the flow-related enhancement, which depends on several factors, including tissue-specific parameters (*e.g.*, T1), sequence-specific parameters (*e.g.*, flip angle and repetition time), and geometric parameters (*e.g.* slice thickness and orientation or blood flow velocity).<sup>25</sup>

Time-of-flight MRA consisted of two different imaging parameters: one-slab imaging for the intracranial arteries and three-slab imaging for the extracranial arteries. Scout images, magnetization-prepared rapid acquisition gradient echo images and markers were used to assess and correct the three-directional movement of the subject’s head to minimize shifts in position from scan to scan before and after SGB. Before the first MRI scan, three fiducial markers were attached to each subject’s temples and glabella, and their position in relation to the MRI positioning laser was noted. After the scan, SGB was administered in a separate procedure room; after 10 min of bed rest, the subjects reentered the MRI scanner in the same position guided by the positioning laser and the three markers. The blood pressure and tympanic temperature were also measured before SGB and 1 h afterward.

### SGB

A blind technique for SGB was used as described previously.<sup>26</sup> With the subject lying supine, 6 ml of mepivacaine hydrochloride, 1% (Mevan; Hanlim Pharm. Co, Seoul, Republic of Korea) were injected at the anterior tubercle of the sixth cervical transverse process. Subjects in whom the transverse process was not detected easily were excluded. We determined successful interruption of

sympathetic innervation to the head with the appearance of Horner syndrome and conjunctival injection. We recognize that these findings assure only that sympathetic innervation to the head has been blocked, rather than blockade of the stellate ganglion *per se*, but we will use the terminology “SGB” by convention.

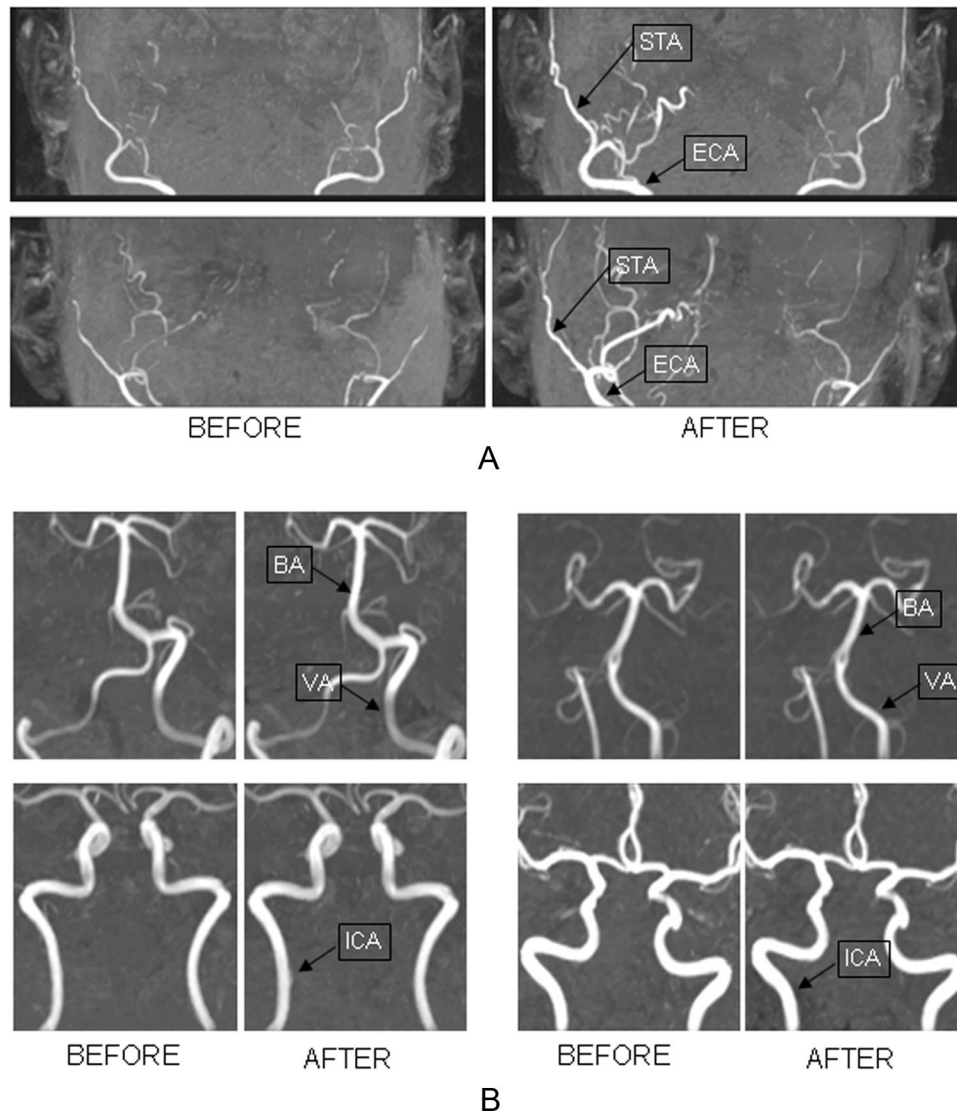
### Data Analysis

We measured the signal intensity changes before and after SGB in nine vessels: the internal carotid artery (ICA), external carotid artery (ECA), superficial temporal artery (STA), middle cerebral artery (MCA), posterior cerebral artery, ophthalmic artery (OA), vertebral artery (VA), basilar artery (BA), and occipital artery (OPA) (fig. 1). The facial artery and meningeal artery (MA) were also investigated; however, their signal intensity changes were not measured, because they were not visible in the MRA images taken before SGB in the majority of subjects. The region of interest of each vessel was selected on an empirical basis. To minimize region-of-interest selection errors, the selection was repeated three times and the values were averaged. We also measured the vessel diameter by determining their full-width half-maximal value using volume imaging in neurologic research, coregistration, and regions-of-interest included software (VINCI; Max-Planck Institute, Köln, Germany).<sup>27</sup>

### Statistics

Differences in signal intensity and vessel diameter before and after SGB were determined using the two-tailed paired *t* test. Statistical significance was defined as  $P <$





**Fig. 2.** Vascular changes induced by stellate ganglion block (SGB). (A) Vascular imaging of the external carotid artery (ECA) group, the ECA and superficial temporal artery (STA), of two representative subjects before and after right SGB treatment. (B) Images of the basilar artery (BA), vertebral artery (VA), and their branches before and after SGB. ICA = internal carotid artery.

0.05. All statistical analysis was carried out using SPSS 15.0 (SPSS Inc., Chicago, IL).

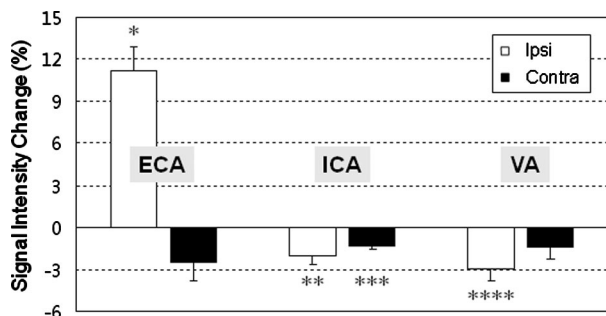
## Results

After adjusting the subject's position with the aid of the positioning laser, the three fiducial markers, and scout image and magnetization-prepared rapid acquisition gradient echo image correction, the average differences in imaging position before and after SGB were  $0.8 \pm 0.7$  mm translationally and  $1.0 \pm 1.1$  degrees rotationally, when magnetization-prepared rapid acquisition gradient echo images were analyzed using SPM2 (Wellcome Department of Cognitive Neurology, London, United Kingdom). These values were sufficiently small such that any effect on the MRI data was negligible with respect to the imaging resolution. The mean blood pressures and core temperatures of the subjects did not

significantly change after SGB ( $P = 0.42$  and  $P = 0.074$ , respectively).

The images of the target vessels before and after SGB are shown in figure 2. Substantial changes were observed in the main trunk of the ECA, and its branches as displayed in figure 2A, whereas the ICA, VA, and BA showed no visible changes induced by SGB in figure 2B.

We measured the signal intensity changes of the major arteries on the ipsilateral and contralateral sides before and after SGB. A significant signal increase was observed in the main trunk of the ECA ipsilateral to the SGB site ( $11.2 \pm 1.7\%$ ;  $P < 0.001$ ) as shown in figure 3. The signal change in the ICA was  $-2.0 \pm 0.6\%$  ( $P = 0.001$ ) ipsilaterally and  $-1.3 \pm 0.3\%$  ( $P = 0.002$ ) contralaterally. In the VA, the change was  $-3.0 \pm 0.8\%$  ( $P = 0.002$ ) ipsilaterally but not significantly changed on the contralateral side (fig. 3). Vessel diameter measurements revealed that only the ip-



**Fig. 3.** Signal intensity changes in the major arteries after stellate ganglion block (SGB). The percentage change of the signal intensity of the major cerebral arteries before and after SGB treatment. The error bars represent SEM. The values are also presented in table 2. \* $P < 0.001$ ; \*\* $P < 0.001$ ; \*\*\* $P = 0.002$ ; \*\*\*\* $P = 0.002$ . Contra = contralateral; ECA = external carotid artery; ICA = internal carotid artery; Ipsi = ipsilateral; VA = vertebral artery.

silateral ECA increased in response to SGB, whereas there were no significant changes in the diameters of the ipsilateral VA, ICA, or the contralateral vessels as summarized in table 2.

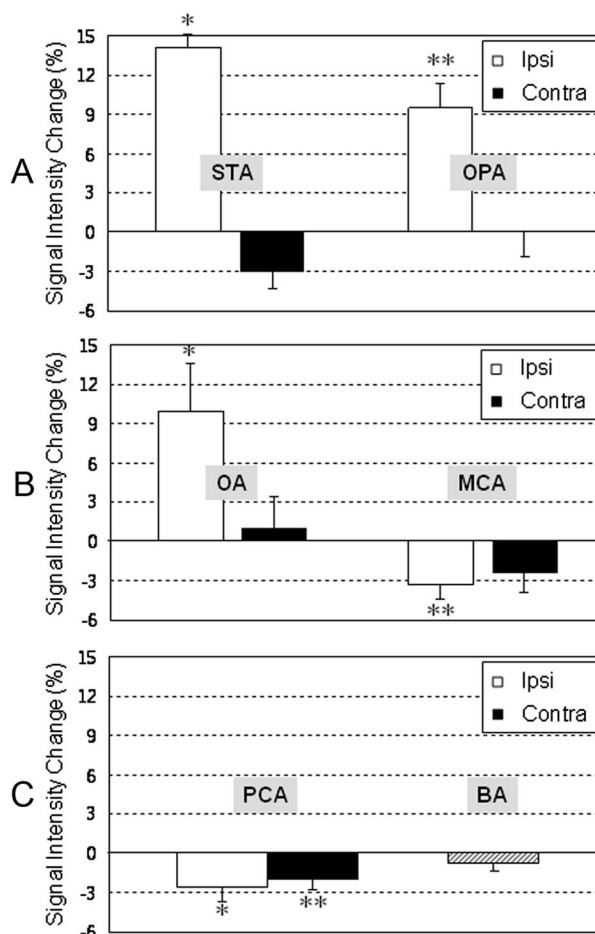
We categorized the daughter vessels of the major arteries into three branch groups for further analysis of the smaller vessels. The ECA group was composed of the STA and OPA, the ICA group included the OA and MCA, and the VA group included the posterior cerebral artery and BA. In the ECA group (fig. 4A), the ipsilateral STA showed substantial increase of signal change after SGB ( $14.1 \pm 2.1\%$ ;  $P < 0.001$ ). The ipsilateral OPA signal was also increased ( $9.5 \pm 1.8\%$ ;  $P < 0.001$ ). The contralateral STA and OPA were not significantly affected. In the ICA group (fig. 4B), the signal of the ipsilateral OA increased significantly ( $10.0 \pm 3.7\%$ ;  $P = 0.008$ ), whereas the signal of the ipsilateral MCA decreased ( $-3.3 \pm 1.1\%$ ;  $P < 0.014$ ). The contralateral OA and MCA were not significantly influenced by SGB. In the VA group (fig. 4C), both the ipsilateral and contralateral posterior cerebral artery signals were slightly decreased ( $-2.6 \pm 1.1\%$ ;  $P = 0.046$  and  $-2.1 \pm 0.7\%$ ;  $P = 0.005$ , respectively). The ipsilateral and contralateral BA showed no significant changes. The statistical analyses for the signal intensity changes of all nine vessels are summarized in table 3.

In addition, we observed striking changes in the ipsilateral MA and facial artery that were rarely detectable by routine

**Table 2.** Changes in Diameter of Intracranial and Extracranial Arteries after SGB Treatment

Artery	IP Change, %	P Value	CL Change, %	P Value
ECA	26.5 ( $\pm 1.5$ )	<0.001	-2.6 ( $\pm 0.8$ )	0.239
VA	-0.3 ( $\pm 0.1$ )	0.337	0.3 ( $\pm 0.3$ )	0.721
ICA	-0.7 ( $\pm 0.4$ )	0.363	-0.9 ( $\pm 0.2$ )	0.337

Change data are presented as mean percentage ( $\pm$ SEM). CL = contralateral; ECA = external carotid artery; ICA = internal carotid artery; IP = ipsilateral; SGB = stellate ganglion block; VA = vertebral artery.



**Fig. 4.** Signal intensity changes in the branches of the major arteries before and after stellate ganglion block (SGB). The percentage changes of the signal intensity of the branches of the external carotid artery (ECA), internal carotid artery (ICA), and vertebral artery (VA) are shown in A, B, and C, respectively. The values are also presented in table 2. (A) \* $P < 0.001$ ; \*\* $P < 0.001$ . (B) \* $P = 0.008$ ; \*\* $P = 0.014$ . (C) \* $P = 0.046$ ; \*\* $P = 0.005$ . BA = basilar artery; Contra = contralateral; Ipsi = ipsilateral; MCA = middle cerebral artery; OA = ophthalmic artery; OPA = occipital artery; PCA = posterior cerebral artery; STA = superficial temporal artery.

MRA before SGB (fig. 5A and B). The MA and facial artery were not quantitatively analyzed because of the inability to visualize these vessels before SGB in several subjects. Representative OA images before and after SGB are also displayed in figure 5C.

**Discussion**

Although sympathetic nerves are known to modulate the response of peripheral arteries and arterioles, the role of sympathetic activity in the induction of CBF change has been controversial. Several studies in animals failed to find a correlation between sympathetic tone and changes in CBF after the surgical denervation of the superior cervical sympathetic ganglion.<sup>16-19</sup> However, many studies in subjects without neurologic disease and studies involving

**Table 3.** Changes in Signal Intensity of Intracranial and Extracranial Arteries after SGB Treatment

Artery	IP, %	P Value	IP, %	P Value	CL, %	P Value	CL, %	P Value
ECA	11.2 ( $\pm$ 1.7)	<0.001*			-2.5 ( $\pm$ 1.3)	0.053		
STA			14.1 ( $\pm$ 2.1)	<0.001*			-3.0 ( $\pm$ 1.3)	0.230
OPA			9.5 ( $\pm$ 1.8)	<0.001*			-0.0 ( $\pm$ 1.8)	0.905
VA	-3.0 ( $\pm$ 0.8)	<0.002*			-1.4 ( $\pm$ 0.9)	0.107		
PCA			-2.6 ( $\pm$ 1.1)	0.046*			-2.1 ( $\pm$ 0.7)	0.005*
BA			-0.7 ( $\pm$ 0.6)	0.428			-0.7 ( $\pm$ 0.6)	0.428
ICA	-2.0 ( $\pm$ 0.6)	0.001*			-1.3 ( $\pm$ 0.3)	0.002*		
OA			10.0 ( $\pm$ 3.7)	0.008*			0.9 ( $\pm$ 2.5)	0.321
MCA			-3.3 ( $\pm$ 1.1)	0.014*			-2.5 ( $\pm$ 1.4)	0.134

\*  $P < 0.05$ .

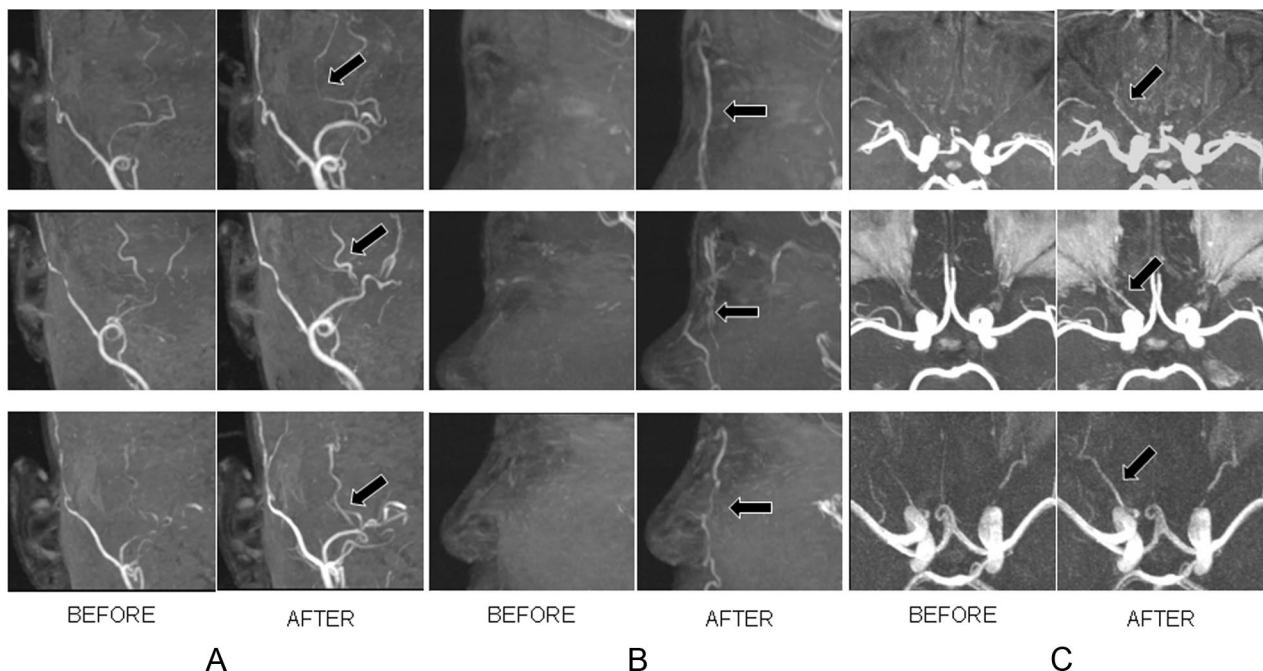
BA = basilar artery; CL = contralateral; ECA = external carotid artery; ICA = internal carotid artery; IP = ipsilateral; MCA = middle cerebral artery; OA = ophthalmic artery; OPA = occipital artery; PCA = posterior cerebral artery; SGB = stellate ganglion block; STA = superficial temporal artery; VA = vertebral artery.

patients with subarachnoid hemorrhage have reported an improvement in cerebral perfusion as a result of increased CBF triggered by cervical sympathetic nerve block.<sup>22,28</sup> The results of these studies were highly dependent on the subjects selected and the experimental methods used; as yet, a comprehensive effort to analyze the physiologic response of the cerebral blood vessels to sympathetic blockade has been lacking.

In the present study, we investigated the cerebral vascular effects of SGB detected by MRA. The results demonstrated that significant changes occurred in the blood flow and diameter of the cerebral vessels after SGB and are summarized in table 4. The signal intensities in the ECA, STA, OPA, and OA ipsilateral to the site of SGB injection were increased by approximately 10% after SGB. A small reduction in the signal intensity of all the intracranial arteries was observed, al-

though only changes in the ipsilateral MCA and ipsilateral and contralateral posterior cerebral artery were significant. The distinctly opposite effects of SGB on the external and internal arteries might arise because of the different types of nerves controlling the blood vessels in these regions; the extracranial vessels are regulated by sympathetic tone, whereas the intracranial vessels are controlled mainly by perivascular nerve activity.<sup>29</sup>

It is noteworthy that the signal intensity of the OA, which is located intracranially and originates from the ICA, was found to increase substantially. This may be because the OA and its peripheral branches are not in contact with brain and supply blood to extracranial organs (*i.e.*, the eyes and orbit), which are regulated by the sympathetic nervous system.<sup>30-32</sup> Moreover, the facial artery and MA, which for the most part could not be detected in MRA images taken before SGB,



**Fig. 5.** Changes in the meningeal, facial, and ophthalmic arteries before and after stellate ganglion block (SGB). Magnetic resonance angiography images of three representative subjects show changes in the meningeal artery (A), facial artery (B), and ophthalmic artery (C) before and after SGB, respectively.



**Table 4.** Ipsilateral Intensity Changes in Intracranial and Extracranial Arteries after SGB

Vessels	Intensity Change
ECA	++
STA	+++
OPA	++
FA	+++
MA	++
ICA	–
MCA	–
OA	++
VA	–
BA	–
PCA	–

– = –5% to 0 change; ++ = 5–10% change; +++ = 10–15% change; BA = basilar artery; ECA = external carotid artery; FA = facial artery; ICA = internal carotid artery; MA = meningeal artery; MCA = middle cerebral artery; OA = ophthalmic artery; OPA = occipital artery; PCA = posterior cerebral artery; SGB = stellate ganglion block; STA = superficial temporal artery; VA = vertebral artery.

were clearly visualized after SGB, underscoring the fact that SGB has a profound effect on the extracranial arteries rather than the intracranial arteries.

The results of this study may explain the therapeutic benefits of SGB for some diseases such as sensorineural hearing loss and ischemic optic neuropathy. Firat *et al.*<sup>33</sup> demonstrated that hearing, especially at higher frequencies, responded to increased cochlear blood flow after SGB and that patients with cochlear hearing disorders, particularly in sudden sensorineural hearing loss, responded to SGB treatment. The increase in signal intensity we observed for the ECA, STA, and maxillary artery (and potentially its branches, such as the anterior tympanic artery, which supplies blood to the adjacent areas of the cochlea and inner ear) may explain some of the benefits observed by others for SGB for hearing loss of unknown etiology.<sup>5,11,34,35</sup> In contrast we found no change in the BA, from which arises the anterior inferior cerebellar artery and the labyrinthine artery, which supplies the inner ear. Liu *et al.*<sup>36</sup> reported that SGB treatment improved the picture (visual) evoked potential as well as OA and ICA blood velocity measured by TCD in patients with ischemic optic neuropathy. Our findings, which showed a 10% increase in the signal intensity of the ipsilateral OA after SGB, corroborate these findings; however, we were unable to detect any changes in the ICA. The increased signal change in the MA, which supplies the facial nerve through its branch the petrosal artery, may support the role of SGB role in the alleviation of Bell palsy.<sup>37</sup> Studies at higher resolution may elucidate the response of these small arteries to sympathetic blockade.

Various procedural and technical difficulties were encountered as a result of the nature of this study. First, MRI measurements are extremely sensitive to the subject's position and movement. We chose this method nonetheless because of its high resolution and large field of view compared with TCD and its noninvasiveness and procedural ease com-

pared with single photon emission computed tomography and digital subtraction angiography. To overcome this obstacle, we used steps to accurately position the subject in the MRI scanner.

Second, effective nerve block should be elicited across all subjects for the uniformity and reproducibility of the experiment. However, the location of stellate ganglion varies with the individual and, even in the same subject, the left and right locations of the stellate ganglion may be different.<sup>38,39</sup> In the present study, all subjects showed obvious physiologic signs of Horner syndrome and conjunctival injection after SGB treatment, except one who also had no changes on MRA. In the present study, the success of the block of the head and neck could be determined by the appearance of Horner syndrome and conjunctival injection, whereas the skin temperature for the successful block of the chest and/or arm should be measured in addition.<sup>1</sup>

Third, serious medical risks are associated with SGB. It can rarely lead to potentially fatal complications such as pneumothorax, epidural block, subarachnoid block, convulsions, and retropharyngeal hematomas.<sup>5,40</sup> Although the stellate ganglion lies in the chest, below the seventh cervical transverse process, SGB is often performed by an anterior paratracheal approach at the level of the sixth cervical transverse process to avoid serious complications. Fluoroscopy can be used to identify the anterior tubercle at the sixth cervical transverse process in patients whose bony structure cannot be palpated. In this study, we used the blind approach without any serious complications, and all volunteers except for one successfully presented with Horner syndrome and conjunctival injection.

MRA benefits substantially from increasing the main MRI field strength to 3 T. The increased signal-to-noise ratio yields improved vessel contrast at high spatial resolution and the longer T1 relaxation times of background tissues further increase contrast by improving background suppression. Increased contrast and spatial resolution are highly desirable for more sensitive detection of small arteries.<sup>41</sup> Therefore, subtle intracranial change might have been not reflected well in the present study using 1.5-T MRI. In addition, the tissue perfusion by SGB would be easily detectable because of the higher sensitivity of 3-T MRI relative to the conventional 1.5-T MRI. These factors were not addressed in the current study; however, the further studies should be performed to intensively determine the contribution to small vessels as well as tissue perfusion as a result of SGB.

In addition, time-of-flight MRA has been used widely for the clinical diagnosis of the vascular disease in the noninvasive manner for several decades. However, the signal sources of time-of-flight MRA are complex, such as sensitivity to the flow velocity and direction, and imaging parameters, such as repetition time and/or slice thickness. Therefore, time-of-flight MRA signal intensity could not provide direct physiologic values, but only the relative change of the physiologic features such as blood flow and volume. Therefore, we have adopted the imaging of two different slabs: one for the local

and intracerebral vessels and the other for the whole cerebral vasculature, which is sensitive to the number of slices. This property has been one of major weaknesses in the time-of-flight MRA imaging, so some supplementary technologies have been introduced, such as phase contrast MRA or contrast-enhanced MRA, to quantify the vascular physiology. In this study, we used mainly time-of-flight MRA as the first step to investigate the SGB effect, providing the MRI as a methodological approach. It is certainly necessary for further investigation of the SGB effect with physiologic values.

In conclusion, we have demonstrated that MRA can be used to observe the overall changes in the cerebral blood vessels that are due to SGB treatment, especially in the flow and diameter of the extracranial arteries and the OA of the intracranial arteries. Our findings suggest a possible relationship between the therapeutic effects of SGB and vessel changes induced by SGB, a mechanism that has not been clearly elucidated. Further insight into the mechanism of SGB remains to be discovered, especially in disease states.

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## ANESTHESIOLOGY REFLECTIONS

### The 20-cent Virginia Apgar Stamp



Anesthesiologist Virginia Apgar (1909–1974) presented in 1952 what the “Butterfield epigram” popularized later as the “APGAR Score” for assessing newborns’ well-being (Appearance, Pulse, Grimace, Activity, and Respiration). The American Society of Anesthesiologists’ first female officer (Treasurer, 1941–1945) and Distinguished Service Award recipient (1961), Dr. Apgar also directed a division of the March of Dimes. The insatiably curious Apgar hand-fashioned and played her own violin, mezzo violin, and cello; she even “liberated” a payphone booth’s curly maple shelf to carve a viola. Dr. Apgar also loved to fish, golf, and watch baseball. Lecturing as rapidly as she drove cars and piloted airplanes, Dr. Apgar raced through life seeking low golf, high baseball, higher musical, and, of course, the highest neonatal scores. A stamp collector herself, Virginia Apgar, M.D., M.P.H., was honored posthumously on a U.S. postage stamp in 1994 (see above). (Copyright © the American Society of Anesthesiologists, Inc. This image appears in color in the *Anesthesiology Reflections* online collection available at [www.anesthesiology.org](http://www.anesthesiology.org).)

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