J Anesth (1995) 9:338–342

Journal of
Anesthesia

Effects of stellate ganglion block on cardiac coronary circulation

Iwao Sasaki, Tadanobu Kaneko, Naofumi Iwatsuki, and Yasuhiko Hashimoto

Department of Anesthesiology, Tohoku University School of Medicine, 1-1 Seiryomachi, Aoba-ku, Sendai, 980 Japan

Abstract: Since the stellate ganglion contains cardiac sympathetic nerves, stellate ganglion block (SGB) may influence cardiac and coronary hemodynamics. We investigated this influence of SGB by measuring the heart rate (HR), the left circumflex coronary artery blood flow (CBF), the maximum rate of increase of the left ventricular pressure (LV max dP/dt), the cardiac output (CO), the myocardial oxygen consumption (MVO_2), and the myocardial oxygen extraction ratio (MOER) in nine dogs before and after performing SGB by means of injection of 2 ml 1% mepivacaine. Left SGB resulted in a decrease of 10% in CBF and a decrease of 15% in LV max dP/dt, but HR, CO, and MVO_2 remained unchanged. On the other hand, right SGB resulted in a decrease of 30% in CBF and a decrease of 25% in LV max dP/dt, as well as a decrease of 20% in HR, 15% in CO, and 25% in MVO_2. SGB on either side resulted in an increase in MOER that was slight but nonetheless significant ($P < 0.05$) in that it suggested a relative deficit in CBF with respect to MVO_2. Inhalation of 100% oxygen decreased MOER to the pre-SGB level in either side, thus improving the myocardial oxygen supply–demand relationship. This study suggests the possibility that SGB has deteriorative effects on the myocardial oxygen supply–demand relationship. Those effects were counteracted by the inhalation of 100% oxygen.

Key words: Stellate ganglion block, Coronary blood flow, Myocardial oxygen consumption, Myocardial oxygen extraction ratio

Introduction

Stellate ganglion block (SGB) is a reversible procedure that blocks the cervical sympathetic trunk, the inferior cervical ganglion, and the superior thoracic sympathetic ganglion by means of the injection of a local anesthetic into the fascia of the longus colli. SGB is the most frequently used therapy in the pain clinic field and is regarded as therapeutically significant in the treatment of such ailments as herpes zoster (upper thoracic, neck and face), circulatory disorders of the upper extremites and face, and pain-related disorders in which the sympathetic nerves are involved [1]. Since the cardiac sympathetic nerves pass through the stellate ganglion [2], SGB is expected to have an ameliorative effect on impaired coronary circulation and cardiac function and thus to be well suited to the treatment of angina pectoris and myocardial infarction [3]. However, some clinical reports have shown that SGB does not improve coronary spasm [4]. Moreover, research information concerning any precise study of the effects of SGB on coronary hemodynamics and the myocardial oxygen supply–demand relationship has been scarce. In an attempt to clarify the influence exerted by SGB on the myocardial oxygen supply–demand relationship, we have examined the effects of left and right SGB on cardiac coronary circulation as well as on cardiac function in dogs.

Address correspondence to: I. Sasaki, Department of Anesthesiology, Jisei-kai Inomata Hospital, 3-97 Ohmachi, Haramachi, Fukushima, 975 Japan

Received for publication on December 8, 1993; accepted on May 8, 1995

Materials and methods

This study was conducted with the approval of the Animal Care Committee of the Tohoku University School of Medicine.

Implantation of instruments

Nine dogs, weighing 8–11.5 kg, were anesthetized with intravenous pentobarbital at a rate of 25 mg·kg^{-1}, and their tracheas were intubated. Anesthesia was then maintained with continuous infusion of pentobarbital at a rate of 2.0 mg·kg^{-1}·h^{-1} and pancuronium at 0.16 mg·kg^{-1}·h^{-1}. Respiration was controlled with an animal ventilator (Aika R-60, Aika, Chiba, Japan) to maintain $PaCO_2$ in the range 35–40 mmHg, and end-tidal CO_2 was monitored (Model 78356A, Hewlett Packard. Boeblingen, Germany). A foreleg vein was cannulated for the infusion of lactated Ringer's solution at a rate of 10 ml·kg^{-1}·h^{-1} throughout the experiment.

ECG and HR were continuously monitored with an oscilloscope. To monitor arterial blood pressure and to sample arterial blood, a catheter was placed in the right femoral artery. After thoracotomy on the left side, a catheter was placed, via the left external jugular vein, in the coronary sinus for the purpose of coronary sinus blood sampling. Electromagnetic flow probes (Nihon Kohden 1100, 2100 and 3100, Nihon Kohden, Tokyo, Japan) were placed on the ascending aorta, the left common carotid and the left circumflex coronary artery to measure flow at these locations. To measure the left ventricular pressure and its maximum rate of increase (LV max dP/dt) a catheter tip pressure transducer was inserted into the left ventricle through the femoral artery on the side not being used for the blood pressure measurement. Left and right SGB were performed by inserting two catheters, one into the left fascia and one into the right fascia of the longus colli just over the stellate ganglion.

Experimental protocol

After baseline hemodynamic and blood gas data had been obtained in relation to inhalation of the room air, left SGB was performed by injecting 2ml 1% mepivacaine through the catheter and once again hemodynamics were measured and blood gas samples were taken. Then while left SGB was still effective (about 15 min after SGB) the animals were ventilated with 100% oxygen and measurements and sampling were repeated. About 2h later, when hemodynamic data had returned to baseline and the effect of left SGB had disappeared, the same procedure was followed to investigate the effect of right SGB. The order of left and right SGB did not change their effects.

SGB was determined to have been effective when, in the case of left SGB, an increase in the left common carotid blood flow (L-CaBF) could be observed and when, in the case of right SGB, a decrease in HR could be observed [5,6]. Also, at the end of the experiment the correct positioning of the SGB catheter was confirmed by observing the extent to which a dye injected through the catheter actually colored the stellate ganglion area.

Myocardial oxygen consumption (MVO_2) and the myocardial oxygen extraction ratio (MOER) were calculated by means of the following equations: $MVO_2 = (CaO_2 - CsO_2) \times CBF/100$; $MOER = (CaO_2 - CsO_2) \times 100/CaO_2$. In these equations MVO_2 is expressed in ml·min^{-1}, CaO_2 is the oxygen content expressed in ml·dl^{-1} arterial blood, CsO_2 is the oxygen content expressed in ml·dl^{-1} coronary sinus blood, CBF is coronary blood flow (left circumflex coronary artery blood flow) expressed in ml·min^{-1}, and MOER is expressed as a percentage.

The experimental data were statistically analyzed using analysis of variance (ANOVA), and Student's paired t-test was used to test the difference between the other conditions of each group. Changes were considered to be significant when the probability (P) value was <0.05. All data are expressed as the mean ± standard deviation (SD).

Results

Effects of left SGB

Left SGB decreased cardiac output (CO) and systolic BP by 6 ± 19% and 7 ± 15%, respectively ($P < 0.05$). However, HR was unchanged. LV max dP/dt decreased by 15 ± 11% ($P < 0.01$), and CBF decreased by 10 ± 15% ($P < 0.05$). MVO_2 did not change significantly. MOER increased from 65 ± 12% to 67 ± 11% ($P < 0.05$) (Tables 1 and 2; Figs. 1–3).

Effects of right SGB

Right SGB decreased HR by 21 ± 13%, CO by 15 ± 32%, systolic BP by 6 ± 18%, LV max dP/dt by 25 ±

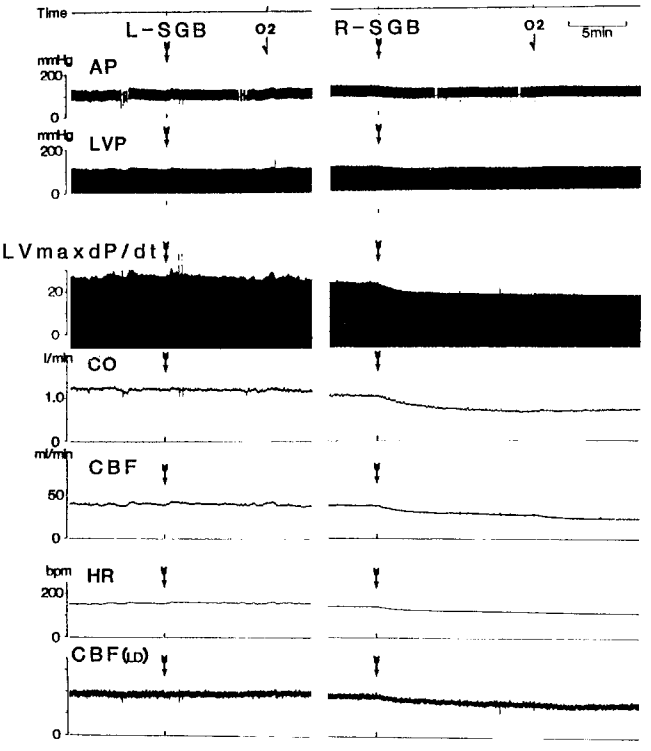

Fig. 1. Typical changes in systemic and coronary hemodynamics produced by stellate ganglion block (SGB) and inhalation of 100% oxygen (O_2). *L-SGB*, left stellate ganglion block; *R-SGB*, right stellate ganglion block; O_2, inhalation of 100% oxygen; *AP*, arterial pressure; *LVP*, left ventricular pressure; *CBF*, coronary blood flow; *CO*, cardiac output

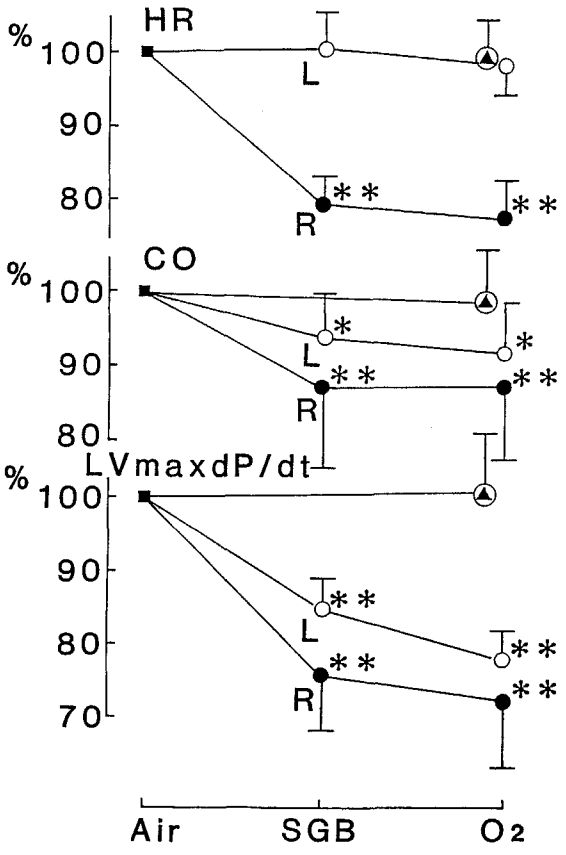

Fig. 2. Percent changes in HR, cardiac output (CO), and maximum rate of increase of left ventricular pressure (LV max dP/dt) after stellate ganglion block (SGB) and inhalation of 100% oxygen (O_2). *Closed square*, control and before SGB; *closed triangle inside open circle*, inhalation of 100% oxygen; *open circle*, left stellate ganglion block; *closed circle*, right stellate ganglion block; *air*, inhalation of air; O_2, inhalation of 100% oxygen; L, left stellate ganglion block; R, right stellate ganglion block. $^*P < 0.05$ *vs* air; $^{**}P < 0.01$ *vs* air

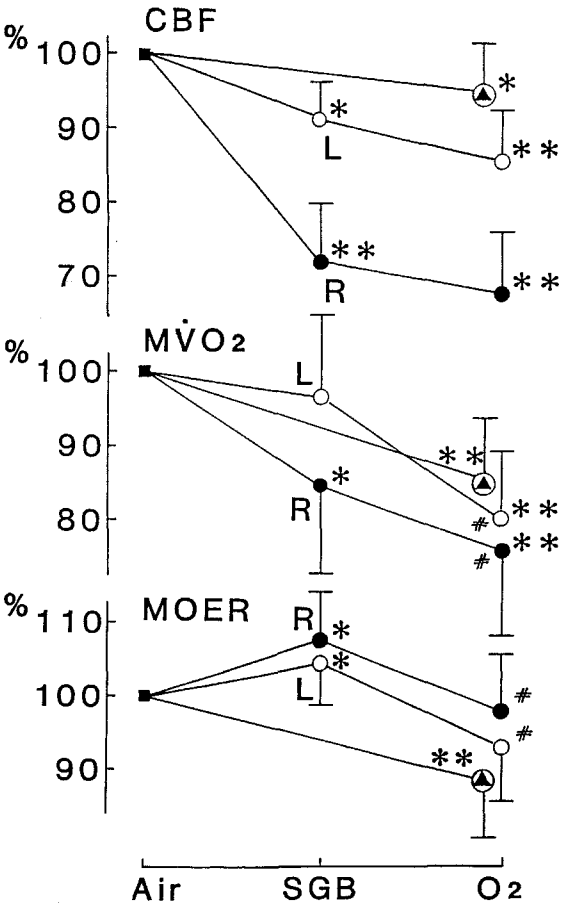

Fig. 3. Percent changes in coronary blood flow (CBF), myocardial oxygen consumption (MVO_2), and myocardial oxygen extraction ratio (MOER) after stellate ganglion block (SGB) and inhalation of 100% Oxygen (O_2). *Closed square*, control and before SGB; *closed triangle inside open circle*, inhalation of 100% oxygen; *open circle*, left stellate ganglion block; *closed circle*, right stellate ganglion block; *air*, inhalation of air; O_2, inhalation of 100% oxygen; L, left stellate ganglion block; R, right stellate ganglion block. $^*P < 0.05$ *vs* air; $^{**}P < 0.01$ *vs* air; $^{\#}P < 0.05$ *vs* SGB

Table 1. Effects of SGB on systemic and coronary hemodynamics

	Left SGB		Right SGB	
	Before	After	Before	After
HR (beats·min^{-1})	188 ± 28	189 ± 26	175 ± 28	139 ± 22**
SAP (mmHg)	172 ± 22	160 ± 26*	163 ± 26	153 ± 30**
DAP (mmHg)	104 ±	101 ± 22	101 ± 21	90 ± 19**
CO (l·min^{-1})	1.39 ± 0.28	1.30 ± 0.27	1.29 ± 0.37	1.12 ± 0.41**
L-CaBF (ml·min^{-1})	44 ± 27	70 ± 34**	58 ± 35	41 ± 32*
CBF (ml·min^{-1})	52 ± 12	47 ± 8*	49 ± 12	35 ± 11**
(ml·min^{-1}·kg^{-1})	5.2 ± 1.2	4.7 ± 0.8*	5.0 ± 1.3	3.6 ± 1.1**
LV max dP/dt (mmHg·s^{-1})	31 ± 5	26 ± 4**	25 ± 7	19 ± 6**
MVO_2 (ml·min^{-1})	537 ± 202	509 ± 164	499 ± 178	415 ± 180*
MOER (%)	65 ± 12	67 ± 11*	66 ± 13	70 ± 14*

All data are expressed as the mean ± SD; $n = 9$.
SGB, stellate ganglion block; SAP, systolic arterial pressure; DAP, diastolic arterial pressure; CO, cardiac output; L-CaBF, left carotid blood flow; CBF, coronary blood flow; LV max dP/dt, maximum rate of increase of left ventricular pressure; MVO_2, myocardial oxygen consumption; MOER, myocardial oxygen extraction ratio. $^*P < 0.05$; $^{**}P < 0.01$ *vs* before.

Table 2. Effects of inhalation of oxygen on CBF and MOER before and after SGB

			FiO_2 (mmHg)	
			0.21	1.0
CBF	Control		49 ± 10	46 ± 11*
($ml \cdot min^{-1}$)			5.0 ± 1.0	4.7 ± 1.1*
($ml \cdot min^{-1} \cdot kg^{-1}$)	Left SGB	Before	52 ± 12	
			5.2 ± 1.2	
		After	47 ± 8*	44 ± 11**
			4.7 ± 0.8*	4.5 ± 1.2**
	Right SGB	Before	49 ± 12	
			5.0 ± 1.3	
		After	35 ± 11**	33 ± 11**
			3.6 ± 1.1**	3.4 ± 1.2**
MOER (%)	Control		66.3 ± 10.3	58.3 ± 13.7**
	Left SGB	Before	64.7 ± 12.1	
		After	67.4 ± 11.1*	59.9 ± 13.6***
	Right SGB	Before	65.6 ± 13.1	
		After	69.9 ± 13.9*	63.7 ± 16.6***

All data are expressed as the mean ± SD; $n = 9$.
SGB, stellate ganglion block; CBF, coronary blood; MOER, myocardial oxygen extraction ratio.
*$P < 0.05$; **$P < 0.01$ *vs* control; ***$P < 0.05$ *vs* after SGB.

24%, and CBF by 30 ± 22% (all $P < 0.01$). MVO_2 decreased by 17 ± 37%, while MOER increased from 66 ± 13% to 70 ± 14% ($P < 0.05$) (Tables 1 and 2; Figs. 1–3).

Effects of inhalation of 100% oxygen

Following the inhalation of 100% O_2 (without performing SGB), CBF decreased by 6 ± 22% ($P < 0.05$), MVO_2 decreased by 15 ± 26% ($P < 0.01$), and MOER decreased by 12 ± 21% ($P < 0.01$). BP, CO and LV max dP/dt were unchanged. These decreases in CBF, MVO_2 and MOER were also observed when 100% O_2 was inhaled while the effects of SGB on either side were present. Therefore the increased MOER that was induced by SGB was suppressed by the inhalation of 100% O_2 (Table 2; Figs. 1–3).

Discussion

The most significant finding in our study is the fact that SGB on either side decreased CBF. Two explanations are under consideration about the main mechanisms contributing to this decrease in CBF. One is that SGB directly influences coronary vascular tone in a way that decreases CBF. The other is that coronary vascular tone is indirectly changed by SGB as a result of the fact that SGB decreases the heart's demand for oxygen by the decrease in HR, BP, and CO on right SGB, while there is a decrease in systolic BP and an increase in L-CaBF on left SGB. In the latter case, the decrease in CBF should correspond to the decrease in the operation of the heart (i.e., the heart's demand for oxygen), and this would indicate a balanced myocardial oxygen supply–demand relationship. However, in our study left SGB decreased CBF by 10% but did not change MVO_2, while right SGB decreased CBF by 30% but decreased MVO_2 by only 17%. Therefore, the decrease in CBF exceeded the decrease in MVO_2. As a result, MOER increased slightly but significantly in response to SGB on either side. This suggests that the myocardial oxygen supply–demand relationship deteriorates with SGB. This unbalanced state further suggests that the decrease in CBF mediated by SGB is at least partially independent of myocardial metabolic change. The fact that inhalation of oxygen decreased MOER, thereby improving the oxygen supply–demand relationship, was observed even in the presence of the effects of SGB on either side. Inhalation of oxygen may therefore be one of the ways to counteract the deleterious effect of SGB on the myocardial oxygen supply–demand relationship.

The possibility that the decrease in the activity of the heart may be contributing to the decrease in CBF deserves discussion. This hypothesis would similarly seem to require that the degree of decrease in CBF corresponds to the degree of decrease in the activity of the heart. However, in our study, the decrease in CBF was far greater than the decrease in the activity of the heart. Our results would thus seem to indicate that decreased activity of the heart cannot be viewed as the major contributor to the decrease in CBF. The depression of both HR and LV max dP/dt mediated by right SGB and the depression of LV max dP/dt and increase of L-CaBF

mediated by left SGB in this study are consistent with previous reports [5,6] which demonstrated the effect of SGB on both sides. These results therefore prove that our procedure of SGB is acceptable.

In contrast with previous reports involving human subjects [7,8], our results would seem to question the value of using SGB on either side on patients with ischemic heart diseases because SGB on either side not only failed to improve but actually deteriorated coronary hemodynamics. Of course, extrapolation of our results to humans with ischemic heart problems is a matter requiring greater scrutiny. Our experiment was performed on dogs with normal coronary circulation (i.e., no ischemic heart problems), and the ways in which the canine coronary circulation and the human coronary circulation respond to SGB may differ. Moreover, the influence of general anesthesia on the effect of SGB on coronary circulation cannot be neglected. The next stage in the overall study should be an evaluation of the effects of SGB on coronary hemodynamics in a model of the ischemic heart.

In summary, SGB on either side decreased coronary blood flow and increased the MOER in a canine model with normal coronary circulation under general anesthesia. Inhalation of oxygen improved the deteriorated myocardial oxygen supply–demand relationship that was mediated by SGB.

Acknowledgments. The authors would like to express their gratitude to Syoichi Obara for his expert technical assistance. This study was supported in part by a grant in aid for scientific research from the Ministry of Education, Science, and Culture, Japan, #02670674.

References

1. Bonica JJ (1990) Regional analgesia with local anesthetics. In: Bonica JJ (ed) The management of pain. Lea & Febiger, Philadelphia, PA, London, pp 1883–1966
2. White JC (1974) Sympathectomy for relief of pain. Adv Neurol 4:629–638
3. Klassen GA, Bramwell RS, Bromage PR (1980) Effect of acute sympathectomy by epidural anesthesia on the canine coronary circulation. Anesthesiology 52:8–15
4. Fukuuchi A, Fujita M, Suzuki H, Kawamata M, Mukubo Y, Yokoyama M (1990) Effects of stellate ganglion block on prevention of variant angina (in Japanese). Pein Kurinikku (Pain Clin) 11:214–218
5. Randall WC, Rohse WG (1956) The augmenter action of the sympathetic cardiac nerves. Circ Res 4:470–475
6. Inoue K (1984) Antiarrhythmic effects of stellate ganglion block (in Japanese). Masui (Jpn J Anesthesiol) 33:1187–1195
7. Tarazi RC, Estafanous FG, Fouad FM (1978) Unilateral stellate block in the treatment of hypertension after coronary bypass surgery. Am J Cardiol 42:1013–1018
8. Bidwai AV, Rogers CR, Pearce M (1979) Preoperative stellate ganglion blockade to prevent hypertension following coronary artery operations. Anesthesiology 51:345–347