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Stellate Ganglion Block Is Associated with Increased Tibial Nerve Muscle Sympathetic Activity in Humans

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Background: **Left stellate ganglion block has been shown to increase heart rate and blood pressure, possible because of blockade of afferent vagal fibers from arterial baroreceptors in the aortic arch. Because efferent muscle sympathetic nerve activity (MSNA) is influenced by the arterial baroreflex, the hypothesis that left stellate ganglion block increases efferent MSNA recorded from the tibial nerve of humans was tested.**

Methods: **Twenty healthy male volunteers were sequentially assigned to one of three groups: stellate ganglion block (n = 10), in which 7 ml 1% mepivacaine was injected into the left stellate ganglion; placebo (n = 5), in which 7 ml of saline was injected into the left stellate ganglion; and intramuscular injection (n = 5), in which 7 ml mepivacaine was injected into the left deltoid muscle. Direct intraneural microneurographic recording with a tungsten microelectrode was used to record MSNA in the left tibial nerve. MSNA, heart rate, and blood pressure were recorded before and after injection in all groups. An additional five volunteers were studied with transthoracic echocardiography to examine the effect of stellate ganglion block on preload changes.**

Results: **Tibial nerve MSNA increased after mepivacaine injection to the left stellate ganglion but was unchanged after saline injection to the left stellate ganglion or mepivacaine injection into the deltoid muscle. Heart rate increased significantly after the left stellate ganglion block but did not change significantly after saline injection to the left stellate ganglion or after mepivacaine injection to the deltoid muscle. Systemic blood pressure did not change significantly in all groups. Left ventricular end-diastolic area and left ventricular end-diastolic circumference did not change after stellate ganglion block.**

Conclusions: **Tibial nerve MSNA increased during left stellate ganglion block with mepivacaine. (Key words: Anesthetic techniques, regional: stellate ganglion block. Anesthetics, local: mepivacaine. Techniques: sympathetic microneurography.)**

STELLATE ganglion block with local anesthetics is used for the treatment of a variety of sympathetically mediated diseases, including postherpetic neuralgia[1] and reflex sympathetic dystrophy.[2] The efficacy of stellate ganglion block is evaluated by observing changes in sympathetic effectors, such as Horner's syndrome, skin temperature, skin blood velocity, and skin resistance response.[3] The technique of microneurography has enabled researchers to observe efferent muscle sympathetic nerve activity (MSNA) in humans by means of direct intraneural recording from postganglionic sympathetic efferent fibers.[4] MSNA consists of sympathetic vasoconstrictor nerve impulses leading to skeletal muscle blood vessels and plays an important role in regulating regional blood flows and systemic blood pressure.[5] It is likely that sympathetic nerve activity in the cervicothoracic region and upper extremity would be suppressed because of blockade of sympathetic impulses during stellate ganglion block. However, the effect of stellate ganglion block on sympathetic nerve activity in other regions is uncertain. Left stellate ganglion block has been shown to increase heart rate and blood pressure, which might be because of blockade of vagal fibers from arterial baroreceptors in the aortic arch.[6] Consequently, efferent MSNA to other regions,

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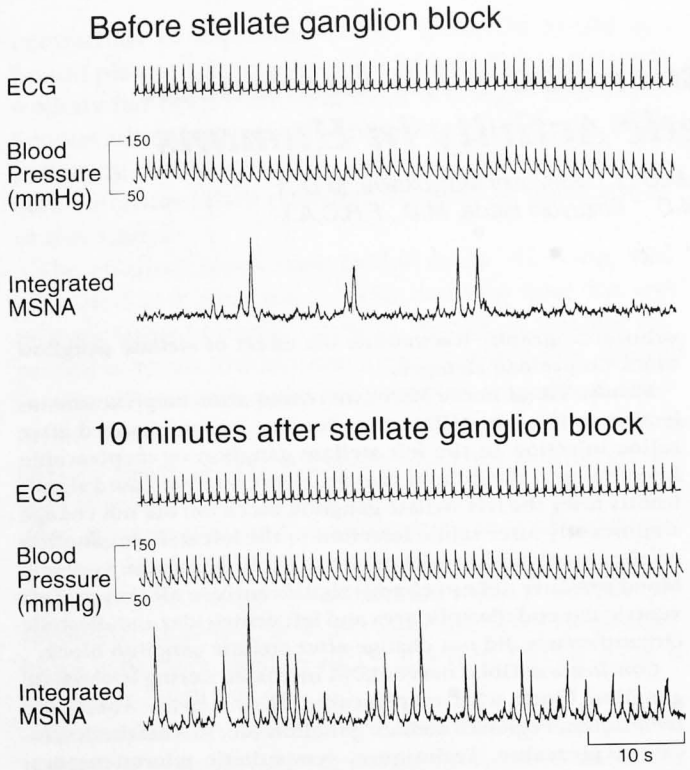

Fig. 1. Representative muscle sympathetic nerve activity (MSNA) tracings from one subject before and 10 min after mepivacaine injection into the left stellate ganglion. After mepivacaine injection, there was a noteworthy increase in MSNA, in both burst frequency and burst amplitude.

which is closely regulated by the arterial baroreflex, may be influenced by left stellate ganglion block. In addition, blockade of glossopharyngeal and vagal fibers from baroreceptors in the neck in humans has been reported to result in increases in MSNA and blood pressure.[7] Accordingly, we tested the hypothesis that left stellate ganglion block increases MSNA in the tibial nerve in humans.

Methods and Materials

Subjects

This study was approved by the Human Research Committee of the Research Institute of Environmental Medicine, Nagoya University, and written informed consent was obtained from all subjects. The study group was comprised of 20 healthy male volunteers ranging in age from 26 to 38 yr. The subjects were sequentially allocated to one of three groups: stellate ganglion block (n = 10), in which 7 ml 1% mepivacaine was injected into the left stellate ganglion; placebo (n = 5), in which 7 ml of saline was injected into the left stellate ganglion; and intramuscular injection (n = 5), in which 7 ml mepivacaine was injected into the left deltoid muscle. All subjects were free of cardiovascular, pulmonary, or neurologic disease, and no subjects were receiving medications before the study.

Recordings of MSNA

Subjects were studied while supine. Multiunit postganglionic efferent discharges of MSNA were recorded from the left tibial nerve. An epoxy-resin-coated tungsten microelectrode (model 26-05-1, FHC, Brunswick, ME) with a shaft diameter of 100 μm, a tip diameter of 1 μm, and an impedance of 3–5 MΩ was inserted into the muscular branch of the tibial nerve at the left popliteal fossa. A reference surface electrode was attached 1–2 cm from the recording electrode. After sympathetic nerve signals were fed into a high-input-impedance preamplifier (DAM-6A, W-P Instruments, Hamden, CT; gain setting 60 dB), they were passed through a bandpass filter (E-3201 A, NF Circuit Design

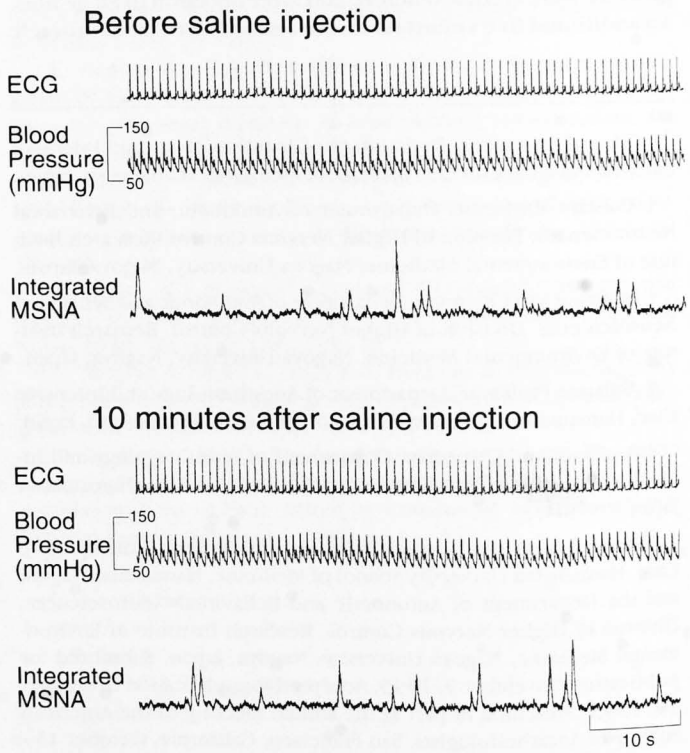

Fig. 2. Representative muscle sympathetic nerve activity (MSNA) tracings from one subject before and 10 min after saline injection into the left stellate ganglion. There was no difference in MSNA between before and after saline injection.

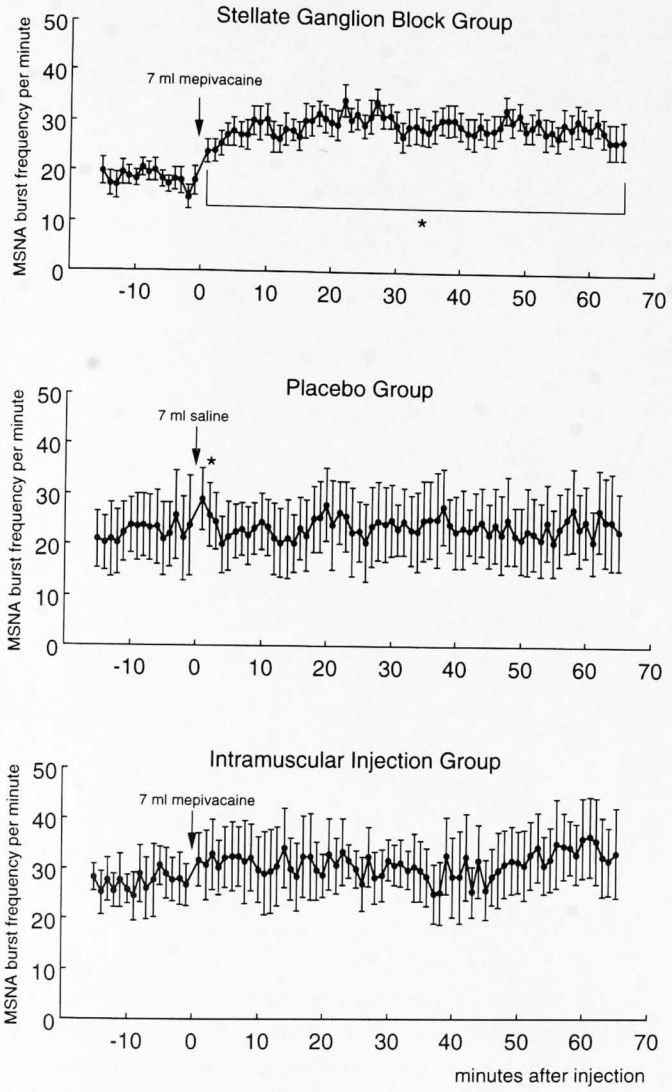

Fig. 3. Average data for muscle sympathetic nerve activity (MSNA) burst frequency in three groups. Compared with baseline MSNA values (18.4 ± 1.8 burst/min), significant increases in MSNA (peak 34.4 ± 3.3 bursts/min) were observed in ten subjects during left stellate ganglion block with mepivacaine. There was no significant increase in MSNA burst frequency except for a transient increase in MSNA burst frequency 2 min after saline injection in five subjects after saline injection into the left stellate ganglion. There was no significant increase in MSNA burst frequency in five subjects after mepivacaine injection into the left deltoid muscle. Data are mean ± SEM. *$P < 0.05$ compared with baseline value.

Block, Yokohama, Japan; bandwidth 500–5,000 Hz) and monitored on an oscilloscope (5113, Tektronix, Beaverton, OR; gain setting 46 dB). Nerve signals were then full-wave rectified, integrated at a time constant of 0.1 s, and recorded on a thermal pen recorder (Recti-Horiz-8K, NEC-San-ei, Tokyo, Japan) and a thermal array recorder (WS-682G, Nihon Kohden, Tokyo, Japan) as a mean-voltage neurogram. The following criteria were used to identify MSNA: (1) efferent activity from the muscular branch, (2) spontaneous and pulse-synchronous rhythmic burst discharge,[8] (3) marked accentuation by Valsalva's maneuver,[9] and (4) limited sensitivity to arousal stimuli. To quantify MSNA, the number of pulse-synchronous bursts per minute (burst frequency) and the number of bursts per 100 cardiac cycles (burst incidence) were measured.

Other Recordings

Arterial blood pressure and the surface electrocardiogram were recorded before and after injection in all groups. Skin temperature at the left hypothenar eminence and relative skin blood velocity in the left index finger were measured in a stellate ganglion block group and in a placebo group. Arterial blood pressure was measured at 1-min intervals with a sphygmomanometer (BP-203P, Nippon Colin, Komaki, Japan) and was continuously monitored by mass compensatory photoplethysmography on the right hand (Finapres 2300, Ohmeda, Englewood, CO). Electrocardiogram was monitored from lead II with an electrocardiograph (MXE-6100, Nihon Kohden, Tokyo, Japan). Relative skin blood velocity was measured in the left index finger with a laser-Doppler flowmeter (ALF2100, Advance, Tokyo, Japan) with an averaging time of 0.1 s. Skin temperature was measured at the left hypothenar eminence with a thermistor (E334, Technol Seven, Yokohama, Japan). The output of MSNA, blood pressure, electrocardiogram, skin temperature, and relative skin blood velocity data were recorded using an FM magnetic tape recorder (KS616U, Sony-Magnescale, Tokyo, Japan).

Experimental Protocol

After the subject had rested in the supine position for 30 min and recordings of tibial nerve MSNA were obtained, baseline nerve activity and hemodynamic data were recorded for 15 min. Blood samples were obtained *via* a venous cannula to determine basal plasma concentrations of norepinephrine in a stellate ganglion block group with eight subjects and in a placebo injection group with four subjects. In a stellate ganglion block group and in a placebo group, left stellate ganglion block with 7 ml 1% mepivacaine or injection of 7 ml of saline into the left stellate ganglion using the anterior, paratracheal, C6 level approach was carried out by the same anesthesiologist. In an intramuscular

Table 1. Hemodynamic Data in the Stellate Ganglion Block, Placebo and Intramuscular Injection Groups

	Before Injection	5 min after Injection	10 min after Injection	20 min after Injection	30 min after Injection	40 min after Injection	50 min after Injection	60 min after Injection
Stellate ganglion block (n = 10)								
Heart rate (beats/min)	67 ± 4	67 ± 4	68 ± 4	69 ± 4	69 ± 4	69 ± 4	70 ± 5	71 ± 4*
Systolic blood pressure (mmHg)	128 ± 4	126 ± 6	131 ± 6	133 ± 6	131 ± 6	128 ± 6	129 ± 6	128 ± 6
Diastolic blood pressure (mmHg)	66 ± 3	65 ± 4	66 ± 4	68 ± 4	69 ± 4	68 ± 4	68 ± 4	68 ± 5
Mean blood pressure (mmHg)	86 ± 4	86 ± 4	88 ± 5	89 ± 5	89 ± 4	88 ± 5	88 ± 5	88 ± 5
Placebo (n = 5)								
Heart rate (beats/min)	63 ± 5	62 ± 6	63 ± 6	63 ± 5	63 ± 7	64 ± 5	63 ± 6	63 ± 7
Systolic blood pressure (mmHg)	129 ± 5	128 ± 7	130 ± 6	134 ± 7	126 ± 5	130 ± 7	129 ± 6	127 ± 6
Diastolic blood pressure (mmHg)	63 ± 3	63 ± 5	61 ± 3	64 ± 5	59 ± 3	63 ± 4	58 ± 7	64 ± 7
Mean blood pressure (mmHg)	85 ± 4	85 ± 5	84 ± 3	87 ± 5	81 ± 3	86 ± 5	82 ± 6	85 ± 6
Intramuscular injection (n = 5)								
Heart rate (beats/min)	66 ± 2	64 ± 3	66 ± 2	66 ± 2	64 ± 2	65 ± 3	64 ± 3	68 ± 3
Systolic blood pressure (mmHg)	130 ± 12	129 ± 14	130 ± 10	128 ± 12	126 ± 13	128 ± 13	132 ± 15	132 ± 13
Diastolic blood pressure (mmHg)	67 ± 6	66 ± 7	62 ± 7	64 ± 6	63 ± 6	63 ± 7	67 ± 7	66 ± 6
Mean blood pressure (mmHg)	88 ± 8	87 ± 9	85 ± 8	86 ± 8	84 ± 8	85 ± 9	89 ± 10	88 ± 8

Data are mean ± SEM.

* $P < 0.05$ *versus* baseline value before injection.

injection group, 7 ml 1% mepivacaine was injected into the left deltoid muscle. The success of stellate ganglion block was confirmed by Horner's syndrome. After injection, hemodynamic and neural variables were recorded for 65 min. Venous blood samples were drawn 5, 10, 20, 30, 40, 50, and 60 min after injection for the measurement of plasma concentrations of norepinephrine in a stellate ganglion block group with eight subjects and in a placebo injection group with four subjects, and mepivacaine in a stellate ganglion block group with eight subjects and in an intramuscular injection group with four subjects.

Analysis of Plasma Concentrations of Norepinephrine Mepivacaine

Blood samples from a left antecubital vein were collected through an intravenous catheter filled with heparin solution. Samples were centrifuged (4,000*g*) at 4°C for 10 min, and the plasma was separated. The plasma was maintained at −80°C until analysis. Plasma concentrations of norepinephrine were measured by high-performance liquid chromatography. The minimum detectable norepinephrine concentration was 8.5 pg/ml. Coefficient of variation for norepinephrine was approximately 1.0%. Plasma concentrations of mepivacaine were measured by gas chromatography. The minimum detectable mepivacaine concentration was 0.03 μg/ml. The coefficient of variation for mepivacaine was approximately 9.2%.

Echocardiographic Study

Transthoracic echocardiography was performed in a different group of subjects. This additional study was conducted to demonstrate the effect of stellate ganglion block on preload changes. This group consisted of five healthy male volunteers aged 24–38 yr. Subjects with clinically significant systemic disease or who chronically received medications were excluded. A Hewlett-Packard ultrasound imaging system (77020AC) with a 3.5 MHz transducer was used for transthoracic echo-

Table 2. Plasma Concentrations of Norepinephrine in the Stellate Ganglion Block and Placebo Groups, and Mepivacaine in the Stellate Ganglion Block and Intramuscular Injection Groups

	Before Injection	5 min after Injection	10 min after Injection	20 min after Injection	30 min after Injection	40 min after Injection	50 min after Injection	60 min after Injection
Norepinephrine (pg/ml)								
Stellate ganglion block (n = 8)	138.6 ± 14.7	156.9 ± 23.3	152.5 ± 19.6	161.0 ± 13.9	160.3 ± 14.3	166.6 ± 19.9	157.6 ± 19.7	156.0 ± 19.0
Placebo (n = 4)	206.3 ± 31.1	217.5 ± 38.6	195.5 ± 27.3	202.8 ± 33.0	180.8 ± 25.9	166.8 ± 20.4	144.3 ± 33.2	152.8 ± 26.8
Mepivacaine (μg/ml)								
Stellate ganglion block (n = 8)	—	0.77 ± 0.23	1.12 ± 0.18	1.11 ± 0.15	0.98 ± 0.13	0.93 ± 0.13	0.82 ± 0.09	0.70 ± 0.07
Intramuscular injection (n = 4)	—	0.61 ± 0.10	0.79 ± 0.17	0.98 ± 0.20	0.99 ± 0.11	0.84 ± 0.09	0.82 ± 0.16	0.67 ± 0.11

Data are mean ± SEM.

cardiography. All subjects were recumbent. The transducer was positioned in the fourth or fifth intercostal space along the left sternal border and was directed posteriorly to obtain left ventricular short axis view at the midpapillary muscle level. Two-dimensional echocardiograms were recorded on VHS videotapes. Echocardiographic analysis was performed as follows. We traced the left ventricular short-axis endocardium at end-diastole to obtain end-diastolic area and end-diastolic circumference. Leading-leading methods were used to trace the endocardium.[10] Ventricular end-diastole was identified by the peak of the R wave. The mean of three consecutive beats at end-expiration was used for analysis. Each echocardiogram was analyzed by a 77020AC ultrasonograph system. Left stellate ganglion block with 7 ml 1% mepivacaine was carried out by the same anesthesiologist using the anterior, paratra-

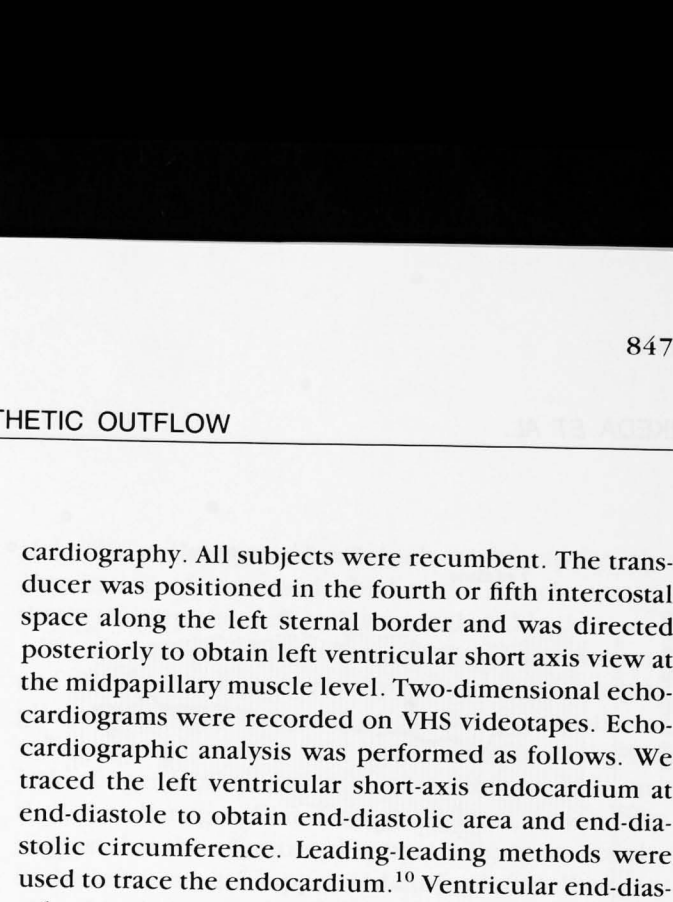

Fig. 4. Average data for skin temperature at the left hypothenar eminence and relative skin blood velocity in the left index finger in ten subjects during left stellate ganglion block with mepivacaine. Both skin temperature and relative skin blood velocity showed significant increases after injection. Data are mean ± SEM. *$P < 0.05$ compared with baseline value.

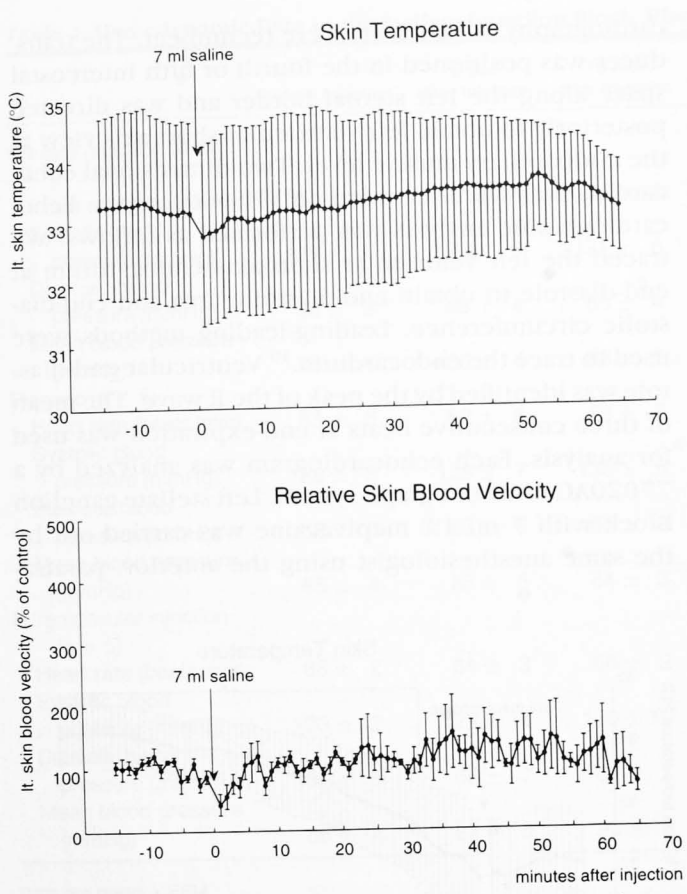

Fig. 5. Average data for skin temperature at the left hypothenar eminence and relative skin blood velocity in the left index finger in five subjects after saline injection into the left stellate ganglion. There were no significant changes in skin temperature or relative skin blood velocity. Data are mean ± SEM.

cheal, C6 level approach. Echocardiographic data were obtained before and 5, 10, 20, and 30 min after injection.

Data Analysis

Data are expressed as mean ± SEM. Each variable, except for those related to plasma concentrations and transthoracic echocardiography, represents an average for 1 min. One-minute averages were compared with the control period, which was an average for 15 min. Time-dependent changes were evaluated by analysis of variance. Plasma concentrations of mepivacaine between two groups were compared using unpaired *t* tests. Differences were considered statistically significant when the *P* value was less than 0.05.

Results

Muscle Sympathetic Nerve Activity

Figure 1 shows representative MSNA tracings obtained from one subject before and 10 min after left stellate ganglion block with mepivacaine. After mepivacaine injection, there was a noteworthy increase in MSNA in terms of the burst frequency and the burst amplitude. Figure 2 shows representative MSNA tracings obtained from one subject before and 10 min after saline injection into the left stellate ganglion. There was no difference in MSNA between before and after saline injection. Figure 3 shows average data for MSNA burst frequency in three groups. Although we looked at the MSNA data both the burst frequency and the burst incidence ways, we found no differences. Thus, we show the MSNA data as burst frequency. Compared with baseline MSNA values (18.4 ± 1.8 bursts/min), marked increases in MSNA (peak 34.4 ± 3.3 bursts/min) were observed during left stellate ganglion block with mepivacaine. MSNA burst frequency increased significantly after mepivacaine injection into the left stellate ganglion. MSNA reached a peak almost 20 min after injection and remained activated until 65 min after injection. There was no significant change in MSNA burst frequency except for a transient increase in MSNA burst frequency 2 min after saline injection in 5 subjects after saline injection into the left stellate ganglion. There was no significant change in MSNA burst frequency in five subjects after mepivacaine injection into the left deltoid muscle.

Table 3. Transthoracic Echocardiographic Data

	Before Injection	5 min after Injection	10 min after Injection	20 min after Injection	30 min after Injection
LVEDA (cm^2)	19.8 ± 0.8	19.5 ± 1.2	19.9 ± 0.7	19.6 ± 1.0	19.3 ± 1.2
LVEDC (cm)	16.7 ± 0.6	16.2 ± 0.4	17.3 ± 0.5	16.9 ± 0.5	16.6 ± 0.6

Data are mean ± SEM; n = 5.

LVEDA = left ventricular end-diastolic area; LVEDC = left ventricular end-diastolic circumference.

Heart Rate and Blood Pressure

Table 1 shows hemodynamic data in three groups. Heart rate increased significantly after left stellate ganglion block but did not change significantly after saline injection to the left stellate ganglion or after mepivacaine injection to the deltoid muscle. Systemic blood pressure did not change significantly in all groups.

Plasma Concentrations of Norepinephrine and Mepivacaine

Table 2 shows the average data for plasma concentrations of norepinephrine and mepivacaine. Although there were no significant changes in plasma concentrations of norepinephrine after mepivacaine injection to the left stellate ganglion or saline injection to the left stellate ganglion, there was a trend for a norepinephrine increase after mepivacaine injection to the left stellate ganglion and a trend for a norepinephrine decrease after saline injection to the left stellate ganglion. There were no differences in plasma concentrations of mepivacaine between a stellate ganglion block group and an intramuscular injection group.

Skin Temperature and Skin Blood Velocity

Figure 4 shows the average data for skin temperature at the left hypothenar eminence and relative skin blood velocity in the left index finger during left stellate ganglion block with mepivacaine. Both skin temperature and skin blood velocity showed significant increases after mepivacaine injection. There were no significant changes in skin temperature or relative skin blood velocity after saline injection into the left stellate ganglion (fig. 5).

Echocardiographic Data

Echocardiographic data are summarized in table 3. Left ventricular end-diastolic area and left ventricular end-diastolic circumference did not change during left stellate ganglion block.

Discussion

In the current study, using direct intraneural microneurographic recording, we demonstrated that MSNA in the tibial nerve is markedly enhanced during left stellate ganglion block with mepivacaine. Although there were no significant changes in plasma concentrations of norepinephrine after left stellate ganglion block, there was a trend for a norepinephrine increase after left stellate ganglion, which might support the observation of increased tibial nerve muscle sympathetic activity after left stellate ganglion block.

We also observed the changes in skin temperature and relative skin blood velocity during left stellate ganglion block. Skin temperature at the left hypothenar eminence and relative skin blood velocity in the left index finger increased after mepivacaine injection into the left stellate ganglion. Thus, at the least, these results suggest that the effectors of sympathetic nerve activity in the left arm were suppressed during left stellate ganglion block to some extent.

The precise mechanism for the increase in tibial MSNA during left stellate ganglion block is uncertain. However, we propose a possible mechanism: the blockade of vagal impulses from arterial baroreceptors in the aortic arch by mepivacaine. Arterial baroreceptors at the carotid sinus send afferent impulses to the nucleus tractus solitarius *via* the glossopharyngeal nerve, and arterial baroreceptors in the aortic arch and cardiopulmonary baroreceptors send afferent impulses to the nucleus tractus solitarius *via* the vagus nerve. The nucleus tractus solitarius transmits inhibitory signals to the rostral ventrolateral medulla. Fagius *et al.* demonstrated that unilateral lidocaine blockade of glossopharyngeal and vagal fibers from baroreceptors in the neck in humans resulted in increases in MSNA and blood pressure and that bilateral blockade resulted in marked increases in MSNA and blood pressure.[7] They also observed that pulse synchrony disappeared after bilateral blockade of the glossopharyngeal and vagus nerves, whereas pulse synchrony was retained after unilateral blockade. It has been demonstrated that solution injected into the stellate ganglion spreads to surrounding areas.[11] Because vagal fibers from arterial baroreceptors in the aortic arch run immediately adjacent to the stellate ganglion, it is possible that these vagal fibers from arterial baroreceptors in the aortic arch were somewhat suppressed by mepivacaine.

MSNA is governed by inhibitory influences from arterial baroreceptors.[12,13] It has been noted that a decrease in blood pressure results in the activation of MSNA and that an increase in blood pressure results in the attenuation of MSNA. In our study, diastolic blood pressure did not decrease during stellate ganglion block. Thus, unloading of arterial baroreceptors was not responsible for the increase in MSNA during stellate ganglion block. MSNA is governed by inhibitory influences from cardiopulmonary baroreceptors.[14] In our study, transthoracic echocardiography showed there

were no changes in left ventricular end-diastolic area and left ventricular end-diastolic circumference during stellate ganglion block. Therefore, cardiopulmonary reflexes might not be involved with the increase in MSNA during stellate ganglion block.

There were no differences in plasma concentrations of mepivacaine between those receiving intramuscular mepivacaine and those undergoing stellate block with mepivacaine while MSNA increased only in the stellate ganglion block group. Thus, the possibility that plasma concentrations of mepivacaine act within the central nervous system to augment sympathetic outflow is unlikely.

In conclusion, sympathetic neural outflow to skeletal muscle in the tibial nerve was markedly activated during left stellate ganglion block with mepivacaine. This activation might be due to the blockade of vagal fibers from arterial baroreceptors in the aortic arch by mepivacaine.

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