Serotonin in the Rat Sympathetic Ganglion: Microdetermination of Monoamines and Their Metabolites by High-Performance Liquid Chromatographic Electrochemical Detection

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Abstract—We attempted to determine the existence of a functional serotonergic small intensely fluorescent (SIF) cell in the rat superior cervical sympathetic ganglion (SCG) by evaluating the effects of pargyline, decentralization and electrical stimulation on the preganglionic sympathetic fibers. The objective was to assess changes in serotonin (5-HT) metabolism compared with findings in the case of dopamine (DA) metabolism. The contents (ng/ganglion, n=8, average±S.E.) in the adult male Wistar rat SCG of 5-HT and 5-hydroxyindoleacetic acid (5-HIAA) were 1.19±0.11 and 0.11±0.01, respectively, determined by our reverse-phase high-performance liquid chromatographic electrochemical method. A relatively large amount of 5-HT was detected in the capsule of the SCG (0.62±0.16 ng/capsule, n=8) without detection of 5-HIAA. The pargyline injection increased the DA content and decreased the 3,4-dihydroxyphenylacetic acid (DOPAC) content rapidly. The increase of the 5-HT content was observed without changes in the capsule 5-HT. The mode of pargyline induced-increase in the 5-HT content and decrease of the 5-HIAA content was slow compared to that of DA and DOPAC. Decentralization did not alter the 5-HIAA content, whereas the DOPAC content was markedly reduced. Although electrical stimulation significantly increased the DOPAC content, changes in the 5-HIAA content were not observed. It would thus appear that 5-HT is produced in the rat SCG, and if so, then support for existence of a serotonergic SIF cell in the rat sympathetic ganglion can be obtained. A direct connection between pre- and postganglionic sympathetic neurons through the serotonergic SIF cell was not demonstrated.

Mammalian sympathetic ganglia seem to possess various types of small intensely fluorescent (SIF) cells, among which the dopaminergic SIF cell is best understood (1, 2). The SIF cells in the sympathetic ganglia are composed of not only dopaminergic cells, but also other types of cells (3). The possible presence of a serotonergic SIF cell system in addition to the catecholaminergic cells in the rat SCG was suggested by the recent morphological studies of Verhofstad et al. (4). Taken in conjunction with earlier data that exogenous serotonin (5-HT) and its analogues stimulate ganglion cells and modify neuronal transmission (5–7), the question arises as to whether the serotonergic SIF cell is functional in trans-synaptic neuronal transmission in the SCG. We compared the biochemical characteristics of 5-HT metabolism in the rat SCG with that of dopamine (DA) metabolism. The present study was designed 1) to confirm the existence of a 5-HT production system in the rat SCG and 2) to determine whether the activity of the ganglionic 5-HT cell depends on preganglionic sympathetic neurons. In addition, our newly developed and sensitive quantitative analytical method of
catecholamines (CAs), 5-HT and their acidic metabolites, 3,4-dihydroxyphenylacetic acid (DOPAC), homovanillic acid (HVA) and 5-hydroxyindoleacetic acid (5-HIAA), based on high-performance liquid chromatography (LC) with electrochemical detection (EC) is described.

Materials and Methods

Animals, drug treatments and surgical procedures: Male Wistar rats weighing 250–300 g were fed a standard diet (F-2, Funabashi Farm Co., Chiba, Japan) and water ad libitum and housed at 24°C. These rats were given a 10% sucrose solution orally 24 hr before decapitation because the cereal chow contained tyrosine and DOPA which modify the level of the endogenous metabolites of biogenic amines (8).

To confirm the existence of the 5-HT production system in the SCG, the experimental group and the control group were given pargyline, a monoamine oxidase inhibitor (75 mg/kg, i.p.) or the vehicle (0.9% NaCl) alone, respectively. Three groups of five experimental animals were decapitated at 5, 15 and 30 min after pargyline had been given i.p. Increases in the DA and 5-HT contents were measured within a few hr after the decapitation.

To determine whether or not the activity of the ganglional 5-HT cell depends on the input from preganglionic neurons, two experiments were done. In the first, the SCG was decentralized by aseptic removal of several millimeters of the right sympathetic chain caudal to the ganglion. The decentralized and sham operated rats were decapitated 7 days after this surgery and ganglia were rapidly removed and placed on a cooled glass plate on ice. Ptosis in the right eye was taken as evidence of decentralization. In the second experiment, electrical monophasic stimulation (10 V, 10 Hz, 1 msec duration) to the right preganglionic nerves was performed after the bilateral SCG and preganglionic nerve were exposed, under conditions of urethane (1.2 g/kg, s.c.) anaesthetization. The stimulation was provided by a bipolar platinum electrode, a stimulator (3F–46, San-ei, Tokyo, Japan) and an isolator (5361, San-ei) for 3 and 10 min, respectively. At the end of this stimulation, both ganglia were dissected out and placed on a cooled glass plate on ice.

Sample preparations: After the connective and fatty tissues had been removed and the residual pre- and postganglionic fibers were cut off the SCG, the capsule was carefully desheathed on an ice-cooled glass plate under a stereomicroscope (×20). The isolated SCG and capsule were extensively washed in an ice-water bath and frozen on dry ice. The decapsulation is critical because the capsule of the SCG contained some amounts of 5-HT (Data are given in Table 1).

Determination of amines and metabolites: Within a few hr after sample preparations, amines and metabolites were assayed. The frozen tissues were homogenized in 100 µl of 10% ZnSO₄-1.0% EDTA-2Na solution containing N-methyl-5-HT (10 ng/ml), 3,4-dihydroxyphenylpropionic acid (DOPPA) (20 ng/ml) and 3,4-dihydroxybenzylamine (DHB) (50 ng/ml). N-methyl-5-HT, DOPPA and DHB were used as the internal standards for indoles (5-HT, 5-HIAA), acidic metabolites of CAs (DOPAC, HVA) and CAs, respectively. Homogenization was performed in an ice-water bath for 30 sec using an ultrasonic probe homogenizer (SONIFIER Model 185, Branson, U.S.A.) at a setting of 5 (20 KHz, 4 W, 30 sec). Thirty five µl of 0.5 N NaOH was added to 50 µl of homogenate, and 10 µl of 3% HClO₄ was added to the residual homogenate. After vortexing and centrifugation (15,000 rpm, 4°C, 15 min), 70 µl of the supernatant from the ZnSO₄-NaOH homogenates was injected into the LCEC system for the quantitation of 5-HT and 5-HIAA. Fifty µl of ZnSO₄-HClO₄ homogenates was injected into the LCEC system for the quantitation of DOPAC and HVA.

Isolation of CAs from the residual supernatant was achieved by absorption on alumina (Wolem Neutral activity Grade I) activated by the method of Anton and Sayre (9). The supernatant was adjusted to pH 8.6 by the addition of 0.5 ml of 3 M Tris-HCl buffer and then transferred to a 0.5 ml conical reaction vial containing 20 mg of activated alumina. The CAs were absorbed into the alumina by vortexing for 5 min,
After washing with distilled water, the CAs were eluted by 100 µl of 0.1 N HClO₄. Eighty µl of the HClO₄ eluent was injected into the LCEC system for quantitation of CAs.

The LCEC system was composed of a 6000A pump, U6K universe injector, µ-Bondapak C₁₈ reverse phase column (30×0.39 cm) (Waters Assoc., U.S.A.) and an electrochemical detector (LC-4A, Bioanalytical System, U.S.A.) equipped with a glassy carbon electrode (TL-5). The working electrode potential was maintained at +0.55 V versus the Ag/AgCl reference electrode in the case of the quantitation of 5-HT and 5-HIAA and +0.70 V in the case of the quantitation of CAs, DOPAC and HVA. In the mobile phase, 0.15 M monochloroacetate buffer, pH 3.5, containing 9% methanol in the case of the quantitation of 5-HT, 5-HIAA, DOPAC and HVA and 0.5 mM sodium octyl sulfate in the case of the quantitation of CAs were pumped at a rate of 0.6 ml/min at room temperature. The retention times of noradrenaline (NA), DHB and DA were 9.0, 15.3 and 20.6 min, respectively. The concentration of each compound was calculated from the peak height ratio to the respective internal standard and compared with authentic compounds which were run through the whole procedure.

**Drugs and others:** DHB HBr was obtained from the Aldrich Chemical Co., WI, U.S.A. N-methyl-5-HT oxalate, 5-HT creatinine sulfate, 5-HIAA, DA HCl, NA bitartrate, DOPAC and HVA were purchased from the Sigma Chemical Co., MO, U.S.A. 3,4-Dihydroxyphenylpropionic acid (DOPPA) was prepared from 3,4-dihydroxycinnamic acid (Aldrich Chemical Co.) by catalytic hydrogenation at room temperature and atmospheric pressure using 10% palladium on carbon as a catalyst. Values are expressed as the average±S.E. The statistical significance of differences between mean values was analyzed using Student’s t-test.

**Results**

Our quantitation method for 5-HT and 5-HIAA used in this study is based upon that of Lackovic et al. (10). First, the isolated SCG was deproteinized by 10% ZnSO₄ solutions and the homogenate alkalinized by the addition of NaOH. The advantage of this method is that a minute amount of 5-HIAA can be measured because 5-HIAA is more stable in alkaline than in acidic solution. The CAs, DOPAC and HVA are stable in acidic solution; therefore, we divided the ZnSO₄ homogenate into two parts, and 0.5 N NaOH was added to one part for the quantitation of the 5-HT and 5-HIAA, and 3% HClO₄ was added to the other part for the quantitation of the CAs, DOPAC and HVA. Figure 1 and 2 show typical chromatograms obtained from rat SCGs (A) and capsule (B). The DOPAC (No. 1 peak in chromatograms), 5-HT (No. 2), N-methyl-5-HT (No. 3), DHB (No. 4), 5-HIAA (No. 5) and HVA (No. 6) were eluted from a reverse phase µ-Bondapak C₁₈ column in the LCEC system with retention times of 15.0, 16.5, 21.5, 27.0 and 36.0 min, respectively. Under the conditions described in the section on Materials and Methods, a good separation of each compound was obtained.

Chromatograms of Fig. 1 were obtained from ZnSO₄-HClO₄ homogenates from rat SCGs (A) and capsule (B). The decrease of the peak height of DOPAC (No. 1) and HVA (No. 6) in a chromatogram (A2) obtained from the SCGs of a rat decapitated 15 min after pargyline (75 mg/kg, i.p.) treatment was observed when compared with that (A1) from SCGs of a control rat. The peaks of DOPAC and HVA were not observed in chromatogram (B) obtained from the capsule of the SCG. Although all peaks of the object compounds were present on the chromatogram (A1), calibration curves illustrated in the upper portion of the figure indicate the lack of linearity in 5-HT and 5-HIAA (the dotted lines), unlike the strict linearity of DOPAC and HVA. Standard curves for DOPAC and HVA were linear between 50 pg to 2 ng and 100 pg to 2 ng, respectively. A detection limit of 10–20 pg of DOPAC and 50 pg of HVA was obtained when it was defined as the amount giving a peak twice as high as the fluctuation from the baseline. As mentioned above, 5-HT and 5-HIAA could not be quantified under the conditions for measurement of DOPAC and HVA.
5-HT and 5-HIAA obtained from ZnSO₄-NaOH homogenates of rat SCGs are shown in Fig. 2. The pargyline (75 mg/kg, i.p.) treatment increased the peak height of 5-HT and decreased that of 5-HIAA, 15 min after injection (A2 in Fig. 2). The peak of HVA was not observed as HVA cannot be oxidized at

Fig. 1. Typical chromatograms of ganglional DOPAC and HVA. (A1) Chromatogram of two combined SCGs of a control rat, (A2) chromatogram of the SCGs of a rat treated with pargyline (75 mg/kg, i.p., 15 min), and (B) chromatogram of the capsule of a control rat SCG. Peak identities: 1, DOPAC; 2, 5-HT; 3, N-methyl-5-HT (internal standard); 4, DOPPA (internal standard); 5, 5-HIAA; and 6, HVA. These chromatograms were obtained under the conditions for the quantitation of DOPAC and HVA. The DOPAC, DOPPA and HVA were eluted from a reverse phase μ-Bondapak C₁₈ column in a LCEC system with retention times of 15.0, 24.6 and 36.0 min, respectively. Calibration curves of DOPAC (●), HVA (▲), 5-HT (○, dotted line) and 5-HIAA (△, dotted line) are illustrated in the upper portion of the figure. No lineairities of 5-HT and 5-HIAA were observed, unlike the strict lineairities of DOPAC and HVA. The decrease of the peak heights of DOPAC (1) and HVA (6) are illustrated in the chromatograms (A1, A2) after the pargyline treatment. No peaks of DOPAC and HVA were observed in the chromatogram of the capsule (B).

Fig. 2. Typical chromatograms of ganglional 5-HT and 5-HIAA. (A1) Chromatogram of two combined SCGs of a control rat, (A2) chromatogram of the SCGs of a rat treated with pargyline (75 mg/kg, i.p., 15 min), and (B) chromatogram of the capsule of a control rat SCG. Peak identities: 1, DOPAC; 2, 5-HT; 3, N-methyl-5-HT (internal standard); 4, DOPPA (internal standard); 5, 5-HIAA. The chromatograms were obtained under the conditions for the quantitation of 5-HT and 5-HIAA. The 5-HT, N-methyl-5-HT and 5-HIAA were eluted from a reverse phase μ-Bondapak C₁₈ column in a LCEC system with retention times of 16.5, 21.5 and 27.0 min, respectively. Calibration curves of 5-HT (●), 5-HIAA (▲) and DOPAC (○, dotted line) are illustrated in the upper portion of the figure. The linearity of DOPAC was not observed, unlike the strict linearity of 5-HT and 5-HIAA. As illustrated in the chromatogram of A2, pargyline increased the peak height of 5-HT (2) and decreased the peak height of 5-HIAA (5).
the potential of +0.55 V of a carbon electrode vs. Ag/AgCl reference electrode (11). As in Fig. 1, although we can observe the peak of DOPAC, the calibratory linearity of DOPAC was not observed, as indicated by the dotted line in the calibration curves cited in the upper portion of Fig. 2. Standard curves for 5-HT and 5-HIAA were linear between 50 pg to 2 ng. A detection limit of 20 pg of 5-HT and 5-HIAA was obtained.

The CAs, DOPAC, HVA, 5-HT, 5-HIAA contents of SCG are summarized in Table 1. A minute amount of HVA was detected. The 5-HT was found both in the capsule of SCG and the SCG itself. As shown in Table 1, although the 5-HIAA, a main metabolite of 5-HT, was detected in the SCG, 5-HIAA was not measurable or was a smaller value than our minimum detection limit of 20 pg/sample in the rat SCG capsule. Additionally, when we calculated the molecular ratio of DOPAC to DA and 5-HIAA to 5-HT, the former was 0.57±0.05 (Each value is the average±S.E. of 8 determinations.) and the latter was 0.10±0.01. We found that the DOPAC/DA ratio was significantly higher than the 5-HIAA/5-HT ratio (P<0.001, Student’s t-test).

The treatment with pargyline (75 mg/kg, i.p.), a monoamine oxidase inhibitor, increased the content of DA and 5-HT with a decline of DOPAC, HVA and 5-HIAA. Figure 3A shows that the DA content rapidly increased, and the DOPAC content decreased exponentially. The increase of DA reached a plateau 5 min after pargyline treatment. The decline of HVA was slower than that of DOPAC. HVA was not detected 30 min after

<table>
<thead>
<tr>
<th>Table 1. Biogenic amines (5-HT, DA and NA) and their acidic metabolites (5-HIAA, DOPAC and HVA) contents in rat SCG and capsule</th>
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<tbody>
<tr>
<td>5-HT</td>
</tr>
<tr>
<td>SCG (ng/ganglion)</td>
</tr>
<tr>
<td>Capsule (ng/capsule)</td>
</tr>
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</table>

Results are the average±S.E. of 8 determinations.

Fig. 3. Changes in amines and metabolites contents (ng/ganglion) in the rat SCG after treatment with pargyline. Pargyline was injected i.p., and the rats were decapitated at the time intervals indicated. The contents of DA (●), DOPAC (●, dotted line) and HVA (▲, dotted line) are illustrated in (A) of the figure. 5-HT (●) and 5-HIAA (●, dotted line) in (B) of the figure. In (B), no changes of capsule 5-HT content (▲) are indicated after treatment with pargyline. Results are the average±S.E. of 5 rats. *P<0.05, **P<0.01 and ***P<0.001, when compared with 0 time.
the pargyline treatment. Although the increment of DA was rapid, the 5-HT content increased gradually (Fig. 3B). There were no significant differences between the values at 0, 5, and 15 min. Interestingly, the content of 5-HT in the capsule remained the same at all times after the pargyline treatment.

Decentralization of the right side SCG induced a significant decrease of DOPAC content (RS-decentralized SCG) with a small increase in the left, sham-operated side (LS-sham operated SCG). Unexpectedly, we observed no changes in the content of 5-HIAA both in the decentralized side and the sham-operated side of the SCG (Table 2). This observation is noteworthy compared to the findings on the change of DOPAC content after decentralization.

The effects of preganglionic stimulation on the contents of DOPAC, HVA and 5-HIAA are presented in Table 3. Whereas the content of DOPAC increased by 91% and 666%, respectively, at 3 and 10 min after stimulation (10 V, 10 Hz, 1 msec duration) we observed no increase in 5-HIAA content in the stimulated-right side of the SCG (RS-stimulated SCG).

Table 2. Effect of decentralization on biogenic amines (5-HT and DA) and their acidic metabolites (5-HIAA, DOPAC and HVA) contents (ng/ganglion) in rat SCG

<table>
<thead>
<tr>
<th>Condition</th>
<th>5-HT (ng)</th>
<th>5-HIAA (ng)</th>
<th>DA (ng)</th>
<th>DOPAC (ng)</th>
<th>HVA (ng)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>1.20±0.10</td>
<td>0.11±0.02</td>
<td>3.05±0.10</td>
<td>1.61±0.25</td>
<td>0.12±0.02</td>
</tr>
<tr>
<td>RS-sham operated SCG</td>
<td>1.18±0.22</td>
<td>0.13±0.02</td>
<td>2.78±0.15</td>
<td>1.67±0.26</td>
<td>0.12±0.01</td>
</tr>
<tr>
<td>Decentralization</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RS-decentralized SCG</td>
<td>1.16±0.18</td>
<td>0.08±0.02</td>
<td>2.69±0.22</td>
<td>0.91±0.10*</td>
<td>0.09±0.02</td>
</tr>
<tr>
<td>LS-sham operated SCG</td>
<td>1.08±0.18</td>
<td>0.14±0.03</td>
<td>3.04±0.21</td>
<td>1.90±0.17</td>
<td>0.14±0.03</td>
</tr>
</tbody>
</table>

Decentralization was achieved by aseptic removal of the right sympathetic chain caudal to the ganglion (RS-decentralized SCG). The decentralized and sham operated rats were decapitated 7 days after surgery. RS: right side, LS: left side. Results are the average±S.E. of 5 determinations. *P<0.05 when compared with the RS-sham operated SCG.

Table 3. Effect of electrical stimulation on 5-HIAA, DOPAC and HVA contents (ng/ganglion) in rat SCG

<table>
<thead>
<tr>
<th>Condition</th>
<th>5-HIAA (ng)</th>
<th>DOPAC (ng)</th>
<th>HVA (ng)</th>
</tr>
</thead>
<tbody>
<tr>
<td>LS-control SCG</td>
<td>0.14±0.02</td>
<td>1.70±0.24</td>
<td>0.11±0.02</td>
</tr>
<tr>
<td>RS-stimulated SCG</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3 min</td>
<td>0.12±0.02</td>
<td>3.24±0.40*</td>
<td>0.18±0.04</td>
</tr>
<tr>
<td>10 min</td>
<td>0.13±0.04</td>
<td>13.03±0.17**</td>
<td>0.27±0.04*</td>
</tr>
</tbody>
</table>

Electrical stimulation (10 V, 10 Hz, 1 msec duration) to the right preganglionic nerves was performed after the bilateral SCG and preganglionic nerves were exposed following anaesthetization with urethane (1.2 g/kg, s.c.) for 3 and 10 min. RS: right side, LS: left side. Results are the average±S.E. of 5 determinations. *P<0.02, **P<0.001 when compared with the LS-nonstimulated (control) SCG.

Discussion

We attempted to determine whether the serotonergic SIF cell is functional in ganglionic transmission between the preganglionic sympathetic neurons. Efforts were made to quantitate trace amounts of 5-HT and 5-HIAA. As described in Figs. 1 and 2, our modifications of the quantitative analysis of biogenic amines and their metabolites led to investigations of endogenous 5-HT systems in the rat SCG. This LCEC method is equally matched in sensitivity to the gas chromatographic-mass spectrometric method (12) and to the radioenzymatic microassay method (13). We measured the 5-HT and 5-HIAA contents in the rat SCG and its capsule separately because the capsule was presumed to have 5-HT storage cells, i.e., the mast cells, and it was technically...
difficult to avoid contamination from surface-agglutinating blood which contains thrombocytic 5-HT, even when the capsulated SCG was extensively washed in an ice-water bath. In fact, a relatively large amount of 5-HT existed in the capsule without detection of 5-HIAA. The low value of the ratio of 5-HIAA to 5-HT in the SCG is noteworthy. This is of particular interest in view of the finding that a high ratio of the central serotonergic neuron system was noted in a study concerning the regional brain contents of 5-HT and 5-HIAA (14). According to the data that the ratio is an index of changes in the activity of the monoaminergic system (15), this result suggests that the rat SCG has a low 5-HT neuronal activity.

The effect of pargyline (75 mg/kg, i.p.) on the content of DA and DOPAC in this study is in complete accordance with findings in previous reports (16, 17). It has been thought that the turnover rate of DA in the dopaminergic SIF cell of the sympathetic ganglion is the same as the central dopaminergic neuron. The mode of the pargyline-induced 5-HT accumulation and 5-HIAA decrease may indicate low neuronal activity of the 5-HT system in the rat SCG compared with findings in the case of the central serotonergic neuron system (18). However, until more data are obtained with regard to the rate of formation of 5-HT and 5-HIAA calculated from the steady-state of kinetics, the low neuronal activity of the rat ganglionic 5-HT system remains only speculative. When we compared the decreased rate of the DOPAC content induced by pargyline, the ganglionic 5-HT content increased gradually. This discrepancy may be explained by the suggestion that the nerve activity of the dopaminergic SIF cell is higher than that of the serotonergic SIF cell. The resulting high DOPAC/DA ratio and the low 5-HIAA/5-HT ratio support this idea, but one might question whether such a difference between dopaminergic and serotonergic metabolism reflects the deamination process of DA and 5-HT. Actually, 5-HT seems to be mainly deaminated by monoamine oxidase (MAO)-A, and the DA is deaminated by MAO-A and MAO-B in the rat brain (19). Moreover, pargyline is an inhibitor of MAO-B (20). If such is indeed the case, then the different increases of DA and 5-HT may be based on neuronal activity, since a high dose (75 mg/kg) of pargyline can block both types of MAO (21). However, the present finding that pargyline did not increase 5-HT content in the SCG capsule, an area considered to contain mast cells and/or a small amount of blood, but did increase the 5-HT content of the SCG, supports the idea that 5-HT is produced by the neuronal structure in the rat SCG. We assumed that the 5-HT detected in the capsule was mainly derived from platelets of agglutinated blood, because platelets have only a small amount of tryptophan-hydroxylase (22), the rate-limiting enzyme of the biosynthesis of 5-HT, and take up the 5-HT from the blood and store it in the granules (23). As rat mast cells contain 5-HT (24), these cells are candidates for the origin of capsule 5-HT.

The presence of specific receptors for 5-HT on ganglion cells in the SCG (25, 26), and the existence of a 5-HT cell indicated by immunohistochemical studies (4) led to the hypotheses that a serotonergic SIF cell may lie between the pre- and postganglionic sympathetic neurons. We did not detect any decrease of 5-HIAA content in the SCG after decentralization or any increase by preganglionic electrical stimulation (Tables 2 and 3). The serotonergic SIF cell does not seem to be stimulated by acetylcholine from the nerve endings of preganglionic sympathetic neurons. The serotonergic SIF cell may not influence the ganglionic neuronal transmission in the rat SCG. Similarly, unique and isolated serotonergic neurons were found in the rat pancreas (27) and in lamina X of the monkey spinal cord (28). Particularly, the latter spinal interneurons are considered to be one of the cerebrospinal-contacting neurons and to control spinal blood flow through vessel innervation (28). These studies suggest the physiological significance of the serotonergic SIF cell in the rat SCG. The possibility that the serotonergic SIF cell is innervated by the recurrent collateral from the postganglionic noradrenergic neuron cannot be entirely excluded since the ganglion cells have specific receptors for 5-HT (25, 26).
The changes in DOPAC presented in this study are in accord with data of other workers (17, 29). Electrical stimulation induced a dramatic increase in the DOPAC content, and the elevation was dependent on the time length of stimulation. Thus, measuring the DOPAC content in sympathetic ganglia may be a useful approach to assess the preganglionic sympathetic activity which is controlled by the descending bulbospinal monoaminergic neurons (30).

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