ORIGINAL RESEARCH

# Anatomical Analysis of the Cervical Sympathetic Ganglion and Spinal Ganglion and its Physiological Significance in the Pathogenesis of Cervical Vertigo

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## ABSTRACT

**Objective** • Investigating the anatomical connections between cervical sympathetic ganglia and spinal ganglia in rabbits and assessing the role of Neuropeptide Y in the pathogenesis of cervical vertigo.

**Method** • Part 1: 32 adult healthy male New Zealand white rabbits (whose skin is very sensitive, so rabbits are generally used for stimulation experiments) were randomly divided into the upper cervical sympathetic ganglia (SCSG) stimulation group and the lower cervical sympathetic ganglia (ICSG) stimulation group, with 16 rabbits in each group. The two groups were divided into an experimental group and a control group, with 8 rabbits in each group. The cervical ganglia of each group of white rabbits were injected with 4% FluoroGold solution and observed under a section microscope. Part 2: Sixty New Zealand white rabbits were randomly divided into a blank control (n = 12), SCSG stimulation group (n = 12), SCSG sham surgery control (n = 12), ICSG stimulation group (n = 12), and ICSG sham surgery control group (n = 12). The SCSG group and ICSG group were subjected to electrical stimulation (i.e. 30.0Hz, 10.0V, 5-minute pulse width of 0.5 ms square wave pulse), and specimens were made. The expression of NPY was detected using immunohistochemical methods.

**Result** • Neuropeptide Y was weakly expressed in all cervical ganglia (C1-C8). Compared with the sham surgery group, the superior cervical sympathetic ganglion stimulation group showed an increase in Neuropeptide Y positive cells in C2, C3, C4, and C5, with C2 and C3 showing the most significant increase. The number of C6, C7, and C8 Neuropeptide Y positive cells in the 3 C, 3D and 4B, lower cervical sympathetic ganglion stimulated groups was higher than in the sham sham-operated group, and C6 and C7 significantly increased. Neuropeptide Y is like immunoreactive neurons in the cervical spinal ganglia, and the immunoreactive products are small brown particles distributed in the cytoplasm after electrical stimulation of the cervical sympathetic ganglia. The Neuropeptide Y content in the corresponding segment of the cervical spinal ganglia is significantly increased compared to the control group ($P < .05$).

**Conclusion** • In New Zealand white rabbits, nerve fibers are interconnected between the cervical sympathetic ganglion and the cervical spinal ganglion, and this neural fiber connection has a certain segmental nature, providing experimental basis for the existence of the cervical spinal cord external nerve reflex arc and elucidating the pathogenesis of cervical vertigo in terms of neural anatomy. By using neuroelectrophysiological methods, it has been confirmed that electrical stimulation in the cervical spinal ganglia can reach the corresponding cervical sympathetic ganglia on the same side through a certain conduction pathway, providing experimental basis in neuroelectrophysiology for the existence of the cervical extraspinal nerve reflex arc and elucidating the pathogenesis of cervical vertigo. NPY may be involved in the pathogenesis of cervical vertigo, providing a theoretical basis for the clinical diagnosis of cervical vertigo. (*Altern Ther Health Med.* 2024;30(5):228-234)


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## INTRODUCTION

There are three paravertebral nerves in the neck: the superior cervical ganglia, the middle cervical ganglia, and the inferior cervical ganglia. Internodal branches interconnect these three ganglia; The internodal branch is generally one branch, but sometimes the internodal branch between the superior and middle cervical ganglia is two branches, and the internodal branch between the middle and inferior cervical ganglia is multiple branches. The paravertebral ganglia of the cervical spine are located on the deep side of the anterior fascia. The fibers before the festival come from the sympathetic trunk above the chest, so there is a lack of white communicating branches. After the festival, the fibers form the gray communicating branches,

which are respectively connected to all cervical nerves. Cervical vertigo is a common clinical disease, which is a syndrome with the main symptom of dizziness, usually caused by cervical degeneration, soft tissue strain, trauma, dislocation of small joints of the cervical spine, and other cervical factors that cause internal and external disorders of the cervical spine. The common symptoms include dizziness, vertigo, headache, nausea, vomiting, memory loss, palpitation, and other autonomic nervous symptoms and even cataplexy in severe cases.[1-2]

At present, the clinical understanding of the pathogenesis of cervical vertigo is still unclear. The theory of cervical vertigo caused by vascular factors such as vertebral artery stenosis and compression is increasingly questioned by the academic community, while the role of the changes in the humoral factors, plasma endothelin (ET), and calcitonin gene-related peptide (CGRP) induced by the theory of sympathetic nerve stimulation has increasingly been highly valued by the academic circles at home and abroad in the pathogenesis of cervical vertigo.[3-4] Under normal physiological conditions, the levels of ET and CGRP maintain a relative balance, and jointly maintain the vasoconstriction function of cerebral blood vessels to regulate cerebral blood flow changes. When the sympathetic cervical ganglion is stimulated, the ET/CGRP ratio is unbalanced, leading to dizziness.[5] Previous studies have confirmed[6] that sympathetic nerve fibers are widely distributed on and around the vertebral arterial surface and that chemically conducted substances released from sympathetic postganglionic fibers control the relaxation and contraction of the vertebral veins. When the sympathetic nerve is abnormally stimulated, the vertebral artery will undergo spastic changes, which will cause the posterior circulation ischemia to produce vertigo symptoms.[7] Some authors have found that sympathetic nerve fibers are relatively segmental on the wall of the vertebral artery.[8-9]

Neuropeptide Y (NPY) is mainly synthesized by sympathetic and parasympathetic neurons in the peripheral nervous system, and is widely expressed in the central nervous system. It plays different roles in various physiological processes, and neuropeptides closely related to stress play important regulatory roles in both the peripheral and central nervous systems. It is particularly closely related to the cardiovascular system and plays an important role in vasoconstriction, It is currently one of the known vasoconstricting substances.[10-13] However, there is limited information on cervical spinal ganglion NPY expression after cervical vertigo, and the relationship between stress NPY and cervical vertigo is unclear. And by electrically stimulating the cervical spinal ganglia, the changes in neuropeptide Y in the cervical sympathetic ganglia were observed, further confirming the connection between the cervical spinal ganglia and the sympathetic ganglia in terms of neuroelectrophysiological functions, thus further elucidating the pathogenesis of cervical vertigo.

## MATERIALS AND METHODS

### Research materials

Healthy New Zealand Large White rabbits, 2~3 kg, were purchased from Shandong Nongkeyuan Animal Husbandry Development Center, Shandong Province, China; viral vectors (The production license number is SCXK2003-0003) pAAV-CAG-MCS-mCherry-3FLA AOV003, and viral vectors, pAAV-CMV-MCS-EGFP-3FLAG, were purchased from Hoyuan Bio-technology (Shanghai) Company Ltd. in Shanghai, Shanghai, China; fluorescence microscope (model no. BX51) was purchased from Olympus Corporation, Olympus Trading (Shanghai) Co., Ltd, Pudong New Area, Shanghai, China; micro-syringe (5 μL) was purchased from Shanghai Medical Laser Instrument Factory, Minhang District, Shanghai, China; image acquisition and processing system (-Instrument model: 4.5.0.19) was purchased from Shanghai Yuyan Scientific Instrument Company Limited, Minhang District, Shanghai, China; and anti-sheep polyclonal antibody-NPY was purchased from Able Antibodies, Shanghai Free Trade Zone, Shanghai, China. Ltd. in Shanghai Pilot Free Trade Zone, China; horseradish enzyme labeled rabbit anti-goat IgG polymer was purchased from Guangzhou Dingguo Biotechnology Co. All animal manipulations were performed in accordance with National Institutes of Health guidelines and approved by the Ethics Committee for Animal Experimentation of Shandong University (Approval number: SYYXY2015010601). Conforming to ethical beliefs, moral attitudes, and behavioral norms regarding the relationship between humans and animals

### Methods

**Virus vector method.** (1) Grouping: Using New Zealand white rabbits for the experiment, 32 adult healthy male New Zealand white rabbits weighing 2 kg~2.5 kg were randomly divided into 2 groups, with 16 rabbits in each group 16 of them were placed in groups receiving stimulation to the superior cervical sympathetic ganglia (SCSG) and 16 more were placed in groups receiving stimulation to the inferior cervical sympathetic ganglia (ICSG). Eight rabbits from the SCSG group were split into a control group and an experimental group. The same division of ICSG was made, with 8 rabbits per group, into a comparison group and an experimental group.

(2) Animal tracer injection: The New Zealand white rabbits were placed on the laboratory platform in prone position and given intravenously chloral hydrate (60 mg/kg) to anesthetize. Under sterile conditions, the matching cervical and symphonic ganglion is visible in New Zealand White rabbits. A 2 mL dose of 4% solution of FluoroGold was infused into the relevant ganglion. While the comparison rabbit's ganglia received a standard saline injection. The pAAV-CAG-MCS-mCherry-3FLA and AOV003 cis-transsynaptic virus vectors were used for cis-labeling.

(3) Preparation and injection of virus vector: The virus vector technology GRASP GFP (green fluorescence protein, GFP) technology was used. Chloral hydrate was used to

anesthetize the white rabbits, and the white rabbits were fixed on the operating table, with the head facing the operator. An opening about one centimeter was cut in the neck of the white rabbits after shaving and disinfection, and a miniature vascular clamp was used to separate it to find the sympathetic ganglion bluntly. The sympathetic ganglia were fixed, and 5 μL was injected with a microinjector. After injection, the needle was pulled out after keeping for 5 minutes, and the sympathetic ganglion was put back in place. Then, the skin was sewed and sterilized. To prevent the overflow of tracer from the injection site, the injection should be done gently and the needle should be left in the spinal ganglion for 5 minutes. The New Zealand white rabbits were then fed and given 14 days of unrestricted movement. The same source should be used to obtain new animals to replace lost or damaged rabbits throughout the trial.

(4) Making section and observation: The rabbits were anesthetized, and then normal saline was injected to kill them. Following perfusion, the sympathetic ganglia and cervical spinal cord were promptly severed, preserved with formalin for six hours, and submerged entirely in 10% sucrose. Using a fluorescence microscope, the frozen sections were sliced into 30 micron-thick pieces, placed in a 0.1% phosphate buffer (including 5% sucrose), and examined.

**Neuroelectrophysiological study.** (1) Grouping: Sixty adult healthy male New Zealand white rabbits weighing 2 kg to 2.5 kg were randomly divided into five groups: blank control (n = 12, without any treatment), superior cervical sympathetic ganglia (SCSG) stimulation group (n = 12), SCSG sham surgery control (n = 12, rabbits received the same SCSG exposure and electrode penetration surgery without electrical stimulation) Inferior cervical sympathetic ganglia (ICSG) stimulation (n = 12), and ICSG sham surgery control group (n = 12, rabbits received the same ICSG exposure and electrode penetration surgery without electrical stimulation).

(2) Electrical stimulation of sympathetic nerve: Intraperitoneal infusion of 10% chloroform acetate hydrate (150 mg/kg of body mass) was utilized to anesthetize the animals before the rabbits were placed on the experimental platform in the supine posture. To prevent contamination from bacteria or other microbes, the experiment was carried out in sterile settings. After administration of chloral hydrate, the neck fur was removed, and the corresponding SCSG or ICSG was exposed monolaterally via a lateral ventral incision in the neck. Then, rubber gloves separated the corresponding ganglia from the surrounding tissues and stimulated the electrode with internal acupuncture. The electrical stimulation (i.e., 30.0Hz, 10.0V, 5-min square wave pulse with a pulse width of 0.5 ms) used in the SCSG group and ICSG group was the same, which was delivered by pulse generators (Nihon Kohden, SEN-7103). The identical electrode penetration surgery and cervical sympathetic ganglion exposure were administered to the rabbits in the equivalent comparison group without any electrical stimulation.

(3) Sample collection: After electrical stimulation, normal saline (37°C, 300-400 mL) was immediately used to rapidly perfuse the left ventricle, then 4% paraformaldehyde was used to slowly perfuse the left ventricle, and then the animals were killed at room temperature. The cervical ganglion (C1-C8) of the ipsilateral spine was removed and stored for a period of 4 hours at room temperature after perfusion with 4% paraformaldehyde for one hour. The samples were then completely immersed in 10%, 20%, and 30% sucrose concentrates for gradient rehydration. Frozen samples were divided into 8 mm thick slices along the long axis of the ganglion. For further immunohistochemical testing, three glass slices per sample were selected from the ganglion's anterior, middle and posterior parts. Detect the expression of NPY protein. Take paraffin specimens from each group and bake them overnight at 65°C. Dewax and inactivate them by hydration. Rinse them with PBS three times and seal them for 10 minutes. Pour the serum and incubate them at room temperature with the first antibody (anti-NPY antibody 1:100) for 1 hour. Rinse them with PBS three times. Incubate them with the second antibody (horseradish peroxidase-labeled second antibody) for 25 minutes. Color them with DAB solution for 10 minutes. Retain them with hematoxylin, bluish them, dehydrate them with gradient alcohol, dry them, and seal them with xylene transparent and neutral gum; randomly select 10 400 × Field of view, calculate the staining result based on the staining intensity and percentage of positive cells in the section. NPY protein is mainly present in the cytoplasm, and the degree of cell coloration is divided into 0 (basically non-staining), 1 (slightly lighter color), 2 (moderate color), and 3 (darker color) points. The proportion of positive cells is divided into 0 (non staining cells), 1 (staining cells<30%), 2 (30% ≤ staining cells<60%), and 3 (staining cells ≥ 60%) points. The combined score of the two is negative (0-2 points, -), positive (3-4 points,+), and strongly positive (5-6 points,++).

### Statistical methods

SPSS 13.0 was utilized to statistically analyze all the data in this research (SPSS, Chicago, IL). The information was presented as mean ± standard variation. The optical density readings were contrasted using the Student's *t* test (OD). It was deemed Statistically significant when $P < .05$.

## RESULTS

### Analysis of the distribution of virus vectors

Twenty-one days after GFP infusion into the upper or lower carotid sympathetic ganglion, no GFP labeling was seen in the bilateral spinous ganglion of the controlling group that received normal saline infusion, but sporadic and variable numbers of GFP-labeled nociceptors were seen in the ipsilateral spinous ganglion of the experimental group, as illustrated in Table 1 and Figure 1. After GFP infusion into the superomedial cervical sympathetic ganglion, FG GFP-labeled neurons were mainly seen in the ipsilateral C2-C5 spinous ganglion, while more GFP-labeled neurons were

**Table 1.** Distribution of virus vector labeled neurons on SCG/IGG

| | Superior cervical sympathetic ganglion | | | Inferior cervical sympathetic ganglion | | |
|---|---|---|---|---|---|---|
| | experimental group | | | experimental group | | |
| | Ipsilateral | Contralateral | control group | Ipsilateral | Contralateral | control group |
| C2 | 9 | 0 | 0 | 0 | 0 | 0 |
| C3 | 9 | 0 | 0 | 1 | 0 | 0 |
| C4 | 5 | 0 | 0 | 7 | 0 | 0 |
| C5 | 1 | 0 | 0 | 8 | 0 | 0 |
| C6 | 0 | 0 | 0 | 10 | 0 | 0 |

**Figure 1.** GFP-labeled neurons in the experimental group after injecting GFP into the superior or inferior cervical sympathetic ganglia

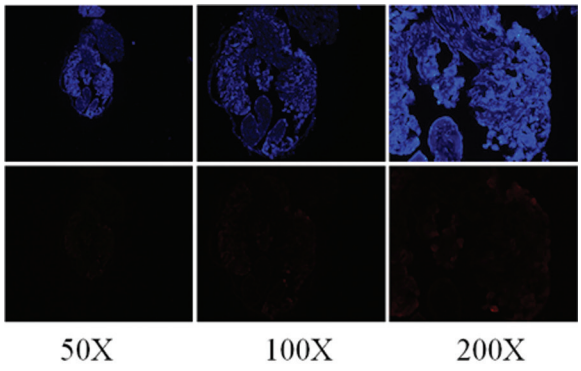



**Figure 2.** GFP-labeled neurons in the ipsilateral spinal ganglia of the experimental group after injecting FG into the cervical sympathetic ganglia

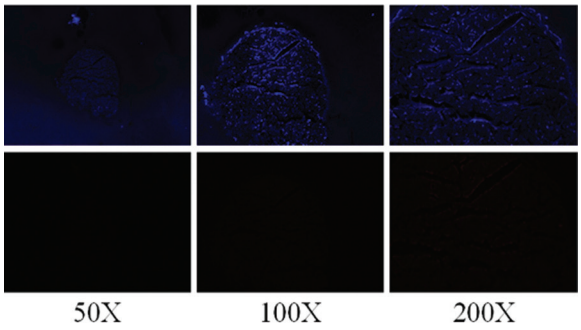
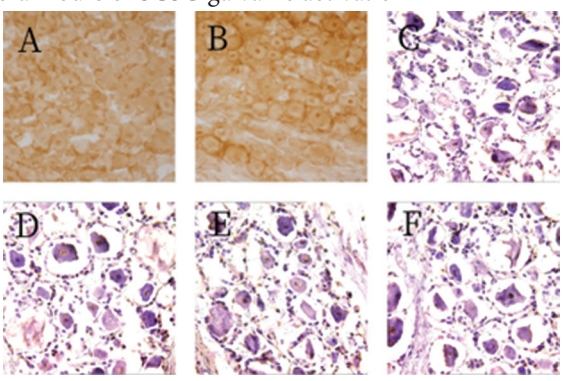
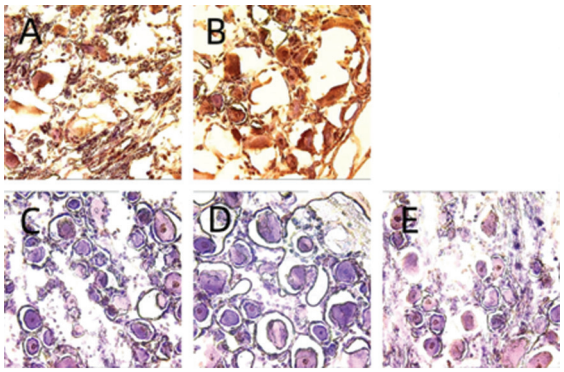



**Figure 3.** Immunohistochemical staining results of cerebrospinal peptide Y in C2-C5 carotid ganglion after several hours of SCSG galvanic activation




Note: Typical immunohistochemical images of neuropeptide Y were magnified 200 times in null controlling (A, C2), fictitious manipulation (B, C2), and SCSG stimulation (C, C2; D, C3; E, C4; F, C5). Dark-colored cells with nuclei containing positive cells for ceruloplasmin Y.

**Figure 4** After electrical stimulation of the inferior carotid sympathetic nerve ganglion, the findings of an immunohistochemical test for NPY in the C6-C8 ganglion (ICSG)




Note: ICSG stimulation group, bogus operation group (B, C6), and blank control group (A, C6) (C, C6), NPY immunohistochemical representative image (200x, D, C7, D, C8) group. The cells with brown nucleus were NPY-positive cells.

seen in the C3 and C4 spinous ganglia, as illustrated in Table 1 and Figure 2. After GFP infusion into the lower neck sympathetic ganglion, GFP labeling was found mainly in the ipsilateral C5-C8 spinous ganglion.

**Changes of NYP expression level in normal cervical spinal ganglion**

NPY immunohistochemistry was utilized to discover the amount of NPY in the nerve ganglion of the cervix under physiological conditions. As shown in the figure below, only a few cells showed positive nuclear staining, indicating that NPY was only minimally expressed in all intracervical vertebral segments (C1-C8).

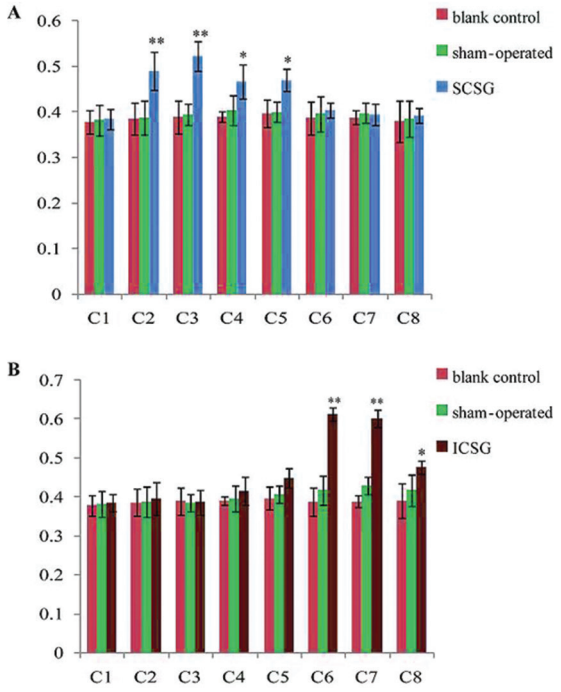
**Effect of electrical stimulation on NPY representation in the posterior cervical C6-C8 ganglion**

Because mechanical stimulation had no effect on the degree of NPY expression, electrical stimulation was carried out in SSCG or ISCG. In order to find out whether NPY was exported in the neck ganglion and immunohistochemical analysis was performed. NPY-positive codes in C2, C3, C4 and C5 in the SCSG-stimulated group increased as compared to the sham surgery group, with C2 and C3 showing the greatest rise. According to Figure 4, more C6, C7, and C8 NPY positive cells were present in the 3C-D and 4B, ICSG stimulation groups than in the sham operation group, and C6 and C7 levels were considerably greater.

**Effects of electrical stimulation on NPY representation in ipsilateral C1-C8 spinous ganglia**

The cervical spinal ganglion included NPY-like immunoreactive neurons, and after electrically stimulating the cervical sympathetic ganglion, the immunoreactive neurons' byproducts were minute brown particles dispersed throughout the cytoplasm. As can be shown in Figure 5, the NPY content in the corresponding cervical spinal ganglion was considerably higher than that in the control group (0.361±0.008, 0.361±0.009,

**Figure 5.** Following ganglion stimulation on the unilateral upper (A) and lower (B) cervical sympathetic, quantitative study of ipsilateral ithermal cervical ganglia (C1-C8) in Neural Peptide Y.




0.376±0.021, 0.375±0.021, 0.383±0.021, 0.310±0.003, 0.374±0.015, 0.351±0.032) ($P < .05$).

## DISCUSSION

Cervical vertigo is the common terminus of cervical lesions.[14] Many other symptoms, such as nerve stimulation and vertebrobasilar insufficiency coupled with vertigo, tinnitus, dizziness, facial discomfort, arm pain, and migraines, may result from lesions in the upper cervical spine (C0-C2). In addition to chronic neck pain, lesions in the lower cervical spine (C3-C7) can also result in muscle spasms, a twisted voice, and/or sensory problems.[15-16] For the research on the pathogenesis of cervical vertigo, the early research mainly focused on the factors of the vertebral artery, namely vascular factors.[17] However, with the deep development of medical practice and imaging technology, it is found that the vertebral artery examination in many patients with cervical vertigo is normal, while there is no clinical symptoms in patients with long-term compression of vertebral artery revealed by imaging. While the role of sympathetic nerve stimulation theory in the pathogenesis of cervical vertigo is increasingly highly valued by academic circles at home and abroad.[18-19] According to the results of this study, phosphor gold grains were found in the associated sensory ganglion after the infusion of phosphor gold dye into the neck nerve ganglion. The cervical sympathetic ganglion and the cervical spinal ganglion have direct nerve fiber connections based on the axoplasmic transport mechanism of nerve fibers. It is clear that these linkages allow the abnormal somatic feeling of neck pain to be transferred to the cervical sympathetic ganglia. Cervical vertigo was previously divided into upper cervical vertigo and lower cervical vertigo according to the position where the patients suffered from neck pain or discomfort and the experiment has proved that the cervical vertebral lamina and the segmental distribution describes the sympathetic nerve endings on the vertebral artery facet and the spinous nerve endings on the dural facet of the spine. The segmental distribution can also be seen in the neural fiber connections between the carotid sympathetic ganglion and the carotid vertebral ganglion. The inferior carotid sympathetic ganglion is mainly associated with the spinous ganglion of C5 and C6, while the superior cervical sympathetic ganglia are primarily connected to the spinal ganglia of C2 and C3. Both the upper and lower sympathetic ganglia are related to the C4 ganglia, but the connection with the lower sympathetic ganglia is stronger than that with the upper sympathetic ganglia. To further understand the etiology of whiplash dizziness, in this investigation, we performed electrical tests to determine the relationship among the carotid sympathetic ganglion and the carotid ganglion.

At present, NPY has been proven to be an efficient vasoconstrictor, which can cause vasoconstriction by directly acting on the smooth muscle of blood vessels, as well as by enhancing other types of vasoconstrictors such as angiotensin II, endothelin (ET), prostaglandin (PG) α Receptor agonists, serotonin, histamine, etc.) produce vasoconstrictive effects. Scholars have found that when patients experience cervical vertigo, neuropeptide Y in their bodies increases significantly compared to normal. There are also studies speculating that NPY may be one of the long-term pathogenic vascular active factors leading to the onset of cervical vertigo, and it is believed that NPY can be a new indicator for predicting the prognosis of cervical vertigo.[20-21] It has long been thought that modulating neuralgia has a fibular association between the psoas and sensory ganglion. NPY functions as a potent vasoconstrictor. On the one hand, vasoconstriction can result from NPY's direct action on vascular smooth muscle cells. This conclusion has been confirmed in the chronic cold stimulation experiment of rats that chronic cold stimulation can induce gene expression. The increased NPY neurotransmission is offset by the significantly decreased overflow induced by the norepinephrine (NE).[22-23] NPY mainly enhances the vasoconstriction induced by norepinephrine in muscle arteries of adrenergic receptors with α-1.[24-25] NPY can improve the blood vessel-dilating effects of other vasoconstrictors, such as norepinephrine.[26] The increase in NPY expression level caused by chronic stimulation can cause continuous contraction, vertebrobasilar artery hypertrophy and stenosis, which may be one of the pathogenic reasons of cervical vertigo.[27] Research has shown that.[28-30] For many ailments, including stress-related mental illnesses, NPY can be exploited as a therapeutic target. In this study, we first explored the representation of NPY in C1-C8 spinal vertebral Ganglion of the neck. We found only a small number of NPY-positive courts in frozen sections of all

central vertebral ganglia, confirming the widespread presence of this protein in the ganglia. More NPY-positive courts appeared in C2, C3, C4, and C5 (C2 and C3 most obviously) after SCSG stimulation, and also in C6, C7, and C8 according to immunohistochemical and quantitative analysis, after ICSG stimulation (C6 and C7 appeared significantly increased), suggesting that galvanic irritation of sympathetic ganglia can segmentally augment cervical spinal cord neurons' NPY expression.

In this study, we injected the virus vector into the sympathetic ganglion and confirmed that there was a direct nerve fiber connection between the cervical sympathetic ganglion and the spinal ganglion, and its distribution was segmental: There were nerve fibers from the C2 to C5 spinal ganglion in the superior cervical ganglion; there were nerve fibers from the inferior cervical ganglia, C5–C8 spinal ganglia. The aforementioned results demonstrate that a distinct neural pathway exists between the cervical sympathetic ganglion and the cervical spinal ganglion. Usually, these two types of ganglion are regarded as the morphological units that undertake the different nerve activity functions of somatic sensation and visceral movement. Therefore, it can be inferred that the direct neural connection of these different peripheral nerve types (spinal ganglion and sympathetic ganglion) is the neuromorphological basis for the internal connection between clinical somatic symptoms and visceral symptoms. Abnormal cervical stimulation (such as pain) can act on the sympathetic cervical ganglion through this direct nerve connection and then cause corresponding secondary visceral syndrome (such as vertigo) symptoms. For patients with cervical spondylosis, the abnormal nerve activity can be transmitted to the sympathetic ganglia through the corresponding affected cervical spinal ganglia, regardless of whether the main pathological changes are located in the upper cervical vertebra or the lower cervical vertebra, and then the corresponding types of cervical vertigo can be produced.

In conclusion, In New Zealand white rabbits, nerve fibers are interconnected between the cervical sympathetic ganglion and the cervical spinal ganglion, and this neural fiber connection has a certain segmental nature, providing experimental basis for the existence of the cervical spinal cord external nerve reflex arc and elucidating the pathogenesis of cervical vertigo in terms of neural anatomy. By using neuroelectrophysiological methods, it has been confirmed that electrical stimulation in the cervical spinal ganglia can reach the corresponding cervical sympathetic ganglia on the same side through a certain conduction pathway, providing experimental basis in neuroelectrophysiology for the existence of the cervical extraspinal nerve reflex arc and elucidating the pathogenesis of cervical vertigo. NPY may be involved in the pathogenesis of cervical vertigo, providing a theoretical basis for the clinical diagnosis of cervical vertigo. Although this study provides a preliminary introduction to the role of sympathetic nerves in the pathogenesis of cervical vertigo, it is still at the clinical level and requires further in-depth research. For example, in the pathogenesis of cervical vertigo, further research in molecular biology is needed regarding the function of receptors, pathways of neurotransmitter action, and mediations within the sympathetic nervous system. In addition, exploring the pathological changes of neural microcirculation and the specific mechanisms of brain blood supply regulation and compensatory balance that cause cervical vertigo also requires further research in related basic disciplines such as neurophysiology, neuromorphology, and anatomy. Finally, as vertigo involves related disciplines such as otology, ophthalmology, spinal surgery, and neurology, in-depth interdisciplinary research should be conducted to elucidate the pathogenesis of cervical vertigo and guide clinical treatment. This study still uses New Zealand white rabbits as the research object, and further validation is needed on animals with anatomical and functional characteristics closer to humans. During the experiment, spinal nerve ganglion stimulation was performed on live experimental animals, which cannot avoid the influence of the central nervous system and surrounding tissues on the experimental results. Further exploration and improvement of experimental methods are needed to minimize the interference of related factors such as surrounding tissues on the experimental results.

## DATA AVAILABILITY

The experimental data used to support the findings of this study are available from the corresponding author upon request.

## CONFLICTS OF INTEREST

The authors declared that they have no conflicts of interest regarding this work.

## ACKNOWLEDGMENTS

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## AUTHOR CONTRIBUTIONS

Siqiang Qiu and Yong Meng contributed equally to this work.

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