Water stimulation of the posterior oral cavity induces inhibition of gastric motility

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Kobashi, Motoi, Masatoshi Mizutani, and Ryuji Matsu. Water stimulation of the posterior oral cavity induces inhibition of gastric motility. Am J Physiol Regulatory Integrative Comp Physiol 279: R778–R785, 2000.—The response of gastric motility to the administration of water and saline in the larynx and epiglottis was investigated in urethan-chloralose anesthetized rats. Administration of water inhibited motility of the distal stomach, but 0.15 M NaCl did not induce the inhibitory response. Bilateral sectioning of the superior laryngeal nerve (SLN) abolished the inhibitory response induced by water. Bilateral cervical vagotomies abolished the inhibitory responses, although spinal transection did not affect the inhibitory response. These inhibitory responses have been observed in immobilized animals. The degree of inhibition by water and hypotonic saline was negatively correlated with the sodium concentration. In contrast, the degree of inhibition to hypertonic saline was positively correlated with the sodium concentration. The proximal stomach also showed a reduction in intragastric pressure in response to the administration of water. These findings suggest that water-responsive afferent neurons in the SLN suppress gastric motility via the vagal efferent nerve.

Reduction in gastric tone is mediated, in part, by the extrinsic nervous system. Mechanical stimulation of antrum stretch receptors reduces intragastric pressure (1). Osmolarity, caloric density, and macronutrient content inhibit gastric motility or slow gastric emptying, suggesting the presence of intestinal chemoreceptors (4, 15, 21). Thus ingesta either mechanically or chemically stimulate abdominal receptors to cause a reduction in gastric tone. In addition, gastric relaxation can occur before food reaches the stomach and facilitates the reservoir function of the stomach. Cannon and Lieb (9) demonstrated relaxation of the canine stomach during swallowing. Mechanical stimulation of the pharynx and distension of the esophagus had been assumed to induce such relaxation (2). Thus swallowed food itself stimulates the upper alimentary tract, causing relaxation of the stomach.

Theafferent neurons in the superior laryngeal nerve (SLN), which bifurcates from the vagal nerve, are responsible for protective reflexes such as apnea (40) and reflex swallowing (39). The SLN, which innervates taste buds distributed on the laryngeal surface of the epiglottis and on the larynx, responds to mechanical and chemical stimulation of the larynx and epiglottis (40). Taste buds distributed on the larynx are anatomically similar to those found throughout the oral cavity (44). However, because of their location, it is unlikely that they are involved in conscious taste sensation. Individual fibers of the SLN are broadly chemosensitive as are other gustatory nerves (8, 38). The most obvious difference between the sensitivities of chemosensitive fibers of the SLN and those in other gustatory nerves is in the response to water (8, 20, 38). Several studies have suggested that afferent neurons in the SLN responding to water are involved in important functions, such as diuresis, prandial drinking, and cardiovascular responses (18, 29, 37). These nerves convey sensory information to the caudal portion of the nucleus of the solitary tract (NTS) near the dorsal motor nucleus of the vagus (DMV), where vagal preganglionic neuron cell bodies are located (16, 19, 41). Because the vagal preganglionic neurons in the dorsomedial medulla are considered to regulate various gastric functions (12, 22), the SLN might affect gastric motility and/or relaxation via the vagus nerve.

The present study was undertaken to clarify the role of the superior laryngeal afferent signals elicited by water and saline on gastric motility. The effect of laryngeal stimulation by water and saline on gastric motility. The effect of laryngeal stimulation by water and saline on gastric contractility was investigated. Their neural mechanisms were also considered.

Materials and Methods

General methods. Animal care was in accordance with the guidelines of the Physiological Society of Japan. Male Sprague-Dawley rats (8–10 wk) were used. Each animal was fasted for 1 day to empty the stomach before the experiment was started. Each animal was anesthetized with an intraperitoneal injection of urethan-chloralose (urethan, 0.8 g/kg; chloralose, 65 mg/kg body wt). Subsequent anesthesia was administered as required through an unocclusive catheter constructed from the tip of an injection needle (23 gauge) and

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Silastic tubing (OD: 1.0 mm, ID: 0.5 mm) inserted into the right jugular vein. Each animal had a tracheal cannula made from polyethylene tubing (OD: 2.07 mm). The esophagus was ligated to prevent the test solutions from entering the stomach. Animals were placed in the supine position during the course of the experiments.

After an abdominal incision was made, an intragastric balloon, created from thin latex rubber and plastic tubing (OD: 1.7 mm), was introduced into the stomach from the greater curvature just proximal to the limiting ridge toward the antrum (Fig. 1). The balloon was secured by purse sutures around the gastric wall using 4-0 silk thread. At the end of each experiment, the abdomen was reopened and the position of the balloon was confirmed. Balloons were always positioned in the body of the stomach. Another tubing (OD: 2.0 mm) was also introduced into the fundus to drain the gastric juices and was ligated with the gastric wall around the tubing. The gastric balloon was inflated with warm water at 37°C to a volume of 3.0 ml/kg body wt. Before each experiment, it was confirmed that this volume of water did not induce pressure in the balloon outside the body. This volume was comparable to that used in a previous study (23).

After inflation of the gastric balloon, animals were left for at least 20 min until the gastric contraction was stabilized. The distal end of this tubing was connected with a strain-gauge pressure meter (NEC-Sanei, 6M82) to measure intragastric pressure.

To administer the test solutions, an incision was made between the thyroid and circular cartilage after intubation of the intragastric balloon. Polyethylene tubing (PE-50, Intramedic) connected to a syringe was inserted into the larynx. Test solutions (0.1 ml) were administered manually toward the laryngeal surface of the epiglottis for about 10 s at room temperature. Stimulation by water was confirmed by reflex swallowing and apnea. Ten minutes after each administration of water, the larynx was rinsed twice with 0.15 M saline.

The interval between the stimuli was at least 20 min. Preliminary confirmations showed that three injections of water induced similar magnitudes of inhibition of gastric motility in five animals tested. The mean ratios of the area under the contraction curve between 2 min before injection and 2 min after injection were 0.60 ± 0.06 (n = 5, at 1st injection), 0.58 ± 0.05 (n = 5, at 2nd injection), and 0.58 ± 0.04 (n = 5, at 3rd injection). All animals used in the present study underwent surgery for recording the intragastric pressure and for stimulation before the other surgeries, i.e., nerve section or spinal transection.

The differences of gastric responses to the administration of water and those to the administration of 0.15 M saline were evaluated in 12 animals. Among them, 10 animals were used in the other series of experiments to evaluate the effects of electrical stimulation of the SLNs and those of sectioning the SLNs.

All experiments were conducted at room temperature. The body temperature of the rats was maintained at 36–38°C with the use of a heating pad placed under the body and with an infrared lamp (Nihon Kohden, ATB-1100). At the end of each experiment, the animals were killed by an overdose of the anesthetic agent.

**Electrical stimulation.** In seven animals, inhibition of gastric motility induced by the electrical stimulation of the SLN was observed. The sternohyoid and omohyoid muscles were retracted. The SLNs were isolated from the surrounding tissue and bilaterally sectioned. The central cut end of the SLN was stimulated using a repeat electric pulse (20 Hz, 0.3 ms in duration, 0.3 mA in intensity) for 20 s using platinum bipolar electrodes.

**SLN sections.** To determine the ascending pathway, both SLNs were sectioned in six animals. Before the experiments, both SLNs were isolated from the surrounding tissue. After evaluation of the inhibition of contractile activity by the administration of water, sectioning of both SLNs was performed. Sectioning of the SLNs always induced a transient decrease in intragastric pressure that returned to the presectioning level after a few minutes. After stabilization of gastric contractility, water was again administered.

**Vagotomy and splanchnicotomy.** To determine the descending pathway, vagotomies were performed in five animals. Both vagi were carefully separated from the left and right carotid arteries. After evaluation of the inhibition of the contractile activity induced by the administration of water, sectioning of both vagi was performed. Both vagi were sectioned at the cervical level below where the SLN entered the vagal trunk, allowing their afferent signals into the central nervous system. More than 10 min after the sectioning, the response of gastric contractility to administration of water was measured again. Specific verification of successful nerve sectioning was not performed, because these nerves can be observed with a binocular microscope. Both vagotomies induced a gradual increase in intragastric tone and slowed the respiratory phase.

In addition, transection of the spinal cord was performed in seven animals. Before each experiment, the spinal cord was exposed by removing the spinal process and arches of T3 and T4. After evaluation of the inhibition of the contractile activity by the administration of water, the exposed spinal cord was sectioned between T3 and T4 using a microspatula. Sixty minutes after the transection, the response of gastric contractility to the administration of water was measured again. The portion of sectioning of each animal was confirmed visually at the end of each experiment.

**Concentration-response function.** To further examine the characteristics of the effects of different concentrations of

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**Fig. 1.** Schematic representation of recording sites for measuring intragastric pressure. The sites where the balloon was situated are indicated by shaded and hatched areas. The shaded area shows the balloon situated in the proximal stomach. The hatched area shows the balloon situated in the distal stomach. Note, in most experiments, the balloon was situated only in the distal stomach as shown by the hatched area.
saline on gastric motility, stimuli of water and eight concentrations of saline solutions were given in 15 animals. Different methods for the administration of the test solutions were used for these experiments. Both the chorda tympani and glossopharyngeal nerves innervate taste buds (13, 17). Therefore, each animal underwent bilateral sectioning of the chorda tympani and glossopharyngeal nerves before the surgery for recording intragastric pressure to prevent the test solutions activating these nerves. Both tympanic membranes were punctured, and the mallei were removed. Then, the chorda tympani nerves were sectioned. The glossopharyngeal nerves were exposed by partial removal of the hyoid process and carefully dissected from the surrounding tissue. Then, both glossopharyngeal nerves were sectioned between the external and internal carotid arteries. Polyethylene tubing (OD: 2.3 mm, ID: 1.5 mm) attached to a 15-gauge injection needle, was introduced into the trachea rostrally and fixed by 3-0 silk suture. Test solutions (1.0 ml) were water or 0.01, 0.03, 0.1, 0.15, 0.3, 0.5, or 1.0 M NaCl and were manually administered at room temperature. Injections of solutions were completed in 20 s. The responses to water were tested in all animals used for this series of experiments. After administration of water, each animal received more than two different concentrations of saline solutions. Saline solutions were delivered in ascending order of sodium concentration. Two minutes after each stimulus, 0.15 M NaCl (1.0 ml) was injected twice to wash out the test solutions. Motility returned to the preinjection level a few minutes after washing. The interval of stimulus was at least 20 min. Injected solutions were allowed to flow into the oral cavity. Other studies (8, 38) used a flow and suction system to apply and remove the stimuli to record afferent discharge from the SLN after sectioning both SLNs to avoid reflex swallowing. This method is adequate to remove the stimulant. However, because both SLNs had to be intact in the present research, reflex swallowing induced by the test solutions disrupted removal of the solutions in our preliminary experiments. Therefore, we did not use this flow and suction system.

Artificial ventilation and recordings from the proximal stomach. In this series of experiments, balloons were intubated in the proximal and the distal stomach in six animals. For the intubation of the two balloons in the stomach, large animals (450–500 g) were used. After intubation of the gastric balloon into the distal stomach, similar tubing with an attached balloon was introduced into the fundus from the minor curvature (Fig. 1). The gastric balloon situated in the proximal stomach was inflated with 0.5 ml water (37°C). The gastric balloon situated in the distal stomach was inflated with 0.8 ml water (37°C). A relatively small inflation in the distal stomach was achieved to prevent the two balloons influencing each other. Therefore, the contraction waves obtained were smaller than those in other experiments. The other procedure was similar to when intragastric pressure of the distal stomach was measured alone.

Six animals were neuromuscularly blocked using gallamine triethiodide (10 mg/kg iv, Sigma) and were ventilated with O2-enriched room air and positive end-expiratory pressure using a positive pressure ventilator (Shinano, SN-480–7) to exclude the influence of swallowing and respiration. The end-tidal CO2 pressure of expired gas was continuously measured with a fast-response CO2 analyzer (Cwe, Capstar-100) and was maintained within a range of 30–40 mmHg. Arterial blood pressure was measured through the polyethylene catheter (PE-50, Intramedic) inserted into the left carotid artery and was carefully separated from the vagi. During neuromuscular blockade, the depth of anesthesia was assessed by monitoring the stability of the arterial blood pressure, heart rate, and the cardiovascular responses to pinching the paws.

Data analysis. The contractile response was quantified by measuring the area under contractions (kPa × min) using the Flextrace program (Tree Star) on a personal computer. The area was measured at 1-min intervals, starting at the beginning of the injection of the test solution. The area before injection was also measured as a control. The mean values of the area 2 min before administration of the solution and 2 min after were used for comparison between pre- and post-treatment (motility area). For normalizing the data, the ratios of the area between 2 min before injection and 2 min after injection were also used for analyses (motility ratio). All numerical values are represented as means ± SE. Significant differences among the mean motility areas were evaluated using the paired t-test (P < 0.05 for significance). Significant differences between each mean motility ratio and 1.0 was also evaluated using the paired t-test (P < 0.05 for significance).

Results
Response of gastric motility to water and 0.15 M NaCl. The administration of 0.15 M NaCl did not induce any change in contractile activity or intragastric pressure. In contrast, the administration of water markedly decreased gastric contractility and the base level of the contraction (Fig. 2A). An inhibition of phasic contractions was always observed. However, the decreases in the basal level of the contraction curve shown in Fig. 2A were not always observed. Inhibition of motility (area under contraction) by water continued until 5 min after initiation of the injection (Fig. 2B). Significant differences (t = 3.74, P = 0.0033, n = 12 at 0–1 min; t = 4.24, P = 0.0014, n = 12 at 1–2 min; t = 3.43, P = 0.0056, n = 12 at 2–3 min; t = 2.30, P = 0.0417, n = 12 at 3–4 min; t = 4.09, P = 0.0018, n = 12 at 4–5 min) were observed compared with the preinjection motility. The difference between the motility after the administration of water and that of 0.15 M NaCl was significant at 1 min (t = 2.51, P = 0.0199, n = 12) and 2 min (t = 3.59, P = 0.0016, n = 12) after initiation of the injection. Because motility was markedly inhibited during the first 2 min immediately after the stimulation by water (Fig. 2B), the ratios of the area between 2 min before injection and 2 min after injection (motility ratio) are presented (Fig. 2C). The mean motility ratio induced by water (0.62 ± 0.05, n = 12) clearly shows significant inhibition (t = 8.07, P < 0.0001, n = 12), but that by 0.15 M NaCl (1.05 ± 0.07, n = 12) was not significant (t = 0.75, NS, n = 12). These findings suggested that the inhibition of motility was induced by the administration of water but not by other mechanical or thermal stimuli.

Effects of electrical stimulation of the SLN on gastric motility. Effects of electrical stimulation of the central cut end of each SLN were investigated. Electrical stimulation markedly inhibited contractility (Fig. 3). Electrical stimulation caused a relatively rapid decrease in gastric pressure, which gradually returned to pre-stimulation levels after cessation of the stimulation. The mean motility ratio when the left SLN was stimulated (0.64 ± 0.05, n = 7; t = 7.26, P = 0.0003) and
when the right SLN was stimulated (0.68 ± 0.04, n = 7; t = 8.69, P = 0.0001) was significantly reduced. No differences were noted between the inhibitory responses induced by stimulation of the left SLN and those of the right SLN.

Effects of sectioning the SLN on the inhibitory response of gastric motility. To determine the afferent pathway, gastric motility responses to the administration of water after bilateral sectioning of the SLN were investigated. No inhibition of motility induced by the administration of water in animals was observed after sectioning of both SLNs. In intact animals, the mean motility area for 2 min after the administration of water (2.60 ± 0.21 kPa × min, n = 6) was significantly smaller (t = 3.30, P = 0.0216, n = 6) than that before administration (5.36 ± 0.94 kPa × min, n = 6). After sectioning the SLN, however, no significant difference was observed (t = 1.40, P = 0.9570, n = 6) between the mean motility area before the administration of water (4.68 ± 0.96 kPa × min, n = 6) and that after the administration (4.35 ± 0.96 kPa × min, n = 6). The mean motility ratio observed before sectioning the SLNs was 0.54 ± 0.07 (n = 6) and was ~1.0 (0.93 ± 0.06, n = 6) after sectioning both SLNs (Fig. 4). A significant difference in the mean motility ratio was observed before sectioning the SLNs (t = 6.78, P = 0.0010, n = 6); however, this was not observed after sectioning the SLNs (t = 1.18, P = 0.2924, n = 6).

Effects of cervical vagotomy and spinal transection on the inhibition of gastric motility. To determine the descending pathway on the inhibition of gastric motility, experiments were performed on vagotomized or spinal sectioned animals (Fig. 5). Inhibitory response
of motility shown after administration of water was not observed after sectioning both vagi at the cervical level. In intact animals, the mean motility area for 2 min after administration of water (1.54 ± 0.15 kPa × min, n = 5) was significantly smaller (t = 6.64, P = 0.0012, n = 5) than that before administration (2.52 ± 0.22 kPa × min, n = 5). After bilateral cervical vagotomy, however, no significant difference was observed (t = 2.27, P = 0.7250, n = 5) between the mean motility area before administration of water (1.96 ± 0.23 kPa × min, n = 5) and that after administration (1.86 ± 0.25 kPa × min, n = 5). The mean motility ratio observed before sectioning the vagi (0.62 ± 0.04, n = 5) became ~1.0 (0.94 ± 0.03, n = 5) after sectioning of the vagi. A significant difference in the mean motility ratio was observed before sectioning the vagi (t = 8.98, P = 0.0420, n = 5); however, this was not observed after sectioning (t = 2.42, P = 0.7270, n = 5).

The inhibition of motility shown after administration of water was also observed after transection of the spinal cord between T3 and T4. In intact animals, the mean motility area for 2 min after administration of water (1.87 ± 0.36 kPa × min, n = 7) was significantly smaller (t = 4.67, P = 0.0034, n = 7) than that before (2.74 ± 0.50 kPa × min, n = 7). After spinal transection, the mean motility area for 2 min after administration of water (1.37 ± 0.17 kPa × min, n = 7) was significantly smaller (t = 4.14, P = 0.0061, n = 7) than that before the administration (2.17 ± 0.35 kPa × min, n = 7). The mean motility ratio observed before spinal transection (0.67 ± 0.05, n = 7) was similar to that after spinal transection (0.65 ± 0.03, n = 7). The significant difference in the mean motility ratio observed before transection (t = 6.61, P = 0.0006, n = 7) was also observed after transection (t = 10.70, P < 0.0001, n = 7) compared with 1.0.

Concentration-response function of the inhibition. To further examine the characteristics of the effect on gastric motility, water and different concentrations of saline were administered to the animals that underwent the bilateral sectioning of both chorda tympani and glossopharyngeal nerves (Fig. 6). The degree of inhibition to water and hypotonic saline was negatively correlated with the increase in sodium concentration. In contrast, the degree of inhibition to hypertonic saline was positively correlated with the increase in sodium concentration. Differences were significant in the mean motility ratio when water (t = 8.10, P < 0.0001, n = 15), 0.01 M NaCl (t = 3.20, P = 0.0109, n = 10), 0.03 M NaCl (t = 2.73, P = 0.0232, n = 10), 0.5 M NaCl (t = 2.78, P = 0.0388, n = 6), or 1.0 M NaCl (t = 14.04, P = 0.0001, n = 9) was administered.

Inhibitory responses observed in immobilized animals. The inhibition of gastric motility was observed in immobilized and artificially ventilated animals. Before artificial respiration, animals showed a marked inhibition of gastric motility induced by the administration of water. After administration of gallamine triethiodide, the magnitude of spontaneous phasic contractions was lowered. As shown in Fig. 7, in voluntary respiring animals, the mean motility area for 2 min after administration of water (0.75 ± 0.35 kPa × min, n = 6) was significantly smaller (t = 3.59, P = 0.0158, n = 6) than that before administration (1.25 ± 0.47 kPa × min, n = 6). After the administration of gallamine, the ampli-
The delivered solution may have spread to the pharynx. In stimuli were the epiglottis and the larynx. A part of the epiglottis in the present study. The targets of the solution delivered toward the laryngeal surface of the vago-vagal reflex arc to the stomach. Nerves. These observations indicate the presence of a water, as did bilateral sectioning of the cervical vagal SLNs abolished the inhibitory response induced by the administration of water. Bilateral sectioning of the and relaxation of the proximal stomach was induced by contractions in the distal stomach were suppressed, water into the larynx inhibited gastric motility. Phasic contractions in the distal stomach. The proximal stomach did not show phasic contraction, but the baseline fell, indicating a decrease in intragastric pressure after administration of water but not on administration of 0.15 M NaCl. Similar inhibition of intragastric pressure in the proximal stomach was observed in three other animals.

**DISCUSSION**

The present study showed that the administration of water into the larynx inhibited gastric motility. Phasic contractions in the distal stomach were suppressed, and relaxation of the proximal stomach was induced by the administration of water. Bilateral sectioning of the SLNs abolished the inhibitory response induced by water, as did bilateral sectioning of the cervical vagal nerves. These observations indicate the presence of a vago-vagal reflex arc to the stomach.

Stimulation was induced by the use of a 0.1-ml solution delivered toward the laryngeal surface of the epiglottis in the present study. The targets of the stimuli were the epiglottis and the larynx. A part of the delivered solution may have spread to the pharynx. In the stimulus condition, the inhibitory responses observed after administration of water were eliminated by sectioning both SLNs. Therefore, test solutions were administered, in the present study, to the area innervated by the SLN. The findings obtained from bolus injection of water into the oral cavity of animals that had received bilateral sectioning of the chorda tympani and glossofaryngeal nerves also indicated that the SLNs have a major role in the inhibition of the stomach induced by the administration of water.

It was shown that NaCl solution was effective in increasing the firing frequency of SLN fibers at concentrations below isotonic saline. The firing frequency increased with decreasing NaCl concentration (34, 35). Similar findings were seen in the response of gastric motility in the present study. The degree of inhibition of gastric motility increased with decreasing sodium concentrations of the test solution below the isotonic level. In the present study, significant inhibitions of gastric motility were also observed after the administration of 0.5 and 1.0 M NaCl. The degree of inhibition increased with increasing concentrations of NaCl in the hypertonic solution. Smith and Hanamori (38) reported that hamster SLN fibers responded to relatively high concentrations of NaCl as well as to hypotonic saline. The response of gastric motility shown in the present study presumably reflects the response characteristics of primary afferent neurons. Water response is not regarded as a specific response to water per se. Response to water was attributed to the sensitivity of the receptor membrane to the outward flow of Cl⁻ in rabbits and dogs (6, 34). In mice, the results of responses of SLN fibers to either electrolytes or nonelectrolytes suggest that water response is induced by the change in osmolality of the solutions (36). The response mechanism appears to be different between animal species. In addition, several studies have dem-
would prevent the intragastric pressure from becoming high during and after food ingestion. Cannon and Lieb (9) demonstrated relaxation of the canine stomach during swallowing, which is the so-called “receptive relaxation.” Another effective stimulus is esophageal distension (2, 43). Because these reflexes inhibit intragastric pressure before ingested food or liquid reaches the stomach, they are considered anticipatory responses. These relaxation responses, abolished by vagotomy but not by splanchnicotomy, are similar to those observed in the present study. Water-responsive afferents in the SLN should elicit similar reflexes such as receptive relaxation. Because water intake is an important signal of the initiation of eating, the reflex arc found in the present study might have a role in facilitating gastric relaxation to accommodate the intake of food and liquid similar to receptive relaxation.

Taste buds are especially concentrated on the laryngeal surface of the epiglottis (5, 7, 28). Because taste buds in this region appear to be ideally situated for protecting the airway during accidental aspiration of food or fluid, inhibitory responses of the stomach observed in the present study might be initiated only when fluid is going to invade the larynx. However, aryepiglottal folds and the upper esophagus also contain taste buds in addition to the laryngeal surface of the epiglottis (5, 28, 42). The epithelium of the rostral portion of the pharyngeal mucosa is innervated by the superior laryngeal nerve (25). Taste buds situated in the pharynx or esophagus would be stimulated during normal food or fluid intake. Further investigation using microstimulation of restricted regions might provide better understanding of the physiological functions of the reflex observed in the present study.

Perspectives

Receptive relaxation, which was proposed by Cannon and Lieb (9), is one of the reflexes caused by the extrinsic nervous system during swallowing and facilitates the reservoir function of the stomach. It was suggested that this inhibitory response of the stomach is caused by mechanical stimulation of the pharynx and esophagus by ingesta (2). Water fibers in the SLN might be a novel neuronal source for the receptive relaxation, although more precise experiments are demanded to clarify this. To provide an accurate view of the participation of SLN water fibers in the receptive relaxation, the response of the stomach to the liquid stimulation of the restricted area, which is usually stimulated by food and fluid intake, should be confirmed. Furthermore, the study of central neurons might provide a better understanding of the control mechanisms of gastric motility. Recently, the central neural mechanisms of gastric relaxation and inhibition of gastric motility evoked by esophageal distension was clarified electrophysiologically (31). Esophageal afferents appear to provoke inhibition of the stomach by activating a vagal inhibitory pathway and inhibiting a vagal excitatory pathway. Finally, it is preferable to clarify how oral signals combine with the other sensations that induce the inhibition of the stomach in the oral cavity.
central nervous system and induced the conformable inhibitions of the stomach that facilitate the reservoir functions of stomach.

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