Restraint stress augments postprandial gastric contractions but impairs antropyloric coordination in conscious rats

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Nakade, Yukiomi, Daisuke Tsuchida, Hiroyuki Fukuda, Masahiro Iwa, Theodore N. Pappas, and Toku Takahashi. Restraint stress augments postprandial gastric contractions but impairs antropyloric coordination in conscious rats. Am J Physiol Regul Integr Comp Physiol 290: R616–R624, 2006. First published October 27, 2005; doi:10.1152/ajpregu.00161.2005.—Central corticotropin-releasing factor (CRF) plays an important role in mediating restraint stress-induced delayed gastric emptying. However, it is unclear how restraint stress modulates gastric motor activity or delays gastric emptying. Inasmuch as solid gastric emptying is regulated by coordination of antropyloric, we hypothesized that restraint stress impairs antropyloric coordination, resulting in delayed solid gastric emptying in conscious rats. Two strain gauge transducers were sutured onto the serosal surface of the antrum and pylorus, and postprandial gastric motility was monitored before, during, and after restraint stress. Antropyloric coordination, defined as a propagated single contraction from the antrum to the pylorus within 10 s, was followed by ≥20 s of quiescence. Restraint stress enhanced postprandial gastric motility in the antrum and pylorus to 140 ± 9% and 134 ± 9% of basal, respectively (n = 6). The number of episodes of antropyloric coordination before restraint stress, 2.4 ± 0.4/10 min, was significantly reduced to 0.6 ± 0.3/10 min by restraint stress. Intracisternal injection of the CRF type 2 receptor antagonist astressin 2B (60 μg) or guanethidine partially restored restraint stress-induced impairment of antropyloric coordination (1.6 ± 0.3/10 min, n = 6). The restraint stress-induced augmentation of antral and pyloric contractions was increased by astressin 2B and guanethidine but abolished by atropine, hexamethonium, and vagotomy. Restraint stress enhanced postprandial gastric motility via a vagal cholinergic pathway. Restraint stress-induced delay of solid gastric emptying is due to impairment of antropyloric coordination. Restraint stress-induced impairment of antropyloric coordination might be mediated via a central CRF pathway.

corticotropin-releasing factor; restraint stress

A GROWING BODY OF EVIDENCE suggests a role for stress in gastrointestinal (GI) physiological and pathophysiological functions, such as gastric emptying, gastric ulcerations, gastric secretion, and intestinal transit (1, 18, 19, 23, 28, 34, 41).

Several brain neuropeptides are known to act as stress-related neurotransmitters that modify GI motor functions (8, 29, 33, 37, 39). Corticotropin-releasing factor (CRF), one of the stress-related neuropeptides, is known to act in the brain to influence GI functions. Exogenous administration of CRF into the central nervous system (CNS) delays gastric emptying and inhibits acid secretion, whereas it stimulates colonic transit through the autonomic nervous system (5, 22, 35). Restraint stress augments CRF mRNA in the amygdala and paraventricular nucleus (PVN) (13, 16), resulting in alteration of GI motor activities. Restraint stress-induced delayed gastric emptying is abolished by central administration of CRF antagonist (23), suggesting that endogenous CRF has an important role in mediating stress-induced delay of gastric emptying. We recently demonstrated that restraint stress delays solid gastric emptying via a central CRF and peripheral sympathetic neuron in rats (30).

Although CRF and restraint stress attenuate gastric emptying (22), little is known about the postprandial gastric motility mediated by restraint stress. It remains unclear how restraint stress modifies gastric motility to delay gastric emptying.

Liquid meal emptying primarily reflects the activity of the fundus (27), whereas gastric emptying of a solid meal is regulated by coordination of the antrum, pylorus, and duodenum (3, 11, 15, 27).

The postprandial gastric motor pattern in response to a solid food involves a grinding and an emptying period in humans (3), dogs (40), and rats (14, 15). Immediately after a feeding, a grinding period, i.e., significant contraction, is observed in the antrum and pylorus. In contrast, the contractile pattern of the antrum is significantly changed from 40 min after a feeding in rats. The coordinated motility between the antrum and pylorus, which seems to be effective in propelling the gastric contents to the duodenum, is observed in this emptying period. Thus coordinated postprandial antropyloric contractions play an important role in the gastric emptying of solids in rats (14, 15).

We previously showed that hyperglycemia impairs antropyloric coordination and delays solid gastric emptying in conscious rats (15). We also demonstrated that injection of neuropeptide Y (NPY) into the CNS increased gastric motility but impaired antropyloric coordination, resulting in delayed solid gastric emptying in conscious rats (14). These results led us to speculate that restraint stress could impair antropyloric coordination and delay solid gastric emptying.

The present study was designed to examine the restraint stress-induced alteration of gastric motility from the viewpoint of postprandial antropyloric coordination. We assessed the mechanisms of restraint stress-mediated antropyloric coordination. In addition, we used a CRF type 2 receptor antagonist, astressin 2B, to examine the role of endogenous CRF in restraint stress-induced alteration of gastric motility.

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Each rat was subjected to the restraint stress loading at and gastric motility was recorded for 90 min. After the animal was able to move its limbs and head but not its trunk (2, 23), a harness consisting of a bandage fabricated from packaging tape. The rats were lightly anesthetized (3% isoflurane) and gastric motor activities were recorded for 90 min before the restraint stress loading. The dose of these antagonists was chosen on the basis of our previous studies (30, 38). Saline (0.3 ml)-injected rats served as controls.

To study the role of the vagus nerve in stress-induced alteration of gastric motility, rats underwent truncal vagotomy: the vagal trunks were cut around the abdominal esophagus at the time the transducers were sutured, as previously reported (32). Sham-operated rats served as controls. The feeding behavior was not affected by truncal vagotomy.

To examine the role of endogenous CRF in restraint stress-induced modification of gastric motility, the CRF type 2 receptor antagonist astressin 2B (60 μg/rat) or saline was injected intracerebrally (i.c) 10 min before the restraint stress loading under light (3%) isoflurane anesthesia. It has been shown that astressin 2B (60 μg ic) had a tendency to prevent the inhibitory effect of urocortin on fasting gastric motility and that astressin 2B (100 μg ic) had a statistically significant effect on the urocortin-induced inhibition of gastric motility in anesthetized rats (4). As shown in Fig. 8, CRF (0.5 μg ic) attenuated postprandial gastric motility in conscious rats. In our preliminary study, 60 μg of astressin 2B almost completely abolished CRF (0.5 μg ic)-induced inhibition of gastric motility.

To investigate whether the sympathetic pathway is involved in endogenous CRF-induced mediation of gastric motility, we injected astressin 2B (60 μg ic) into guanethidine-treated rats.

Recording of antral and pyloric motility in response to intracisternal CRF. After 12 h of fasting, the rats were fasted for 12 h just before monitoring of gastric motility. The wires from the transducers were run under the skin to an opening made in the back of the neck using a rat protective system (Star Medical, Tokyo, Japan). The presence of cerebrospinal fluid in the syringe on aspiration before injection verified the accuracy of needle placement into the cisterna magna, as previously described (30, 31).

Experimental Design

Recording of antral and pyloric motility in restraint stress. After 12 h of fasting, the rats were anesthetized with pentobarbital sodium (45 mg/kg ip). An abdominal incision was performed, and a 10-μl microsyringe (Hamilton, Reno, NV). The wires to the transducers were run under the skin to an opening made in the back of the neck using a rat protective system (Star Medical, Tokyo, Japan). After the transducers were sutured, the abdominal wall was closed.

At 1 wk after the operation, the rats were fasted for 12 h just before monitoring of gastric motility. The wires from the transducers were connected to the recording system (Power-Lab model 8SP; ADInstruments, Colorado Springs, CO). The rats were fed 3 g of rat chow, and gastric motor activities were recorded for 90 min before the restraint stress loading. The rats were lightly anesthetized (3% isoflurane) and placed on a wooden plate with their trunks wrapped in a confining harness consisting of a bandage fabricated from packaging tape. The animal was able to move its limbs and head but not its trunk (2, 23).

Gastric motility was measured during restraint stress for 90 min. After 90 min of restraint stress loading, the rats were immediately released, and gastric motility was recorded for 90 min.

The area under the curve was calculated using a computer-assisted system (MacLab, ADInstruments) and expressed as a motility index. Each rat was subjected to the restraint stress loading at 4-day intervals.

Ascending contraction and descending relaxation play a crucial role in promoting effective peristalsis in the GI tract (9). When contractions occur simultaneously in adjacent parts of the GI tract, the content between those parts would not be expelled effectively. Thus the temporal relation of contraction between adjacent parts of the gut needs to be analyzed. As previously described (14, 15), antropyloric coordination was defined as a single contraction at the antrum that propagated aborally in the pylorus within 10 s and was followed by ≥20 s of quiescence. Individual contractions were defined as >2-g changes of >1-s duration. To investigate how restraint stress alters the motility of the antrum and pylorus, we evaluated the number of episodes of antropyloric coordination during restraint stress, as previously described (14).

Pharmacological and surgical approaches. To investigate whether restraint stress-induced alteration of gastric motility is mediated via a noradrenergic and cholinergic pathway, guanethidine (5.0 mg/kg ip) and atropen sulfate (50 μg/kg ip) were injected 30 min before the restraint stress loading. To evaluate the involvement of nicotinic receptors in the mediation of restraint stress, hexamethonium (20 mg/kg ip) was injected 30 min before the restraint stress loading. The dose of these antagonists was chosen on the basis of our previous studies (30, 38). Saline (0.3 ml)-injected rats served as controls.

At 90 min after feeding, the contraction maxima of the antrum occurred in most cases 3–6 s before the contraction maxima of the pylorus (Fig. 1), as previously reported (14, 15). The number of episodes of antropyloric coordination was 2.4 ± 0.4/10 min before the restraint stress (Table 1).

Restraint stress enhanced antral and pyloric motility compared with the prerestraint state. Restraint stress increased...
the peak amplitude of the antrum and pylorus to 137 ± 11% and 125 ± 12% of basal (n = 6), respectively. Restraint stress increased the frequency of antral and pyloric contractions from 2.2 ± 0.5 to 3.8 ± 0.6 and from 2.8 ± 0.5 to 4.1 ± 0.7/min, respectively. Thus, the motility index of the antrum and pylorus was also increased from 23,724 ± 2,467 to 33,450 ± 2,763 and from 10,524 ± 927 to 14,102 ± 1,242 g/s for 30 min, respectively (Fig. 2). During the restraint stress loading, the time lag between the antral and pyloric contractions was not observed. The contraction maxima of the antrum and pylorus occurred simultaneously (Fig. 1). The number of episodes of antropyloric coordination was significantly reduced to 0.6 ± 0.3/10 min during the restraint stress (Table 1).

Restraint stress-induced augmentation of the antral and pyloric contractions returned to the basal levels within 3–5 min after termination of stress loading (Fig. 1). The time lag between the antral and pyloric contractions was also observed after termination of restraint stress loading (Fig. 1).

Table 1. Episodes on antropyloric coordination mediated by restraint stress or central CRF

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<tr>
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<th>Episodes of Antropyloric Coordination</th>
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<tr>
<td>Control + saline ic</td>
<td>2.4 ± 0.4</td>
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<tr>
<td>Control + atressin 2B (60 μg ic)</td>
<td>2.5 ± 0.6</td>
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<td>Control + guanethidine (5 mg/kg ip)</td>
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<td>Control + CRF (0.5 μg ic)</td>
<td>0.9 ± 0.3†</td>
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<td>Restraint stress + saline (ic)</td>
<td>0.6 ± 0.3†</td>
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<tr>
<td>Restraint stress + atressin 2B (60 μg ic)</td>
<td>1.6 ± 0.3*</td>
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<td>Restraint stress + guanethidine (5 mg/kg ip)</td>
<td>1.5 ± 0.4*</td>
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<td>Restraint stress + guanethidine (5 mg/kg ip) + atressin 2B (60 μg ic)</td>
<td>1.4 ± 0.5*</td>
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Values are means ± SE expressed as number per 10 min (n = 6). CRF, corticotropin-releasing factor. Number of episodes of antropyloric coordination was significantly reduced by restraint stress. Intracisternal (ic) atressin 2B attenuated impairment of antropyloric coordination. *P < 0.05; †P < 0.01 vs control + saline.
Effect of Guanethidine, Atropine, Hexamethonium, and Truncal Vagotomy on Restraint Stress-Induced Alteration of Antral and Pyloric Motility

Guanethidine enhanced restraint stress-induced augmentation of antral and pyloric contractions (Fig. 3). During the restraint stress, guanethidine increased the peak amplitude of the antrum and pylorus to 145/11006 21% and 146/11006 10% of basal (n = 6), respectively. During the restraint stress, guanethidine increased the frequency of antral and pyloric contractions from 2.6/11006 0.5 to 5.0/11006 0.2 and from 3.3/11006 0.8 to 5.1/11006 0.1/min, respectively. The motility index of the antrum was also increased from 25,500/11006 2,467 to 37,211/11006 2,342 g/s for 30 min during stress loading. The motility index of the pylorus tended to be enhanced by guanethidine, although the difference was not statistically significant (Fig. 2). The number of episodes of antropyloric coordination was partially restored to 1.5/11006 0.4/10 min by guanethidine during stress loading (Table 1).

Fig. 3. Effects of saline (A), guanethidine (Gua, B), atropine (Atr, C), and hexamethonium (Hex, D) on restraint stress-induced augmentation of antral and pyloric motility in conscious rats. Guanethidine enhanced restraint stress-induced augmentation of antral and pyloric motility. Atropine and hexamethonium attenuated antral and pyloric motility induced by restraint stress.

Fig. 4. A: effects of truncal vagotomy on restraint stress-induced augmentation of antral and pyloric motility in conscious rats. Truncal vagotomy reduced postprandial antral (B) and pyloric (C) motility indexes. Restraint stress did not augment antral and pyloric motility in vagotomized rats. Values are means ± SE (n = 4–5). *P < 0.05 compared with control. **P < 0.01 compared with respective groups.

Fig. 5. Effect of intracisternal (ic) injection of astressin 2B on restraint stress-induced augmentation of antral (A) and pyloric (B) motility. Restraint stress-induced augmentation of antral and pyloric motility was further enhanced by astressin 2B (60 μg ic). Guanethidine also increased restraint stress-induced augmentation of antral motility. Intracisternal astressin 2B did not further increase antral and pyloric motility in guanethidine-treated rats. Guanethidine also tended to increase restraint stress-induced augmentation of pyloric motility, although this was not statistically significant (NS). Values are means ± SE (n = 4–6). *P < 0.05; **P < 0.01 compared with saline. #P < 0.05 compared with saline/restraint stress.

Effect of Guanethidine, Atropine, Hexamethonium, and Truncal Vagotomy on Restraint Stress-Induced Alteration of Antral and Pyloric Motility

Guanethidine enhanced restraint stress-induced augmentation of antral and pyloric contractions (Fig. 3B). During the restraint stress, guanethidine increased the peak amplitude of the antrum and pylorus to 145 ± 21% and 146 ± 10% of basal (n = 6), respectively. During the restraint stress, guanethidine increased the frequency of antral and pyloric contractions from 2.6 ± 0.5 to 5.0 ± 0.2 and from 3.3 ± 0.8 to 5.1 ± 0.1/min, respectively. The motility index of the antrum was also increased from 25,500 ± 2,467 to 37,211 ± 2,342 g/s for 30 min (Fig. 2A) during stress loading. The motility index of the pylorus tended to be enhanced by guanethidine, although the difference was not statistically significant (Fig. 2B). The number of episodes of antropyloric coordination was partially restored to 1.5 ± 0.4/10 min by guanethidine during stress loading (Table 1).
Pretreatment with atropine and hexamethonium reduced postprandial antral and pyloric contractions and abolished restraint stress-induced augmentation of antral and pyloric contractions (Fig. 3, C and D). During the restraint stress loading, no significant changes were observed in the antrum and pylorus in atropine- and hexamethonium-treated rats (Fig. 2, A and B).

Postprandial antral and pyloric contractions were attenuated in vagotomized compared with sham-operated rats (Fig. 4, B and C). Restraint stress did not affect postprandial antral and pyloric contractions in vagotomized rats (Fig. 4).

**Effect of Intracisternal Injection of Astressin 2B on Restraint Stress-Induced Augmentation of Antral and Pyloric Motility**

Intracisternal astressin 2B (60 μg) did not affect postprandial antral and pyloric contractions (Fig. 5, A and B). Intracisternal astressin 2B significantly enhanced restraint stress-induced augmentation of antral and pyloric contractions (Fig. 6) and increased the motility index of antral and pyloric contractions stimulated by restraint stress (Fig. 5, A and B).

During restraint stress loading, there was an obvious time lag between the antral and pyloric contractions in astressin 2B-treated rats (Fig. 6). The number of episodes of antropyloric coordination was partially restored to 1.6 ± 0.3/10 min during the restraint stress loading (Table 1).

In guanethidine-treated rats, intracisternal astressin 2B did not further increase the motor activities stimulated by restraint stress (Fig. 5, A and B). The time lag between the antral and pyloric contractions was also observed in animals treated with astressin 2B + guanethidine (Fig. 7). The number of episodes of antropyloric coordination was also restored to 1.4 ± 0.5/10 min during the restraint stress loading (Table 1).
Effect of Intracisternal Injection of CRF on Postprandial Antral and Pyloric Contractions

Intracisternal injection of saline or CRF (0.1 μg; B–E) on antral motility. Postprandial antral motility was monitored before and after intracisternal injection of CRF and saline. CRF (0.1 μg ic) did not modify postprandial antral motility. At 0.25 μg, CRF dose dependently attenuated postprandial antral motility. Inhibitory effects of CRF were sustained for up to 30 min.

Effect of Intracisternal Injection of CRF on Postprandial Antral and Pyloric Contractions

Intracisternal injection of saline or CRF (0.1 μg) did not affect postprandial antral contractions (Figs. 8, A and B, and 9). In contrast, intracisternal injection of CRF (0.25, 0.5, and 1.0 μg) inhibited postprandial antral contractions in a dose-dependent manner (Figs. 8, C–E, and 9). The inhibitory effects were observed for 30 min after CRF (1.0 μg) injection (Fig. 8E). The inhibitory effect of CRF on the pyloric contractions was also observed (data not shown).

Intracisternal injection of CRF (0.5 μg) increased the number of simultaneous contractions of the antrum and pylorus (Fig. 10) and significantly attenuated the number of episodes of antropyloric coordination for up to 30 min after the peptide injection (Table 1).

Pretreatment with guanethidine almost completely abolished the inhibitory effects of CRF on postprandial antral and pyloric contractions (Fig. 11).

DISCUSSION

A number of investigators reported that restraint stress delays gastric emptying of a liquid (4, 23, 26) and solid food (30). However, little is known about whether restraint stress modifies gastric motility. Although it has been reported that restraint stress does not affect gastric motility in a fasting state (42), it remains unclear whether restraint stress alters postprandial gastric motility.

Our present study demonstrated that restraint stress significantly augmented gastric contractions of the antrum and pylorus in response to solid food ingestion. We also showed that the augmented gastric motility was restored to basal levels immediately after termination of the restraint stress loading. Because atropine, hexamethonium, and vagotomy blocked restraint stress-induced augmentation of gastric motility, the vagal-cholinergic pathway might be involved in this event.

Previous reports indicate that the CNS produces several neuropeptides in response to restraint stress (16, 20). Cold-restraint stress increases thyrotropin-releasing hormone (TRH) in the brain stem, which stimulates gastric motility and liquid emptying (17, 36). It remains unknown whether endogenous TRH is involved in mediating restraint stress-induced augmentation of postprandial gastric contractions. Some other unknown factors might be involved in the mechanism of restraint stress-induced augmentation of gastric motility.

It is generally accepted that solid gastric emptying is regulated by coordination of the antrum, pylorus, and duodenum (11, 27). In particular, coordination between the antrum and pylorus is an important factor in the emptying of solid foods (3, 6, 14, 21, 27).

In pre- and postrestraint stress, a time lag was observed between the antral and pyloric contractions; most often, the contraction maxima of the antrum occurred 3–6 s before the contraction maxima of the pylorus.

During restraint stress loading, the time lag between the antral and pyloric contractions was not observed frequently and the contraction maxima of the antrum and pylorus occurred simultaneously. The number of episodes of antropyloric coordination was significantly reduced during the restraint stress. Simultaneous contractions of the antrum and pylorus are considered to impair the effective emptying process and delay gastric emptying of a solid food.

It remains unclear whether the augmented gastric contractions are well correlated with the acceleration of gastric emp-
tying. Pentagastrin significantly augments gastric antral contractions, but it does not accelerate gastric emptying (12, 25). Although restraint stress-induced augmentation of gastric contractions was further increased by guanethidine, gastric emptying was not fully restored (30). Thus augmented gastric motility does not necessarily improve delayed gastric emptying. The coordinated motor pattern between the antrum and pylorus seems to be more important than augmented gastric contractions in mediating gastric emptying of solid food.

CRF is known to act in the brain as a neurotransmitter to influence GI function through the autonomic nervous system (5, 7, 22, 30). The present study revealed that intracisternal injection of CRF dose dependently attenuated postprandial gastric motility of the antrum and pylorus for up to 30 min. Furthermore, we demonstrated that intracisternal injection of CRF (0.5 μg) produced simultaneous contractions, resulting in impairment of antropyloric coordination. These results indicated that administration of CRF into the CNS not only attenuates gastric motility but also impairs antropyloric coordination. It is conceivable that endogenous CRF may be involved in the impairment of antropyloric coordination in stress loading.

The pathway through which central CRF attenuates gastric motility remains to be elucidated. A previous report demonstrated that microinjection of CRF at the dorsal vagal complex attenuates bethanechol-stimulated gastric contractions in anesthetized rats (24). The inhibitory effect of CRF is mediated via activation of inhibitory nonadrenergic noncholinergic vagal input to the stomach (24). In contrast, our present study demonstrated that guanethidine abolished inhibition of postprandial gastric motility induced by intracisternal injection of CRF in conscious rats, suggesting involvement of the sympathetic pathway.

We also showed that intracisternal injection of the CRF type 2 receptor antagonist astressin 2B (60 μg) enhanced restraint stress-induced augmentation of gastric motility. The pathophysiological significance of the inhibitory effect of central CRF on postprandial gastric motility remains unclear. It has been reported that hypothalamic microinusions of CRF increase bicarbonate content, decrease gastric acid content, and protect against cold restraint-induced gastric mucosal damage (10). It is conceivable that central CRF may act against stress-induced augmentation of gastric motility. This may provide new information about the role of endogenous CRF in stress-induced alteration of postprandial gastric motility.

Restraint stress-induced impairment of antropyloric coordination was partially restored by treatment with guanethidine. Intracisternal injection of astressin 2B did not produce any further changes in stress-induced impaired coordination in guanethidine-treated rats. These results suggest that the inhibitory effect of endogenous CRF on gastric motility is mediated via sympathetic pathways.

Fig. 10. Effects of intracisternal injection of 0.5 μg of CRF on antropyloric coordination. Intracisternal injection of CRF attenuated gastric contractions in antrum and pylorus. Before CRF injection, time lag was most frequently observed between each contraction of antrum and pylorus. Contraction maxima of antrum and pylorus were observed almost simultaneously for up to 30 min after CRF injection. Arrows, simultaneous contraction of antrum and pylorus. Vertical lines, episodes of antropyloric coordination.

Fig. 11. Effects of saline (A) and guanethidine (B) on intracisternal CRF-induced inhibition of antral (C) and pyloric (D) contraction. Guanethidine abolished CRF-induced inhibition of gastric motility. Values are means ± SE (n = 4–5). **P < 0.01 compared saline.
Although intracisternal injection of astressin 2B did not fully restore impaired antropyloric coordination induced by restraint stress (Table 1), our previous study showed that restraint stress-induced delay of gastric emptying was completely restored by astressin 2B (30). The reason for this difference remains to be elucidated. One possibility may be the difference in the stress models. In our previous study, partial body-tubing stress was applied (30); in the present study, partial body-wrapping stress was used. In the wrapping stress model, stress-induced delay of solid gastric emptying might not be completely inhibited by intracisternal injection of astressin 2B.

We cannot exclude the possibility that other stress-related neuronal factors, in addition to CRF, might be involved in the impaired antropyloric coordination induced by partial body-wrapping stress. Several central coding neuropeptides, such as NPY or TRH, seem to be involved in the mechanism of stress-induced alteration of gastric motor functions (14, 17, 20). We previously showed that centrally administered NPY impairs antropyloric coordination (14). It is of interest to examine whether stress-induced impairment of antropyloric coordination is mediated by these neuropeptides.

In summary, restraint stress augmented postprandial gastric motility but impaired antropyloric coordination in conscious rats. The augmented postprandial gastric motility is mediated via vagal cholinergic pathways. Central CRF in response to restraint stress may play an important role in impairment of postprandial antropyloric coordination. Delayed gastric emptying by restraint stress might be due to impairment of antropyloric coordination. These results provide new evidence that restraint stress modifies gastric motor function.

REFERENCES


