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Effect of prepyloric gastric transection and anastomosis on sphincter of Oddi cyclic motility in conscious dogs

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Purpose. We previously reported significant changes in sphincter of Oddi cyclic motility after proximal duodenal transection and anastomosis. However, the role of intrinsic myoneural continuity between the antrum and duodenum in this respect is not understood. The aim of this study was to elucidate the effects of prepyloric gastric transection on sphincter of Oddi motility in animals in the conscious state. ***Methods.*** Pressures in the bile duct, duodenum, stomach, and sphincter of Oddi and their response to an injection of cholecystokinin-octapeptide were measured in four conscious dogs, with a duodenal cannula, before and after gastric transection and anastomosis 1.5 cm proximal to the pylorus. ***Results.*** Gastric transection did not affect the initiation and propagation of the gastroduodenal migration motor complex. Biliary pressure (5.7 ± 0.15 to 5.5 ± 0.2 mmHg; $P = 0.91$), sphincter of Oddi basal pressure (10.6 ± 0.3 to 10.7 ± 0.2 mmHg; $P = 0.97$), and amplitude (26.0 ± 1.2 to 32.9 ± 1.7 mmHg; $P = 0.304$) did not change after gastric transection. Biliary pressure decreased from phase II to phase III of the duodenal migrating motor complex. Cholecystokinin-octapeptide inhibited sphincter of Oddi phasic waves before and after gastric transection. ***Conclusions.*** Intrinsic myoneural transection at the prepyloric region does not influence sphincter of Oddi cyclic motility. Preservation of pyloroduodenal myoneural continuity in pylorus-preserving gastrectomy would be beneficial to maintain normal sphincter of Oddi motility.

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Introduction

Gallstone formation is a well-known long-term complication after gastric operations.[1–4] Several factors have been suggested to contribute to gallstone formation. Truncal vagotomy reduces gallbladder tone and impairs its contractile activity,[5,6] possibly resulting in bile stasis and retrograde infection through the sphincter of Oddi (SO).[7] On the other hand, the presence of the stomach per se is also likely to affect biliary and duodenal motility. Mechanical or electrical stimulation of the stomach influenced the SO tone, and gastrectomy changed bile flow dynamics.[8,9] Ura et al.[10] showed that antrectomy without vagotomy changed the cyclic motility of the gallbladder. Thus, gastric motility and/or gastroduodenal continuity may be a significant factor in the control of biliary motility.

In most mammalian species during fasting, the lower esophageal sphincter, stomach, and small intestine exhibit a characteristic cyclic pattern of motor and myoelectric activity, called the migrating motor complex (MMC), which consists of four distinct phases, from phase I to phase IV.[11,12] The SO plays an important role in bile delivery to the duodenum and in the prevention of duodenobiliary reflux.[13] It is well known that the SO exhibits phasic contractions superimposed on basal pressure.[14,15] During fasting, the SO phasic contractions and basal pressure exhibit a cyclic change, in coordination with the gastrointestinal MMC.[16,17] A marked difference between the MMC and SO cyclic motility is that the SO continues to contract even during the quiescence of phase I of the MMC. On the other hand, the SO and duodenum show concurrent strong contractile activity during phase III of the MMC.

Extrinsic nerves and gastrointestinal hormones have been reported to affect SO motility. Truncal vagotomy increased the amplitude and frequency of SO phasic activity in anesthetized prairie dogs.[18] Cholecystokinin (CCK) has varying effects on the SO, depending on species.[14,19–22] In dogs and humans, CCK normally inhibits SO activity.[21,22] However, the effect of CCK on the SO becomes excitatory after distal gastrectomy in humans.[23,24] Moreover, it was also reported that translocation of the papilla to the jejunum[25] changed SO motility. Our recent study also showed that duodenal transection and anastomosis 1 cm distal to the pylorus reduced the SO basal pressure and reversed the SO response to CCK, from inhibition to stimulation and from reduction of the basal pressure to elevation.[26] These observations suggest that intrinsic myoneural continuity between the SO and duodenum and/or stomach may play a role in controlling SO motility. However, it has not been clarified whether gastric myoneural continuity plays a principal role in this fine control of SO basal pressure and the SO response to CCK. Accordingly, we carried out this study, aiming to further elucidate the effects of prepyloric gastric transection (GTX) on: (1) interdigestive cyclic motility of the SO, duodenum, and stomach in conscious dogs, and (2) the SO response to CCK-octapeptide (CCK-OP). We chose a canine model because the relevant anatomy and physiological functions are similar to those in humans.[27,28]

Methods

Preparation of animals

This experimental protocol was reviewed by the Committee of Ethics on Animal Experiments at Kyushu University Faculty of Medicine, and the study was carried out according to the Guidelines for Animal Experiments at Kyushu University Faculty of Medicine.

A chronic model for obtaining long-term recording of SO motility was prepared in four male mongrel dogs (weight, 13–20 kg). They underwent celiotomy under intravenous anesthesia with 25 mg/kg of pentobarbital sodium. A modified Thomas cannula was implanted into the duodenum opposite the major papilla, as described previously.[16] The cannula was closed with a rubber plug to allow the dog to take normal food between the experiments. The dogs were allowed to recover for at least 14 days before recordings were done.

After several baseline recordings with and without the administration of CCK-OP, as described later, each dog was reoperated through the previous incision, avoiding any injury to the lesser and greater curvatures. Vagal innervation to the stomach was carefully preserved. Gastric transection and anastomosis were performed 1.5 cm proximal to the pylorus, with two-layer 4-0 polyglycolic acid sutures. The dog was given total parental nutrition for 4 days after GTX. SO manometry was performed at least 14 days after GTX. All the experiments ended within 4 weeks after GTX, because regeneration of intrinsic nerves is known to take place after 1 month.[29]

Manometric system

A double-lumen catheter with a short end-to-side guidewire channel at its tip (Star Medical, Tokyo, Japan) was used for retrograde SO manometry. The outer diameter of the catheter was 1.7 mm and the inner diameter of each lumen was 0.5 mm. Two recording orifices, each 0.5 mm in diameter, were located 20 and 50 mm, respectively, from the tip of the catheter, and were used for manometry of the common bile duct (CBD) and SO, respectively. Two single-lumen side-hole polyethylene catheters were inserted for manometry of the stomach and duodenum. Each lumen of the catheter was infused with distilled water, at a rate of 0.2 ml/min, using a low-compliance pneumohydraulic capillary infusion system connected to a pressure transducer (DX-360; Ohmeda Medical Devices Division, Singapore). The pressures were recorded in a personal computer system (Macintosh Power Book 145B; Apple Computer, Cupertino, CA, USA). Pressure required to overcome catheter resistance and hydrostatic pressure generated by placing the recording orifices at the level of the SO was set at zero reference. Abrupt occlusion of the recording orifices gave a pressure rise rate of more than 300 mmHg per s, and this system was able to trace more than 40 contractions per min. The pressure monitoring system is shown in Fig. 1.

Preparation of drug

CCK-OP sulfate (C2175; Sigma Chemical, St. Louis, MO, USA) was prepared in saline, at a concentration of 1000 ng/ml, and frozen at −70°C until use.

Conduct of experiment

Interdigestive recording

Each dog was fasted for at least 18 h, with free access to water, and placed in a Pavlov sling in a fully conscious state without any sedation. After the rubber plug of the Thomas cannula was removed, the catheters were inserted through the cannula into the CBD, stomach, and duodenum. First, an 0.018-inch guidewire (Cook, Bloomington, IN, USA) was gently inserted into the CBD to facilitate subsequent passage of the manometric catheter into the CBD over the guidewire. A single-lumen polyethylene catheter was placed in the duode-

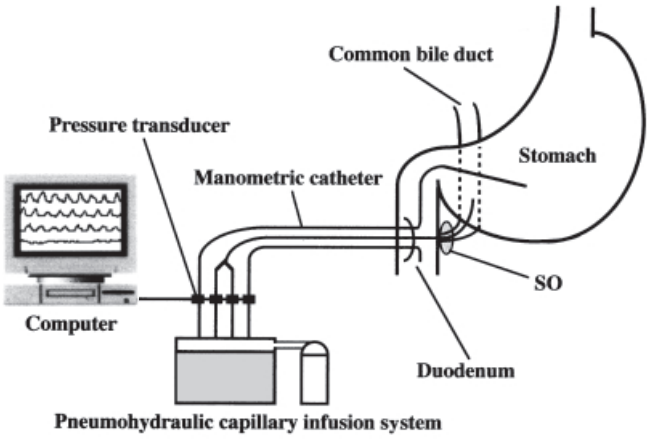

Fig. 1. Arrangement of the pressure monitoring system. A double-lumen catheter was inserted for manometry of the common bile duct and the sphincter of Oddi (*SO*). Two single-lumen catheters were inserted for manometry of the stomach and the duodenum. The pressures were recorded by computer

num at the level of the papilla, and another such catheter was placed into the gastric antrum. The cannula was closed with a rubber plug that had three holes for the manometric catheters. With the use of a stationary pull-through technique, the proper position of the recording orifice was confirmed by the characteristic tracings of the SO. In each dog, four cycles of the MMC were obtained before and after GTX.

Intravenous injection of CCK-OP

After the recording of the interdigestive MMC, 20 ng/kg of CCK-OP was injected as an intravenous bolus 20 min after the end of phase III; the recording was continued for at least 10 min after the injection of CCK-OP.

Analysis of data

Analysis of spontaneous motility of SO and duodenum

Manometric recordings from the CBD, SO, duodenum, and stomach were analyzed by visual inspection. The duodenal MMC was divided into four phases, according to the criteria defined by Carlson et al.[30] The cycle period of the MMC was defined as the time elapsed from the end of duodenal phase III until the end of the next phase III at the same recording site. The parameter of SO and CBD motility was defined as follows. The basal pressure of the SO was defined as the trough pressure between the phasic contractions. The amplitude was measured as the value from the basal pressure to the peak of SO contractions. Each period of the MMC was divided into 5% intervals, and the means of the SO basal pressure, amplitude of the SO phasic waves, and CBD pressures were calculated for each of the 5% intervals. The mean CBD pressure value was also calculated. These three values were calculated with the use of a Macintosh computer application (MacLab; AD Instruments, Castle Hill, NSW, Australia) and expressed as the standardized MMC cycle. For comparisons of the effects of GTX, the average SO basal pressure, average amplitude, and average CBD pressure throughout the MMC cycle were also calculated.

Effect of CCK-OP on SO motility

The effect of CCK-OP on SO motility was determined in terms of relaxation or contraction, and in terms of changes in the basal pressure and amplitude of the SO phasic waves.

Statistical analysis

The period of the MMC cycle and frequency of the SO phasic contractions, the basal pressure and amplitude of the SO phasic contractions, and the CBD pressure were analyzed using analysis of variance (ANOVA). These analyses were conducted using Statview J4.5 software (Abacus Concepts, Berkeley, CA, USA) for the Macintosh computer. All data values were expressed as means ± SEM. A difference in mean values was regarded as significant when the two-tailed P value was less than 0.05.

Results

General aspects

All control and GTX dogs had normal appetites and bowel movements. All experimental data utilized in this study were obtained when the dogs appeared healthy.

SO cyclic motility and gastroduodenal MMC before GTX

We recorded 19 cycles of the MMC from the four dogs before GTX. Phase III started in the stomach and then appeared sequentially in the duodenum in 16 of the 19 cycles. In the other 3 cycles, phase III began in the duodenum. During duodenal phase I, the SO continued to exhibit low-amplitude phasic contractions, while gastric and duodenal contractions were scarcely observed. The contractions gradually increased at all sites of recording during phase II. During phase III, the SO, stomach, and duodenum showed vigorous contractions of maximal amplitude and frequency (Fig. 2A). The configuration of phase III was characterized by repeated brief periods of inhibition of the SO and duodenal contractions at the time of gastric contractions (Fig. 3A). Fluctuations of CBD pressure were caused by the dog's movement and/or respiration.

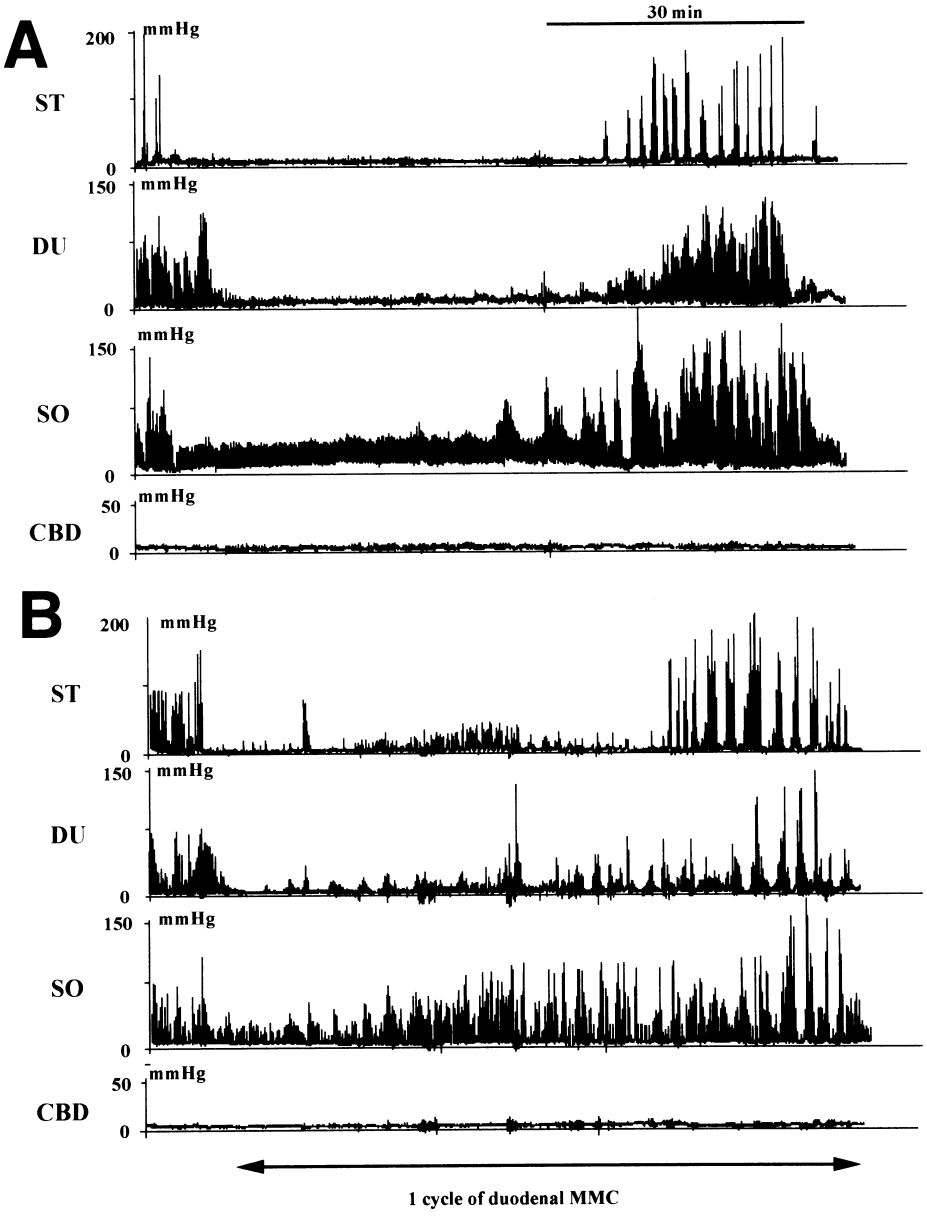

Fig. 2A,B. Manometric recordings from the stomach (*ST*), duodenum (*DU*), sphincter of Oddi (*SO*), and common bile duct (*CBD*) **A** before and **B** after gastric transection. The cyclic change in SO motor activity, coordinated with the gastric and duodenal activities, is clearly seen. The SO continued to show low-amplitude contractions during phase I and phase II before and after gastric transection. During phase III, the SO showed vigorous contractions of maximal amplitude before and after gastric transection

SO cyclic motility and gastroduodenal MMC after GTX

We recorded 16 cycles of the MMC from the four dogs after GTX. Phase III started in the stomach and then appeared sequentially in the duodenum in 11 of the 16 cycles. In the other five cycles, phase III began in the duodenum. Figure 2B shows typical recordings of an MMC cycle after GTX. The gastric and duodenal phase III occurred simultaneously. The SO continued to show low-amplitude contractions during phases I and II, as had occurred before GTX. During phase III, the SO showed maximal contractions, as did the stomach and duodenum. The configuration of phase III showed repeated inhibitions of the SO and duodenal contractions in concert with the gastric contractions, as had occurred before GTX (Fig. 3B).

Effect of GTX on SO motility

Cycle length of SO motility and duodenal MMC

The cycle length of SO cyclic motility and the duodenal MMC was 101 ± 18 min (n = 16) before GTX and 108 ± 5 min (n = 16) after GTX. The difference in

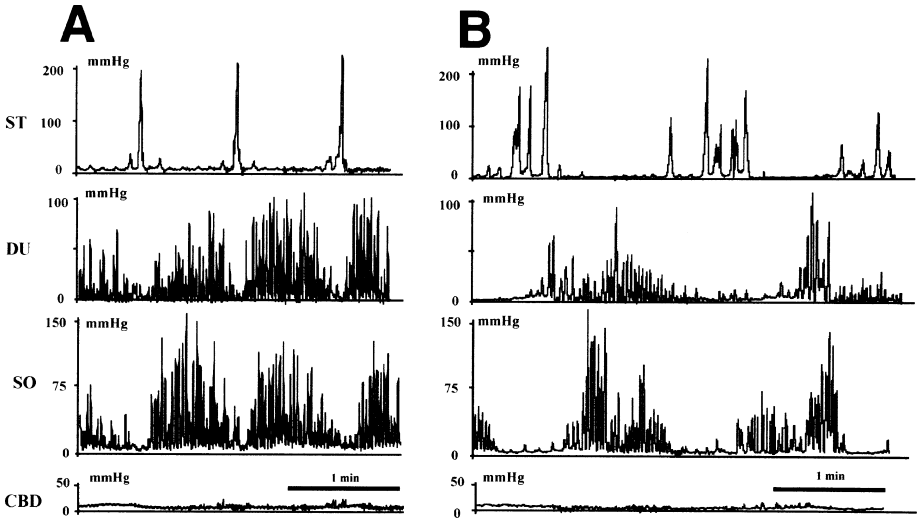

Fig. 3A,B. Expanded tracings obtained during phase III **A** before and **B** after gastric transection. The sphincter of Oddi (*SO*) and duodenum (*DU*) exhibited intermittent inhibitions concurrent with the contractions of the stomach (*ST*) before and after gastrectomy. *CBD*, Common bile duct

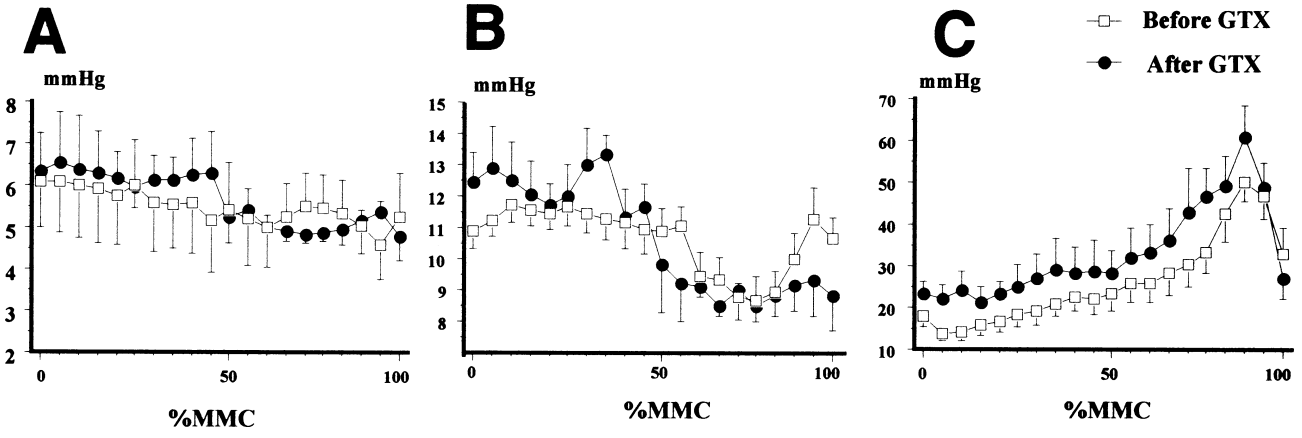

Fig. 4A–C. Comparisons of **A** mean CBD pressure, **B** mean SO basal pressure, and **C** mean SO amplitude before (*open squares*) and after (*filled circles*) a standardized migrating motor complex (MMC) cycle. The CBD pressure gradually decreased from phase I to phase III before and after gastric transection (GTX). The SO basal pressure also decreased from phase II to phase III. The SO amplitude gradually increased from phase I to phase III. This type of cyclic motility did not change after GTX

cycle length was not significant ($P = 0.715$). The duration of phase III before GTX (15.3 ± 0.9 min) was not different from that after GTX (16.0 ± 0.9 min; $P = 0.465$).

Frequency of SO phasic contractions during duodenal phase III

The frequency of the SO phasic contractions was 18.0 ± 0.5/min before GTX. The frequency of the SO phasic waves remained unchanged after GTX, being 17.9 ± 0.7/min ($P > 0.999$).

Common bile duct (CBD) pressure

The change in CBD pressure during a standardized MMC cycle is shown in Fig. 4A. CBD pressures gradually decreased from 0% MMC to 50% MMC before GTX. After GTX, this variation did not change. The mean CBD pressures throughout the whole MMC cycle, before and after GTX, were 5.5 ± 0.2 and 5.7 ± 0.2 mmHg, respectively ($P = 0.909$).

SO basal pressure

The change in the SO basal pressure during a standardized MMC cycle before and after GTX is shown in Fig. 4B. Before GTX, the basal pressure varied cyclically, being stable during phase I and phase II, but becoming low from the end of phase II to phase III (50% MMC to 80% MMC). After GTX, this feature of the SO cyclic change remained unchanged. The mean SO basal pressure throughout the MMC cycle before and after GTX was 10.6 ± 0.2 and 10.7 ± 0.3 mmHg, respectively ($P = 0.975$).

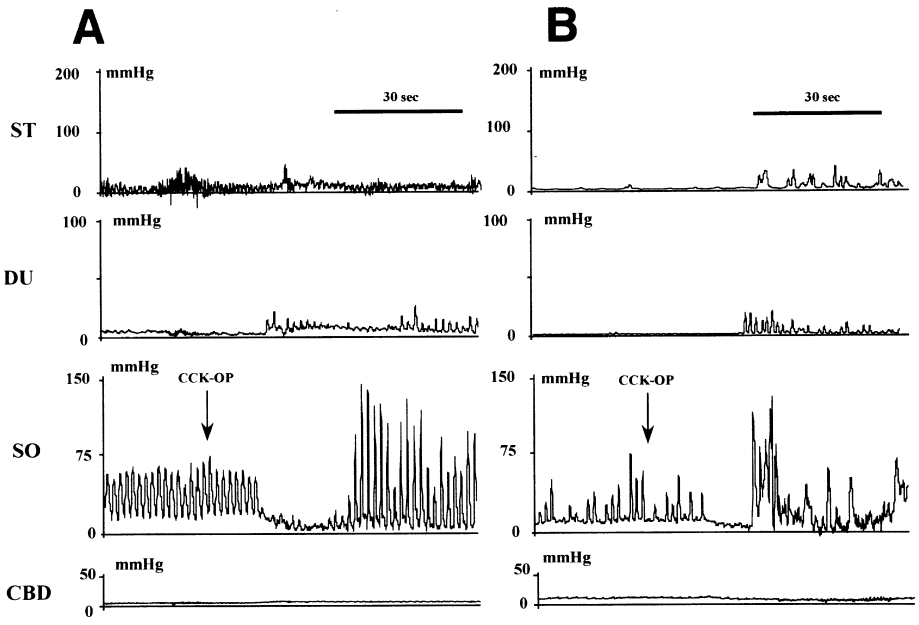

Fig. 5A,B. Responses of pressures in the common bile duct (*CBD*), sphincter of Oddi (*SO*), stomach (*ST*), and duodenum (*DU*) to a bolus injection of cholecystokinin octapeptide (CCK-OP) **A** before and **B** after GTX. After GTX, CCK-OP abolished SO phasic contractions, as had occurred before GTX

Amplitude of SO phasic contraction

The cyclic change in the amplitude of SO phasic waves is shown in Fig. 4C. Before GTX, the SO amplitude gradually increased from phase I to phase II, and reached the maximum during phase III. After GTX, this variation remained unchanged. The mean SO amplitude throughout the whole cycle tended to increase, but the difference was not significant, being 26.0 ± 1.2 mmHg before GTX and 32.9 ± 1.7 mmHg after GTX ($P = 0.304$).

Responses to CCK-OP

Before GTX, CCK-OP abolished SO phasic contractions for less than 30 min ($n = 16$). After the inhibition, the SO showed increased contractile activity (Fig. 5A). The stomach and duodenum exhibited a very slight increase in contractile activity after the CCK-OP injection. After GTX, CCK-OP, similarly, inhibited the SO phasic contractions ($n = 16$) (Fig. 5B). The effect of CCK-OP on the gastric and duodenal motility showed no remarkable change.

Discussion

The present study showed that: (1) the temporal coordination of the MMC between the stomach, duodenum, and SO did not change after GTX, (2) the CBD pressure, SO basal pressure and amplitude during the duodenal MMC showed a cyclic change before GTX, and this change did not disappear after GTX, (3) the mean CBD pressure, mean SO basal pressure and amplitude did not change significantly before and after GTX, and (4) CCK-OP exerted an inhibitory effect on SO motility before and after GTX.

In the neurally intact gut, the gastric MMC was followed by the duodenal MMC. The mechanism of this coordination is not completely understood. Possible mechanisms include extrinsic innervation,[31–33] hormones,[34] and/or intrinsic myoneural continuity. Simultaneous recording of the intraluminal pressures in the stomach, duodenum, CBD, and SO has enabled us to realize the precise coordination of gastroduodenal and biliary motility. In our study, only gastropyloric myoneural continuity was severed, and the temporal coordination of the initiation of phase III between the stomach and duodenum remained unchanged. Tanaka et al.[35] also showed that proximal duodenal transection, 0.5 cm distal to the pylorus, did not alter the period of the MMC and the temporal coordination of phase III between the stomach and duodenum. These data show that gastroduodenal intrinsic myoneural continuity is not essential for the temporal coordination of the gastroduodenal MMC.

Baseline recordings in this study showed repeated inhibitions of SO and duodenal motility during phase III activity of the MMC. Our data clearly showed that this intermittent quiescence occurred at the time of gastric contraction. This observation has also been reported by other investigators. Wyatt[9] was the first to demonstrate that contractions of the gastric antrum often resulted in relaxation of the sphincter of Oddi and increased bile flow. The simultaneous coordination between each contraction of the antrum and quiescence of the duodenum and SO suggests that these two phenomena may be related to a neural reflex mechanism, rather than to hormonal mechanisms, which would take longer to reach the target organ and be unable to control the fine temporal coordination. Our study

showed that this coordination did not disappear after GTX, indicating that extrinsic neural pathways and/or duodenal intrinsic nerves, rather than gastric intrinsic nerves, play a principal role in controlling this phenomenon. Previous studies demonstrated that these intermittent inhibitions of the duodenal activity during phase III of the MMC disappeared after cervical vagal cooling[31] and after proximal duodenal transection and anastomosis.[26]

We showed the presence of a cyclic change in CBD pressure, as well as in SO motility, in association with the MMC cycle in conscious dogs. The CBD pressure and SO basal pressure exhibited a parallel change during the MMC, being stable during 0%–50% of the MMC and gradually decreasing from 50%–80% of the MMC. The SO amplitude gradually increased and reached the maximum during 80%–95% of the MMC. These cyclic changes in SO motility may play a role in delivering bile into the duodenum. Matsumoto et al.[36] and Ura et al.[10] showed a cyclic change in gallbladder motor activity as well. The gallbladder contracts from the end of phase II to phase III. At the time of the gallbladder contractions, gallbladder bile flows into the CBD and is delivered into the duodenum through the SO. Both gallbladder contractions and SO relaxation are important in bile delivery into the duodenum. The decrease in SO basal pressure observed in this experiment seems natural for delivering bile into the duodenum. Because the CBD has no contractile activity,[15] the decrease in CBD pressure from phase II to phase III is thought to reflect the decrease in SO basal pressure.

SO motility is regulated, at least in part, by some kinds of intrinsic neural pathways. Shimura et al.[37] showed that excitatory intramural pathways between the SO and duodenum were primarily cholinergic, but contained an adrenergic component. Helm et al.[38] showed that the opossum SO was innervated by intramural cholinergic excitatory nerves and nonadrenergic noncholinergic inhibitory nerves. More recently, nonadrenergic, noncholinergic inhibitory nerves, mediated by nitric oxide, were proved to regulate interdigestive SO motility.[39,40] Moreover, the existence of long neural projections between the SO and duodenum has also been reported.[41] We found that mean CBD pressure, SO basal pressure and amplitude did not change significantly after GTX. This result indicates that the intrinsic neural pathways at the prepyloric region may not have an influence on SO motility.

A variety of gut hormones and peptides are known to affect SO motility and the gastrointestinal MMC.[14,19–20] In our study, the effect of CCK-OP on the SO was tested before and after GTX. CCK and CCK-OP are well known relaxants of the SO in dogs.[21] The inhibitory effect of cerulein, an analogue of CCK, on the canine SO reverses to excitation after gastrectomy.[24] Although CCK has a direct excitatory effect on gut smooth muscle, there is another inhibitory pathway, only in the SO.[38–40] Our recent experiment showed that CCK exhibited a "paradoxical" response, contraction instead of relaxation, after proximal duodenal transection and anastomosis in conscious dogs.[26] In the present study, GTX did not change the effect of CCK on the SO. These two observations suggest that the proximal duodenal region may include inhibitory neural pathways mediated by CCK, but the prepyloric area of the stomach does not contain neural pathways affecting SO motility.

In this experiment, we showed that prepyloric gastric transection did not change the cyclic variation of SO motility in conscious dogs. Prepyloric gastric transection and preservation in pyloroduodenal continuity are the chief procedures in pylorus-preserving gastrectomy (PPG), first employed by Maki et al.[42] and accepted for the treatment of peptic ulcer and early gastric cancer.[42,43] Although the preservation of pyloric function is claimed to help prevent the occurrence of cholelithiasis after gastrectomy,[42] whether or not the rate of cholelithiasis after PPG decreases is still controversial. With regard to motor functions of the biliary tract, Enjoji et al.[44] showed that gallbladder motility did not change after PPG. The present data, showing that prepyloric gastric transection did not affect SO motility in a chronic animal model, would seem to provide further support for the idea that PPG prevents postgastrectomy cholelithiasis. However, the role of the antrum in the SO function was not evaluated in our study. Further experiments are necessary to elucidate the real effect of PPG on SO cyclic motor function.

References

1. Majoor CLH, Suren TJJ. Gallbladder complications following resection of stomach for peptic ulcer. BMJ 1947;2:8–11.
2. Turunen M, Antila L. Gallbladder disease following gastrectomy. Acta Chir Scand 1964;127:134–7.
3. Inoue K, Fuchigami A, Higashida S, Sumi S, Kogire M, Suzuki T, et al. Gallbladder sludge and stone formation in relation to contractile function after gastrectomy. Ann Surg 1992;215:19–26.
4. Allen B, Bernhoft R, Blanckaert N, Svanvik J, Filly R, Gooding G, et al. Sludge is calcium bilirubinate associated with bile stasis. Am J Surg 1981;141:51–6.
5. Parkin GJS, Smith RB, Johnston D. Gallbladder volume and contractility after truncal, selective and highly (parietal cell) vagotomy in man. Ann Surg 1973;178:581–6.
6. Ihasz M, Griffith CA. Gallstone after vagotomy. Am J Surg 1981;141:48–50.
7. Nakayama F. Cholelithiasis—cause and treatment. Tokyo: Igaku-Shoin; 1997.
8. Wyatt AP. Effect of gastrectomy on biliary dynamics. Gut 1969;10:91–3.
9. Wyatt AP. The relationship of the sphincter of Oddi to the stomach, duodenum and gall-bladder. J Physiol 1967;193:225–43.

10. Ura K, Sarna SK, Condon RE. Antral control of gallbladder cyclic motor activity in the fasting state. Gastroenterology 1992; 102:295–302.
11. Szurszewski JH. A migrating electric complex of the canine small intestine. Am J Physiol 1969;217:1757–63.
12. Sarna SK. Cyclic motor activity: migrating motor complex. Gastroenterology 1985;89:894–913.
13. Oddi R. D'une disposition a sphincter speciale de l'ouverture du canal choledoque. Arch Ital Biol 1887;8:317–22.
14. Honda R, Toouli J, Dodds WJ, Geenen JE, Hogan WJ, Itoh Z. Effect of enteric hormones on sphincter of Oddi and gastrointestinal myoelectric activity in fasted conscious opossums. Gastroenterology 1983;84:1–9.
15. Scott RB, Strasberg SM, EL-Sharkawy TY, Diamant NE. Fasting canine biliary secretion and the sphincter of Oddi. Gastroenterology 1984;87:793–804.
16. Yokohata K, Kimura H, Ogawa Y, Naritomi G, Tanaka M. Biliary motility. Change in detailed characteristics correlated to duodenal migrating motor complex and effects of morphine and motilin in dogs. Dig Dis Sci 1994;39:1294–1301.
17 Deng ZL, Takeda T, Yokohata K, Konomi H, Naritomi G, Tanaka M. Effects of intraduodenal air insufflation on sphincter of Oddi motility in conscious dogs. Dig Dis Sci 1996;41:1557–63.
18. Pitt HA, Doty JE, DenBesten L, Kuckenbecker SL. Altered sphincter of Oddi phasic activity following truncal vagotomy. J Surg Res 1982;32:598–607.
19. Behar J, Biancani P. Pharmacologic characterization of excitatory and inhibitory cholecystokinin receptors of the cat gallbladder and sphincter of Oddi. Gastroenterology 1987;92:764–70.
20. Sarles JC, Bidant JM, Devaus MA, Echinard C, Castagnini C. Actions of cholecystokinin and caerulein on the rabbit of sphincter of Oddi. Digestion 1976;14:415–23.
21. Lin TM, Spray GF. Effect of pentagastrin, cholecystokinin, caerulein and glucagon on the choledochal resistance and bile flow of dogs (Abstract). Gastroenterology 1969;56:1178.
22. Ono K, Suzuki H, Sasaki M, Endoh M. Gastrointestinal hormones and motility of the human sphincter of Oddi. J Smooth Muscle Res 1985;21:Suppl, 69–75.
23. Odani K, Nimura Y, Yasui A, Akita Y, Shionoya S. Paradoxical response to cerulein on sphincter of Oddi in the patient with gastrectomy. Dig Dis Sci 1992;37:904–11.
24. Kobayashi T, Hosoba T, Mori M, Mimura H, Tsuge H, Orita K, et al. Effect of gastrectomy on motility, perfusion pressure, and caerulein-induced relaxation of sphincter of Oddi in dogs. J Smooth Muscle Res 1994;30:85–96.
25. Tanaka M, Senninger N, Runkel N, Herfarth C. Sphincter of Oddi cyclic motility. Effect of translocation of the papilla in opossums. Gastroenterology 1990;98:347–52.
26. Deng Z-L, Nabae T, Konomi H, Takahata S, Yokohata K, Ogawa Y, et al. Effects of proximal duodenal transection and anastomosis on interdigestive sphincter of Oddi cyclic motility in conscious dogs. World J Surg 2000;24:863–9.
27. Boyden EA. The anatomy of the choledochoduodenal junction in man. Surg Gynecol Obstet 1957;104:641–52.
28. Lin TM. Action of gastrointestinal hormones and related peptides on the motor function of the biliary tract. Gastroenterology 1975;69:1006–22.
29. Sarna S, Condon RE, Cowles V. Enteric mechanism of initiation of migrating myoelectric complexes in dogs. Gastroenterology 1983;84:814–22.
30. Carlson GM, Bedi BS, Code CF. Mechanism of propagation of intestinal interdigestive myoelectric complex. Am J Physiol 1972;222:1027–30.
31. Hall KE, El-Sharkawy TY, Diamant NE. Vagal control of migrating motor complex in the dog. Am J Physiol 1982;243:G276–84.
32. Hashmonai M, Go VLW, Szurszewski JH. Effect of total sympathectomy and decentralization on migrating complex in dogs. Gastroenterology 1987;92:978–86.
33. Marik F, Code CF. Control of the interdigestive myoelectric activity in dogs by the vagus nerves and pentagastrin. Gastroenterology 1975;69:387–95.
34. Van Lier Ribbink JA, Sarr MG, Tanaka M. Neural isolation of the entire canine stomach in vivo: effects on motility. Am J Physiol 1989;257:G30–40.
35. Tanaka M, Dalton RR, Smith CD, van Lier Ribbink JA, Sarr MG. The role of myoneural and luminal continuity in the coordination of canine gastroduodenal patterns of motility. J Surg Res 1992;53:588–95.
36. Matsumoto T, Sarna SK, Condon RE, Dodds WJ, Mochinaga M. Canine gallbladder cyclic motor activity. Am J Physiol 1988;255: G409–16.
37. Shimura ME, Harvey JR, Costi D, Baker RA, Toouli J, Saccone GTP. In vitro characterization of mural neural pathways between the duodenum and the sphincter of Oddi of the brush-tailed possum. J Auton Nerv Syst 1997;63:77–84.
38. Helm JF, Dodds WJ, Christensen J, Sarna SK. Intramural neural control of opossum sphincter of Oddi. Am J Physiol 1989;257: G925–9.
39. Kaufman HS, Shermak MA, May CA, Pitt HA, Lilleomoe KD. Nitric oxide inhibits resting sphincter of Oddi activity. Am J Surg 1993;165:74–80.
40. Cullen JJ, Herrmann BM, Thomas PM, Fang S, Murray JA, Ledlow A, et al. The role of antioxidant enzymes in the control of opossum sphincter of Oddi motility. Am J Physiol 1997;272: G1050–6.
41. Padbury RTA, Furness JB, Baker RA, Toouli J, Messenger JP. Projections of nerve cells from the duodenum to the sphincter of Oddi and gallbladder of the Australian possum. Gastroenterology 1993;104:130–6.
42. Maki T, Shiratori T, Hatafuku T, Sugawara K. Pylorus-preserving gastrectomy as an improved operation for gastric ulcer. Surgery 1967;61:838–45.
43. Isozaki H, Okajima K, Nomura E, Ichinoka T, Fujii K, Izumi N, et al. Postoperative evaluation of pylorus-preserving gastrectomy for early gastric cancer. Br J Surg 1996;83:266–9.
44. Enjoji A, Ura K, Ozeki K, Tsukamoto M, Ikematsu Y, Kanematsu T. Cyclic motor activity of the gallbladder maintained in a pylorus-preserving gastrectomy in dog. Jpn J Surg 1996;26: 489–95.