

## Myoelectric Activity of the Diverted Antroduodenum in the Dog<sup>1</sup> (41499)

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**Abstract.** Electrodes were chronically implanted on the gastric antrum and the orad duodenum of four dogs. After implantation, fasted and fed state myoelectric activity was monitored. After control testing, the stomach was bisected at the orad antral margin and the antrum was closed. Bowel continuity was restored with gastrojejunostomy. Extrinsic nerves of the antroduodenal segment were maintained. After surgery, fasted and fed state myoelectric activity were reassessed. Surgical diversion of the antroduodenum was previously shown to result in chronic hypergastrinemia of a postprandial magnitude. It also resulted in separation of the antrum from its myoelectric pacemaker in the orad corpus, and a chronic absence of digesta from the diverted segment. In the present experiments both the antrum and duodenum exhibited unchanged migrating myoelectric complex periods. In addition, the duodenal BER frequency was unchanged. In contrast, the antral BER frequency was reduced by half. The percentage of antral and duodenal BER superimposed with spike potentials in response to liquid or solid meals was unchanged postoperatively despite the absence of food in the diverted segment, and despite the absence of postprandially elevated gastrin. We conclude that a hormone other than gastrin, and/or a neurally conducted impulse generated by the physical presence of the food in the gastric remnant, contributed to the postprandial generation of spike potentials in the diverted antroduodenum.

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Distinct electric and motor patterns have been characterized in dogs in both the fasted and fed states (1, 2). Meals of liquid and solid foods elicit a characteristic antroduodenal myoelectric pattern where one or two duodenal spike potentials immediately follow the antral BER and spike potential (3, 4). This well-timed spike potential activity corresponds to antral and duodenal contractions that coordinate gastric emptying. Emptying probably occurs as a result of an antral contraction pushing chyme into a relaxed duodenum, immediately followed by duodenal contractions that propel the gastric effluent caudally (5, 6). Because this electric pattern exists postprandially only, the physical presence of food seems prerequisite to pattern development (3).

We have recently reported the use of an experimental model where the stomach was bisected, the orad antrum closed, and tract

continuity restored via gastrojejunostomy (antroduodenal exclusion) (7). This procedure resulted in chronic diversion of ingesta from the antrum and duodenum, and a chronic hypergastrinemia which was not further elevated by feeding. The postprandial elevation of gastrin has long been postulated to play a major role in generating gastric (8, 9) and intestinal (10) spike potentials and has tacitly been assigned a role in the generation of postprandial electric patterns. In this study we tested whether the diverted antroduodenum could still exhibit the usual postprandial electric pattern, despite no direct contact with food, and an absence of postprandially elevated gastrin. We report here that the diverted antroduodenum did exhibit a significant, though reduced, amount of coordinated electric activity postprandially. We suggest that an extrinsic neural reflex and/or a non-gastrin hormonal effect may have initiated this activity.

**Materials and Methods.** *Surgical model.* The surgical preparation of this model in our laboratory has recently been de-

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scribed (7). Briefly, four dogs of mixed breed (10–20 kg) were anesthetized with pentobarbital, 30 mg/kg intravenously (Abbott Lab). Silver monopolar electrodes whose construction and method of implantation have been reported (4) were implanted on the gut. One electrode was sutured to the serosa of the gastric antrum ca. 2–4 cm orad to the gastroduodenal junction, and two electrodes were implanted on the duodenum ca. 2 and 4 cm caudad to the gastroduodenal junction. A reference electrode was sewn into the subcutaneous tissue of the left flank. A 2-week recovery period was allowed after which control testing was performed. After testing, the animals were again prepared for surgery. At celiotomy, the stomach and attending neurovascular processes were transected at the gastric incisura. Care was taken to preserve the nerve and vascular supply to the antrum. The antral margin was then closed, removing the gastric antrum from its electric pacemaker in the orad corpus. The gastric remnant was partially closed and anastomosed end-to-side to the jejunum ca. 10 cm caudad to the ligament of Treitz causing antroduodenal exclusion from the normal digestive path (Fig. 1). After a two-week recovery the dogs were retested. In all cases an 18-hr fast preceded the tests.

**Tests.** Several myoelectric parameters in the interdigestive and digestive states were compared pre- and postoperatively to characterize the motility of the diverted antroduodenal segment.

**Interdigestive activity.** Antral and duodenal interdigestive migrating myoelectric complexes (MMC) were identified. The interdigestive state is marked by a continuum of myoelectric activity phases where phases 1, 2, and 3 represent intervals of low, intermediate, and high numbers of spike potentials, respectively, superimposed upon the basic electric rhythm (BER) of the stomach and small bowel (1). The periodicity of the MMC (time interval from phase 3 to the subsequent phase 3, usually ca. 100 min) was measured for the antrum and duodenum and each were compared pre- and postoperatively. Both antral and duodenal BER frequencies were calculated

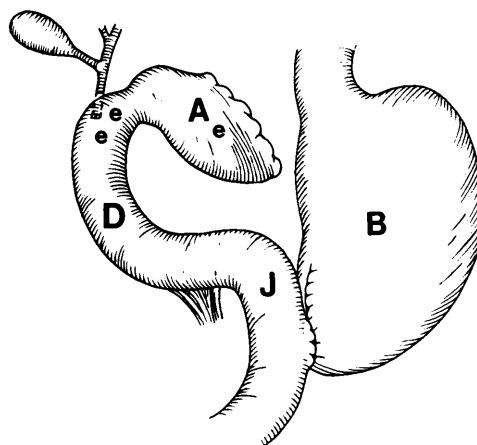


FIG. 1. Surgical diversion of the gastric antrum and duodenum. (A) Antrum, (B) gastric body, (D) duodenum, (J) jejunum, (e) electrode.

from samples taken from the first 30 min of phase 1. Postoperative interdigestive antral records were further examined qualitatively for the presence of antral BER cycles that were of greater than normal frequency ( $>5$  BER/min), or for the presence of duodenal BER superimposed upon the antral BER.

**Digestive state activity.** Antral and duodenal spike potential responses to both liquid and solid meals were each compared pre- and postoperatively. The composition and gastric emptying properties of the liquid meal have been described (11). At the beginning of phase 1 of the interdigestive state, 300 ml of a citrate-fat liquid test meal was fed via an oral-gastric tube. Test meals were administered during phase 1 to prevent confounding of the postprandial myoelectric response by spontaneous (i.e., phase 2 or 3) spike potential activity. The percentage of antral and duodenal BER superimposed with spike potentials in response to the meal was determined for the 30 min immediately following feeding. This method was also used to determine the response to 250 g of canned dog food (Vets, Perk Foods Co. Inc.). However, after feeding of solid food, the response was quantified from 15 to 45 min.

In addition to the presence of duodenal spike potentials, their distribution relative

to the antral BER cycle was examined. In unoperated dogs, postprandial spike potentials are superimposed on the first and/or second duodenal BER that immediately follows the antral BER (3). Because the ratio of duodenal to antral cycles is ca. 4.5:1, the duodenal spike potentials usually appear concurrently with the first half of the antral cycle. This electric relationship was considered to be retained postoperatively if the duodenal spike potentials occurred only during the first half of the antral cycle. The percentage of antral cycles so accompanied by duodenal spike potentials was determined pre- and postoperatively after both liquid and solid meals.

**Data analysis.** Control and postoperative values obtained during the digestive and interdigestive states were each compared using the *t* test for unpaired values. In all cases, values used in the comparisons represent the mean of one to three observations per dog.

**Results. Interdigestive state.** Surgical separation of the gastric antrum from its pacemaker in the orad corpus resulted in a reduced antral BER frequency (bradygastria, Table I), and the cycles were arrhythmic (Fig. 2, A vs B and C). In contrast, the duodenal BER frequency was unaltered postoperatively (Table I). There was no evidence that the duodenal pacemaker hastened the frequency of the un-paced, slowed antral BER, and there was no superimposition of duodenal rhythms upon the antral record. Neither the diverted antrum nor the duodenum exhibited

changes in the MMC period postoperatively (Table I).

**Digestive state.** Feeding abolished migrating myoelectric complexes on the diverted segment. Both the antrum and the duodenum exhibited fed state electric patterns after meals of liquid and solid despite being removed from the digestive path.

Preoperatively, the usual temporal relationship between postprandial duodenal spike potentials and the antral BER cycle, as first described by Allen *et al.* (3) was confirmed. In contrast, after antroduodenal exclusion, postprandial duodenal spike potentials either accompanied the first half of the arrhythmic antral cycle (Fig. 2B), or occurred throughout the duration of the antral cycle (Fig. 2C). These two postoperative patterns appeared with equal frequency after both liquid and solid meals.

Antroduodenal exclusion did not affect the overall percentage of antral or duodenal BER which were associated with spike potentials in response to liquid and solid meals (Table II). However, the distribution of postprandial duodenal spike potentials was changed postoperatively. Preoperatively, after solid food,  $80 \pm 6\%$  of the antral cycles were accompanied by duodenal spike potentials which appeared on the first and/or second BER immediately following the antral cycle (Fig. 2A). Postoperatively, after solid food,  $41 \pm 6\%$  of the antral cycles remained temporally associated with duodenal spike potentials. In contrast, after the liquid meal, the degree of temporally related response was not changed post-

TABLE I. INTERDIGESTIVE BASIC ELECTRIC RHYTHM (BER) FREQUENCIES AND MIGRATING MYOELECTRIC COMPLEX (MMC) PERIODS BEFORE AND AFTER ANTRODUODENAL EXCLUSION

Parameter	Organ	Values		<i>P</i> <sup>a</sup>
		Preop	Postop	
BER frequency (cpm)	Antrum	$5.1 \pm 0.1^b$ (3) <sup>c</sup>	$2.2 \pm 0.4$ (3)	<0.005
	Duodenum	$18.8 \pm 0.7$ (3)	$18.2 \pm 0.3$ (3)	>0.05
MMC period (min)	Antrum	$110 \pm 18$ (4)	$105 \pm 9$ (3)	>0.05
	Duodenum	$111 \pm 7$ (4)	$97 \pm 6$ (3)	>0.05

<sup>a</sup> *t* test for unpaired values.

<sup>b</sup> Mean  $\pm$  SEM.

<sup>c</sup> Number of dogs.

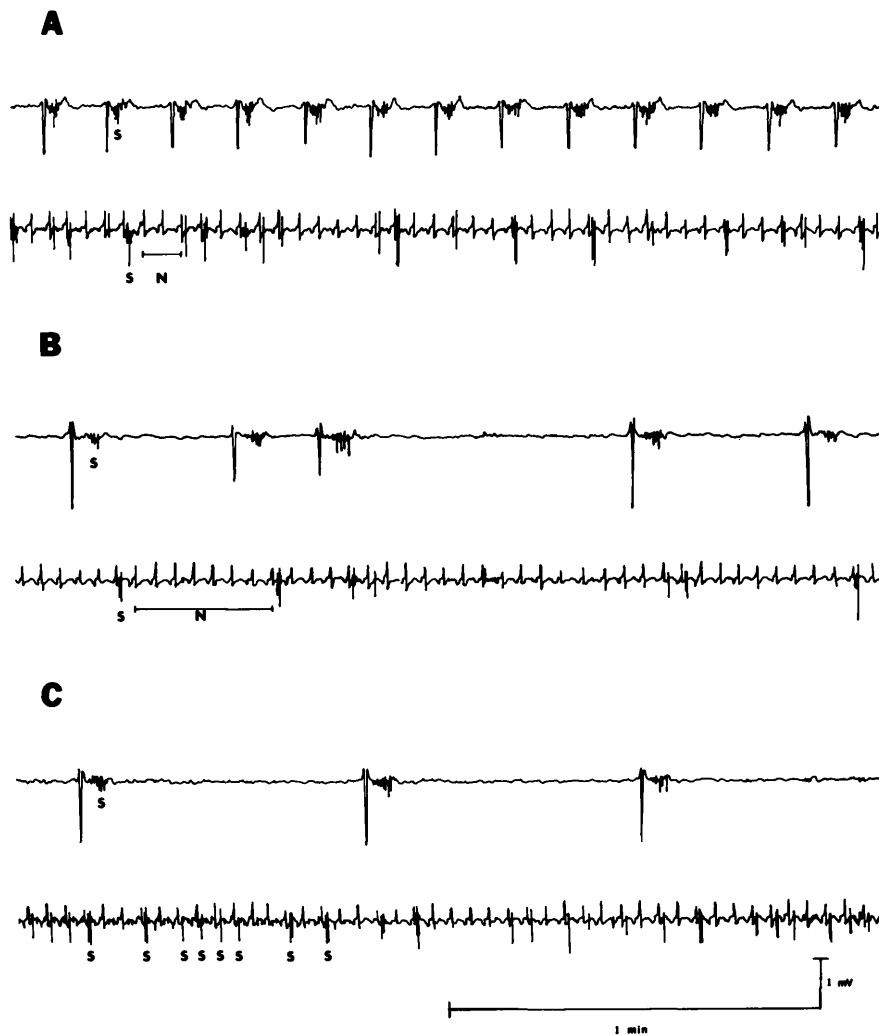


FIG. 2. Postprandial myoelectric records in the preoperative state (A) and after antroduodenal exclusion (B, C). Tracings in A, B, and C were each taken from the gastric antrum and proximal duodenum, respectively. Note that preoperatively, duodenal spike potentials (S) generally occurred on the first one or two duodenal BER cycles after the antral BER, followed by a nonspiking interval (N). After antroduodenal exclusion the antral BER was arrhythmic and slowed (B, C). In addition two postprandial antroduodenal patterns were seen where: (a) the duodenal spike potentials immediately followed the antral BER (B), and (b) the duodenal spike potentials accompanied the whole antral BER cycle (C).

operatively ( $39 \pm 14$  vs  $45 \pm 5\%$  antral cycles temporally associated with duodenal spike potentials, pre- and postoperatively, respectively).

**Discussion.** In the present studies, the antrum and duodenum exhibited fed state myoelectric patterns postprandially despite

being removed from the digestive path. Thus, the contact of food with the antroduodenal segment is not required for postprandial pattern generation. The extrinsic nerves to the diverted segment were intact, and serum gastrin levels were not raised postprandially. This suggests that an

TABLE II. SPIKE POTENTIAL RESPONSES OF THE ANTRUM AND DUODENUM TO BOTH LIQUID AND SOLID MEALS BEFORE AND AFTER ANTRODUODENAL EXCLUSION

Meal	Percentage BER with spike potentials <sup>a</sup>		P <sup>b</sup>
	Preop	Postop	
	Antrum		
Liquid	43 ± 9 <sup>c</sup> (4) <sup>d</sup>	64 ± 12 (3)	>0.05
Solid	89 ± 10 (4)	72 ± 13 (3)	>0.05
	Duodenum		
Liquid	24 ± 2 (4)	27 ± 16 (3)	>0.05
Solid	25 ± 3 (4)	28 ± 16 (3)	>0.05

<sup>a</sup> Cumulated from 0 to 30 min after the liquid, and 15 to 45 min after the solid meals, respectively.

<sup>b</sup> *t* test for unpaired values.

<sup>c</sup> Mean ± SEM.

<sup>d</sup> Number of dogs.

extrinsic nervous reflex, originating in the gastric remnant, or a hormone other than gastrin participated in the spike potential generation in the diverted antroduodenum.

Feeding produces phasic pressure changes (contractile or spike potential correlate) in denervated fundic pouches, suggesting hormonal initiation and maintenance of postprandial motor activity (12). Acute administration of gastrin peptides converts gastric and intestinal interdigestive myoelectric patterns to those resembling postprandial patterns (8–10). However, a major role for gastrin in converting the fasted state electric pattern to that of the fed state has been disputed (13). We have previously reported unaltered intestinal interdigestive myoelectric patterns in dogs exhibiting chronic hypergastrinemia (7). Dogs with antroduodenal exclusion were previously shown to chronically exhibit significant elevations of serum gastrin in the fasted state (74 ± 9 vs 120 ± 13 pg/ml, pre- vs postoperative, respectively) (7). We hypothesized that chronic elevations of gastrin rendered the gastric corpus and jejunum refractory to the muscle stimulating effects of gastrin. If the motor effects of gastrin are factored out of the present model, then the spike potentials in response to food may have been initiated by another

hormone, particularly since feeding did not elevate serum gastrin levels postoperatively (172 ± 26 vs 120 ± 13 pg/ml, postoperative fed vs fasted, respectively) (7). Insulin, cholecystokinin, and secretin are also released postprandially and have been shown to interrupt fasted state patterns or produce fed state-like myoelectric activity (14–16). Thus, in the diverted antroduodenum, gastrin is not required for postprandial spike potential activity; multiple hormone involvement is likely.

The present surgical model does not permit differentiation between the hormonal and nonhormonal generation of spike potentials in the diverted antroduodenum. The extrinsic nerve supplies were not interrupted by antroduodenal exclusion. Thus the presence of food in the gastric corpus may have influenced spike potential generation in the antroduodenal segment via an extrinsic nervous reflex. In addition, contact of food with the jejunal mucosa may have initiated nerve signals, conducted through the intrinsic nerves, to initiate duodenal spike potential activity. Theoretically, food could have entered the diverted segment to initiate duodenal spike potentials. However, antral and duodenal spike potentials appeared immediately after feeding. Further, propagated contractions on the duodenum after meals of liquid and solid occur principally in the caudad direction (17). Both observations argue against ingesta ascending the diverted duodenum to initiate both antral and duodenal spike potentials postprandially.

The gastroduodenal junction markedly reduces the amplitude of antral and duodenal BER conducted through it to function as an electrical insulatory zone (18). This zone is effective in preventing duodenal entrainment of the gastric BER as demonstrated by electrical pacing experiments (19). Further, a hastening ("duodenalization") of the antral BER frequency to 14 cpm has been demonstrated in dogs after resection of the gastroduodenal junction with antroduodenal anastomosis (20). In the present studies, antroduodenal exclusion resulted in the separation of the antrum from its pacemaker in the orad cor-

pus, without damaging the gastroduodenal junction. Postoperative bradygastric signals remained independent of the duodenal pacemaker influence since no evidence of antral tachygastric (>5 cpm) was obtained. This confirms the presence of a functional insulatory zone and supports the conclusion (19) that transpyloric BER conduction does not influence antral or duodenal BER frequencies.

1. Code CF, Marlett JA. The interdigestive myoelectric complex of the stomach and small bowel of dogs. *J Physiol* 246:289–309, 1975.
2. Carlson GM, Ruddon RW, Hug CC Jr, Bass P. Effects of nicotine on gastric antral and duodenal contractile activity in the dog. *J Pharmacol Exp Ther* 172:367–376, 1970.
3. Allen GL, Poole EW, Code CF. Relationships between electrical activities of antrum and duodenum. *Amer J Physiol* 207:906–910, 1964.
4. McCoy EJ, Bass P. Chronic electrical activity of gastroduodenal area: Effects of food and certain catecholamines. *Amer J Physiol* 205:439–445, 1963.
5. Carlson HC, Code CF, Nelson RA. Motor action of the canine gastroduodenal junction: A cineradiographic, pressure, and electric study. *Amer J Dig Dis* 11:155–172, 1966.
6. Bass P, Russell J. Gastric emptying of liquids: Role of the small intestine. In: Chey WY, ed. *Functional Disorders of the Digestive Tract*. Raven Press, New York, in press, 1983.
7. Russell J, Bass P, Shimizu M, Miyauchi A, Go VLW. Canine intestinal ulcer: Myoelectric components and the effect of chronic hypergastrinemia. *Gastroenterology* 82:746–752, 1982.
8. Kelly KA. Effect of gastrin on gastric myoelectric activity. *Amer J Dig Dis* 15:399–405, 1970.
9. Thomas PA, Schang J, Kelly KA, Go VLW. Can endogenous gastrin inhibit canine interdigestive gastric motility? *Gastroenterology* 78:716–721, 1980.
10. Weisbrodt NW, Copeland EM, Kearley RW, Moore EP, Johnson LR. Effects of pentagastrin on electrical activity of small intestine of the dog. *Amer J Physiol* 227:425–429, 1974.
11. Weisbrodt NW, Wiley JN, Overholt BF, Bass P. A relation between gastroduodenal muscle contractions and gastric emptying. *Gut* 10:543–548, 1969.
12. Thomas PA, Kelly KA. Hormonal control of interdigestive motor cycles of canine proximal stomach. *Amer J Physiol* 237:E192–E197, 1979.
13. Eeckhout C, De Wever I, Peeters T, Hellemans J, Vantrappen G. Role of gastrin and insulin in postprandial disruption of migrating complex in dogs. *Amer J Physiol* 235:E666–E669, 1978.
14. Bueno L, Ruckebusch M. Insulin and jejunal electrical activity in dogs and sheep. *Amer J Physiol* 230:1538–1544, 1976.
15. Mukhopadhyay AK, Thor PJ, Copeland EM, Johnson LR, Weisbrodt NW. Effect of cholecystokinin on myoelectric activity of small bowel of the dog. *Amer J Physiol* 232:E44–E47, 1977.
16. Mukhopadhyay AK, Johnson LR, Copeland EM, Weisbrodt NW. Effect of secretin on electrical activity of small intestine. *Amer J Physiol* 229:484–488, 1975.
17. Engstrom ER Jr, Webster JG, Bass P. Analysis of duodenal contractility in the unanesthetized dog. *IEEE Trans Biomed Eng* BME-26:517–523, 1978.
18. Bass P, Code CF, Lambert EH. Electric activity of gastroduodenal junction. *Amer J Physiol* 201:587–592, 1961.
19. Gladen HE, Kelly KA. Independence of canine gastric and duodenal pacesetter potentials shown by electric pacing. *Mayo Clinic Proc* 52:51–53, 1977.
20. Gullikson GW, Okuda H, Shimizu M, Bass P. Electrical arrhythmias in gastric antrum of the dog. *Amer J Physiol* 239:G59–G68, 1980.

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