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Research Article

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ABSTRACT The oppossum sphincter of Oddi (SO) exhibits peristaltic spike bursts with accompanying contraction waves that originate proximally in the sphincter of Oddi and propagate toward the duodenum. In this study we recorded myoelectrical activity of the opossum SO and upper gastrointestinal tract in six conscious animals using chronically implanted electrodes. Biopolar electrodes were implanted in the gastric antrum, duodenum, SO segment, jejunum, and ileum. During fasting the frequency of SO spike bursts, scored as number per minute, showed a cyclic pattern consisting of four phases (A to D). Phase A had a low spike burst frequency of ~2/min that lasted ~20 min. In phase B, the spike burst frequency increased progressively during a 40–45 min interval culminating in a short interval of phase C activity characterized by a maximal spike burst frequency of ~5/min. During phase D, the spike bursts decreased over 15 min to merge with the low frequency of phase A and the cycle repeated. Cycle length of the interdigestive SO cycle, 87±11 SD min, was virtually identical with that of the interdigestive migrating myoelectric complex (MMC) of the upper gastrointestinal tract. The onset of phase C activity in the SO began 1–2 min before phase III of the MMC activity in the duodenum. Feeding abolished the cyclic pattern of spike burst activity in the SO as well as in the upper gastrointestinal tract. After feeding the SO spike bursts occurred at a frequency of 5–6/min for at least 3 h. We conclude that: (*a*) During fasting, the oppossum SO exhibits cyclic changes in its spike burst frequency; (*b*) Maximal spike burst frequency of the SO occurs virtually concurrent with passage of phase III MMC activity through the duodenum and; (*c*) Feeding abolishes the interdigestive cyclic spike burst pattern of the SO as well as that of the gastrointestinal tract.

INTRODUCTION

Few studies exist on long-term observations of biliary tract motor activity in conscious animals. For this reason we evaluated sphincter of Oddi myoelectric activity in awake opossums. The opossum was selected as an experimental model because the sphincter of Oddi segment in this species is mostly extraduodenal (1), thereby allowing study with minimal interference from duodenal activity, and also exhibits spontaneous phasic contractile activity (2, 3) similar to that recorded in the sphincter of Oddi of humans (4, 5). The main goals of the study were to: (*a*) record sphincter of Oddi myoelectric activity in awake opossums during fasting and after feeding and (*b*) correlate sphincter of Oddi myoelectric activity with myoelectric activity recorded from the stomach and small bowel.

METHODS

We studied six opossums weighing 2.4–3.7 kg. After overnight fasting each animal was anesthetized with pentobarbital (100 mg i.p.). At laparotomy bipolar electrodes were placed on the sphincter of Oddi (SO),[1] gastric antrum (GA), duodenum (D), jejunum (J), and ileum (I). The electrode consisted of nichrome wire of 120 μm Diam insulated with

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[1] *Abbreviations used in this paper:* GA, gastric antrum; I, ileum; J, jejunum; MMC, migrating motor complex; SO, sphincter of Oddi.

trimel (Johnson Mathey Ltd., Toronto, Canada). A ring of insulation 1.0–1.5 mm in width was scraped off each electrode 3 cm from its free end.

At laparotomy via a midline incision, the opossum SO is easily identified because of its extraduodenal location. On inspection the SO segment is seen as a thick-walled structure that measures 3–3.5 cm in length from its proximal margin at the junction of the common bile duct and pancreatic duct to its entry into the duodenum. Observable rhythmic muscular contrations originate near the proximal margin of the SO and propagate toward the duodenum.

Two pairs of electrodes were sutured into the anterior wall of the SO. One electrode pair was placed 1 cm proximal to the SO-duodenal junction (site SO_1) and the second pair ~1.0 cm more proximal (site SO_2). One pair of electrodes was positioned on the gastric antrum. Two electrode pairs were placed on the duodenum, 1 cm proximal to the SO-duodenal junction (site D_1) and 1 cm distal to the SO-duodenal junction (site D_2). Lastly, a pair of electrodes was positioned on the jejunum, 10 cm distal to the ligament of Treitz, and another pair on the distal ileum, 10 cm proximal to the ileocecal junction.

For electrode implantation, a 25-gauge needle was passed transversely through the outer half of the SO wall or bowel wall at right angles to the long axis of the tubular structure. Next, we positioned the electrode wire within the needle. The needle was then withdrawn and the electrode adjusted so that its bare area lay within the muscularis of the SO or bowel. The short end of the wire was then twisted around the long limb of the electrode, trimmed to 5 mm in length and the free end buried beneath the serosa. Thus, the electrode was in effect sutured in place so that a serosal distance of 2–3 mm was included within the suture. The two electrodes of a pair were positioned 2 mm apart along the circumference of the viscus. After implantation, the ends of the electrodes were passed subcutaneously from the right lateral abdominal wall to the upper back where they were attached to a small female connector. A specially designed cloth jacket prevented the animal from disturbing the connector.

Recording sessions were begun 1–2 wk after electrode implantation. At this time the animals had recovered completely from the effects of surgery. For recording, the male connector of a wire cable, suspended from the top of the animal's cage, was plugged into the interscapular connector. The animal was then free to move about its cage. Wires from the connecting cable led to an 8-channel Beckman polygraph (Beckman Instruments, Inc., Fullerton, CA). Recordings were made using a low-frequency cut-off filter of 0.16 or 5.3 Hz and a high-frequency cut-off filter of 30 Hz. We generally used a paper speed of 1 mm/s, but occasionally selected other paper speeds for demonstration purposes.

During a 2–3-mo interval, 12 to 18 individual recording sessions were obtained in each of the six animals studied. Each recording session lasted 8 h, from ~9 a.m. to 5 p.m. Throughout the study the animals were fed a measured amount of monkey biscuit (3 biscuits/kg) each day at 5 to 6 p.m., while water was available ad lib. With this routine feeding schedule a feeding habitus ensued, whereby interdigestive migrating motor complexes (MMC) were virtually always present 15 h later when recording sessions were begun at 9 a.m. in the morning. For most recording sessions the animals remained fasted. During two 8-h recording sessions in each animal, however, food was given after recording 1 or 2 complete MMC cycles. For these studies the animals were fasted 24 h. The daily number of monkey biscuits was given 10 min after the end of phase III of the MMC was recorded at D_2. After 2 h any uneaten biscuits were removed and the number of biscuits eaten was recorded.

For analysis of the myoelectric recordings, the 8-h tracings were divided into 2-min intervals and the number of spike bursts for each interval determined for each recording site. From representative recordings obtained using the 0.16-Hz low-frequency filter, slow wave frequencies were scored for 10-min intervals. MMC activity in the duodenum and small bowel was divided into four sequential phases according to the method described by Code and Marlett (6). During phase I, spike bursts accompanied 25% of slow waves. Phase II demonstrated irregular spike bursts on 25–90% of the slow waves, whereas regular spike bursts of high amplitude accompanied 90% of slow waves during phase III. During phase IV, spike burst frequency declined rapidly to the low values of phase I. The scoring method was modified slightly to score electrical activity in the gastric antrum because the antral recording never demonstrated a spike burst with each slow wave. During the anticipated interval of phase III for the stomach, the antral recordings showed regular, high-amplitude spike bursts occurring about once a minute on every second or third slow wave. In two animals simultaneous recording of antral contractile activity with a bipolar electrode and extraluminal force transducer showed that the two methods gave virtually an identical frequency of antral contractile activity. Phase I antral activity had no spike bursts, whereas phase II antral activity exhibited sporadic low-amplitude spike bursts that increased in frequency and amplitude to merge with phase III. During phase IV, antral spike burst frequency declined rapidly to become the silent period of phase I. Because the MMC cycles were observed best at site D_2, phase III activity at this site was selected as a reference for MMC cycle length.

Because an established method for scoring SO myoelectric activity was not available, the necessity existed to develop a method for classifying interdigestive SO myoelectric activity. Because SO slow waves were difficult to identify and often not apparent on the myoelectric tracings, we developed a scoring method based solely on the frequency of SO spike bursts. Analysis revealed that ~90% of spike bursts appearing at site SO_2 propagated caudad to SO_1 and spike burst frequency at SO_1 paralleled that at SO_2. We selected the SO_2 tracings for detailed analysis and scored spike burst frequency for each 2-min interval of every tracing.

On inspection, graphs of spike burst frequency for the SO revealed that the frequency of spike bursts in the SO had a rhythmic pattern with four phases (A to D). During phase A, the spike bursts occurred at a frequency <30% of the maximal spike burst frequency during the cycle. During phase B, the spike burst frequency increased gradually from 30 to 90% of maximal and culminated in phase C, which was characterized by a short interval of maximal spike burst frequency. During phase D, the spike burst frequency decreased to reach the low frequency of phase A. The duration of the interdigestive cycle of SO myoelectric activity was measured as the interval between two successive phases. To evaluate the temporal relationship between gastrointestinal MMC activity and SO myoelectric activity, the termination of the phase III of the MMC at site D_2 was used as the frame of reference.

For tracings obtained during and after feeding, spike burst frequency in the SO and gastrointestinal tract was also scored in 2-min intervals. To simplify the plots of postprandial spike burst frequency of the SO scoring of the tracings was also done in 5-min intervals.

Data was analyzed for significant differences using the paired and unpaired Student's t test. We accepted a P value

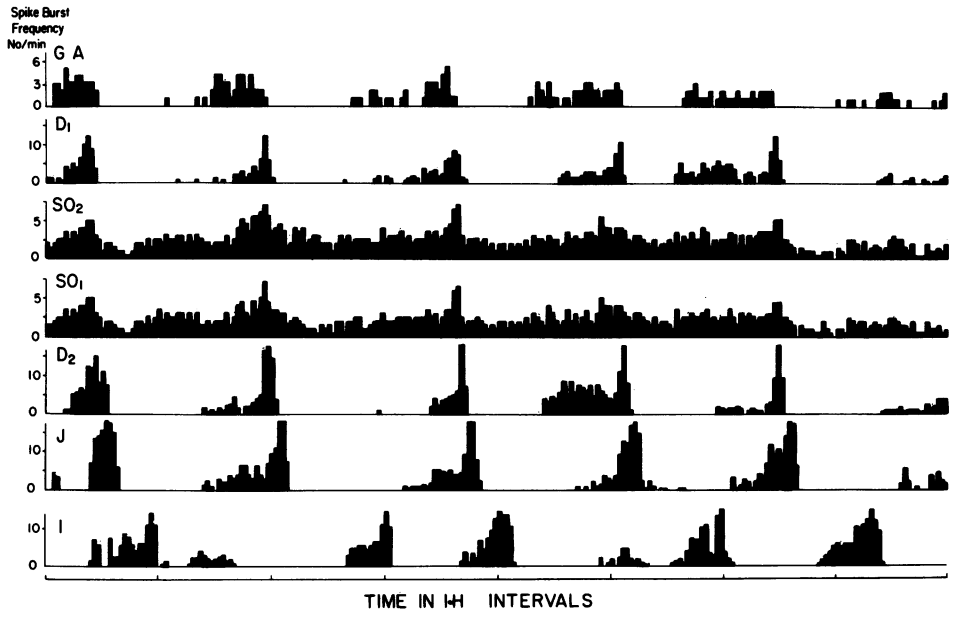

FIGURE 1 Histogram of SO and gastrointestinal tract myoelectric activity in a fasted opossum. Bipolar electrodes are positioned in the GA, duodenum two sites (D_1 and D_2), SO two sites (SO_1 and SO_2), J, and I. The frequency of spike bursts (SB) recorded by each electrode is shown for an 8-h recording. The spike burst frequency is plotted as number per minute. Typical cyclic phase III MMC activity fronts are observed to originate in the stomach and propagate to the ileum. SO spike bursts show a cyclic pattern with maximal activity occurring coincident with passage of phase III of MMC activity through the duodenum.

≤ 0.05 as indicating a significant difference. In the text averaged values are given as mean±1 SD.

RESULTS

Interdigestive myoelectric activity of gastrointestinal tract. In each of the six animals studied, three to six MMC were recorded during each 8-h recording session. For the discernable MMC, 70% began in the stomach and propagated through the duodenum into the small bowel. The remaining 30% either showed feeble activity, demonstrated interrupted activity during phase III, arrested in the gastric antrum, or began at an ectopic location in the duodenum or jejunum. About 20% of the intact MMC originating in the stomach showed sequential propagation of phase III activity from the jejunum to the distal ileum. In the remainder, phase III-type activity was either not recorded in the ileum or did not appear to be in sequence with that in the jejunum. Because only 20% of the gastrointestinal MMC showed coordinated propagation to the ileum the data from the ileum was excluded from additional analysis.

Data from an 8-h recording in one animal is shown in Fig. 1. The cycle length for phase III activity at D_2 for all six animals averaged 87±10 min. The respective duration of MMC phases I to IV recorded at each of the gastrointestinal recording sites is shown in Table I. The duration of phase III activity for the gastric

TABLE I
Interdigestive Gastrointestinal Myoelectric Activity in Six Opossums

	Duration of Phases I to IV			
Site	Phase I	Phase II	Phase III	Phase IV
GA	36.0±12.2	25.8±4.4	25.9±3.5	1.2±1.6
D_1	54.3±10.6	27.9±4.4	4.9±1.0	1.6±1.1
D_2	49.8±9.3	30.1±5.3	6.2±1.6	2.8±1.5
J	51.4±11.0	28.6±6.4	6.3±1.3	3.3±2.0

Values given as mean±1 SD min.

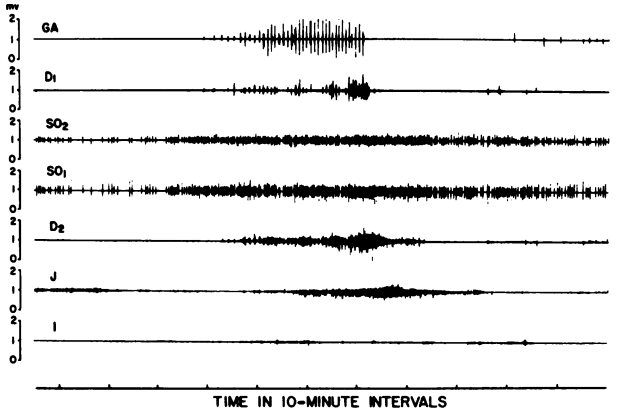

FIGURE 2 Myoelectric spike burst activity in the SO and gastro-intestinal tract of a fasted opossum, recorded at a slow paper speed of 5 mm/min. Electrodes are positioned in the GA, duodenum two sites (D_1 and D_2), SO two sites (SO_1 and SO_2), J, and I. Filtration factors were selected to filter out slow waves. Phase III activity of the interdigestive MMC is seen to pass from the stomach to the jejunum. Spike burst activity of the sphincter of SO, although continuous, intensifies in frequency and reaches a maximal value when the phase III activity moves through the duodenum.

antrum was about five to six times longer than that at any of the other recordings sites. The onset of phase III activity at site D_1 occurred 24±4 min after the onset of phase III activity in the antrum. The time interval between the onset of phase III activity at sites D_1 to D_2 and D_2 to J, respectively, was 2±1 and 7±2 min. Phase III at D_1 usually began 1–2 min before the termination of phase III in the gastric antrum. By inspection, the magnitude of the gastrointestinal spike burst was greater during phase III than during the other phases of the MMC (Fig. 2). Slow waves recorded from the stomach and intestine are shown in Fig. 3. The slow waves in the gastric antrum occurred at a frequency of 4.3±0.5/min. The slow wave frequency at D_1 and D_2, respectively, was 18.4±0.7/min and 17.4±1.1/min. Slow wave frequency averaged 17.4±0.9/min in the jejunum and 16.0±0.8/min in the ileum.

Interdigestive myoelectric activity of the SO. The SO spike bursts demonstrated a rhythmic pattern with periodic increases in spike burst frequency (Fig. 1). The SO spike bursts, however, never disappeared completely as during phase I of the gastrointestinal MMC. From inspection, the pattern of SO spike burst activity was divided into four phases, designated A to D (Fig. 4). Phase A demonstrated a slow spike burst frequency that averaged ~2/min and lasted ~20 min. The second phase, phase B, showed an interval of gradual increase in spike burst frequency, lasting ~40–45 min that culminated in a short interval, phase C, of maximal spike burst frequency in the SO at a rate of 5–6/min. During phase D the spike burst frequency in the

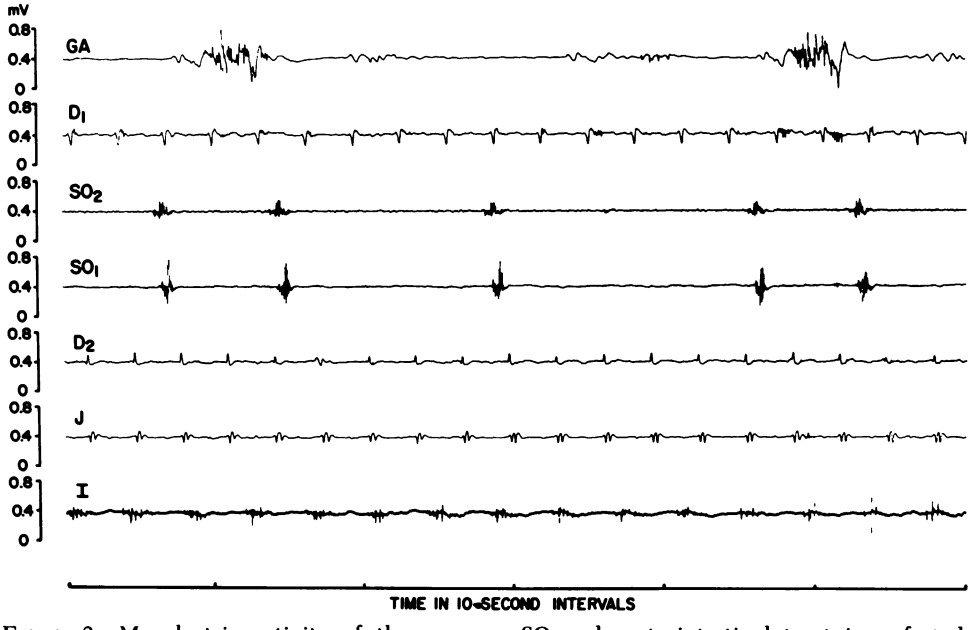

FIGURE 3 Myoelectric activity of the opossum SO and gastrointestinal tract in a fasted animal, recorded at a fast paper speed of 10 mm/s. Electrodes are positioned in the GA, duodenum two sites (D_1 and D_2), SO two sites (SO_1 and SO_2), J, and I. Regular slow waves at characteristic frequencies are observed in the antrum, duodenum, jejunum, and ileum. Spike bursts are observed superimposed on two of the antral slow waves and on all the ileal slow waves. The SO spike bursts are super-imposed on slow waves and propagate as a peristaltic sequence toward the duodenum.

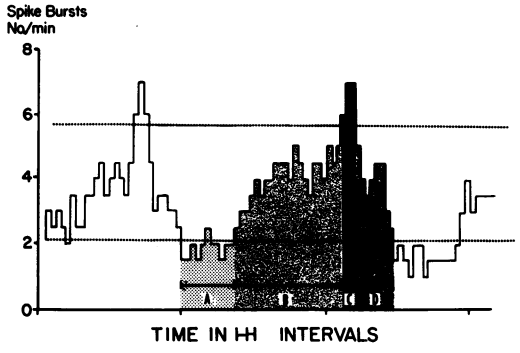

FIGURE 4 Phases of SO spike burst activity during fasting. Spike burst frequency of the SO is plotted for a 3-h interval. The cyclic pattern of SO spike bursts may be divided into four phases. The horizontal dotted lines indicate 30 and 80%, respectively, of maximal spike burst frequency. During phase A, the spike bursts occurred at a frequency ≤30% of maximal spike burst frequency for the cycle. During phase B, the frequency of spike bursts increased gradually from 30-80% of maximal spike burst frequency. During phase D, the frequency of spike bursts decreased to reach the low frequency of phase A and the cycle repeated.

SO rapidly diminished to return to the low spike burst frequency of phase A, thereby completing the cycle. Although not identical, the pattern of interdigestive SO myoelectrical activity was similar in all six of the animals that we studied. For all six animals, A, B, C, and D, respectively, exhibited spike bursts at a frequency of 2.1±0.9, 3.1±1.1, 5.1±1.5, and 3.2±1.5 SD/min. The durations of the four phases of SO myoelectric activity was 23±3 min for A, 43±10 min for B, 6±2 min for C, and 15±4 min for D. The cycle length from phase C to C for all six animals averaged 87±11 min. On visual inspection, no differences in the magnitude of the SO spike were observed among the four phases of interdigestive SO myoelectric activity (Fig. 2).

Relationship of SO and gastrointestinal myoelectric activity. A close relationship existed between the phasic patterns of interdigestive migrating myoelectric activity in the SO with that in the upper gastrointestinal tract (Figs. 1 and 2). The cycle interval for maximal spike burst activity in the SO (87±11 min) was virtually identical to the cycle interval of MMC in the duodenum (87±10 min). The onset of phase B activity in the SO began about the same time as the onset of phase II activity in the gastric antrum. The maximal spike burst activity of phase C in the SO, however, did not occur until 22±9 min after the onset of phase III activity in the antrum and just a few minutes (2.1±3.9 min) before phase III activity appeared at site D_1 in the duodenum. This small difference in time was statistically significant ($P < 0.01$), however, phase C activity of the SO substantially overlapped the phase III activity of the duodenum (Fig. 5). This close correlationship of SO phase C activity and phase III activity of the MMC in the duodenum persisted when the MMC cycle originated in the duodenum, while no phase III pattern developed in the gastric antrum.

Effect of feeding on SO and gastrointestinal myoelectric activity. Feeding abolished the interdigestive cycles of migrating myoelectric activity in the SO as well as in the gastrointestinal tract. An example for one animal is shown in Fig. 6. After the onset of feeding, irregular spike bursts developed in the stomach and small bowel. Feeding rapidly increased the frequency of spike bursts in the SO to ~5/min and this frequency persisted for a minimum of several hours (Fig. 7). Feeding caused no apparent change in the magnitude of SO spike bursts. The high spike burst frequency of the SO after feeding was almost identical to the spike burst frequency of phase C during fasting. About 1–2 h after feeding, MMC activity often began to appear in the duodenum and jejunum, recognized by intervals of high-amplitude regular spike bursts interrupted by periods of quiescence (Fig. 6). The cycle length of these ectopic MMC, 57±7 min, was significantly shorter than that of the nonectopic MMC present during fasting, 87±10 min ($P < 0.001$).

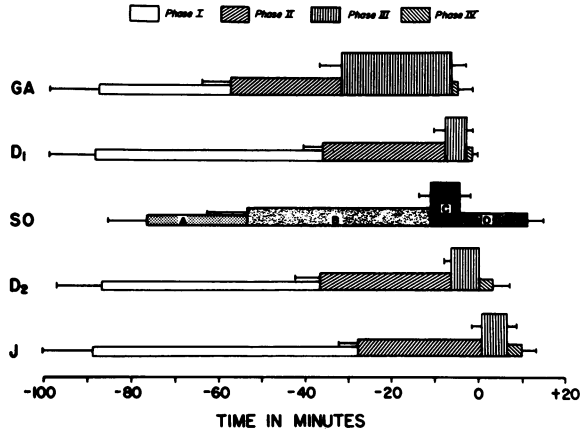

FIGURE 5 Relationship of SO and gastrointestinal myoelectric activity. Composite of data from six animals. The interval of the four phases of MMC activity is shown for the GA, two sites in the duodenum (D_1 and D_2) and J. The horizontal T indicate one SD. The intervals of the four phases of interdigestive SO spike burst activity are also shown. The reference point zero indicates termination of MMC phase III activity at D_2. Phase C of maximal spike burst frequency of the SO is seen to develop ~2–3 min before the onset of the phase III activity in the proximal duodenum (D_1). Then the SO phase C and duodenal phase III activity overlap and are coincident.

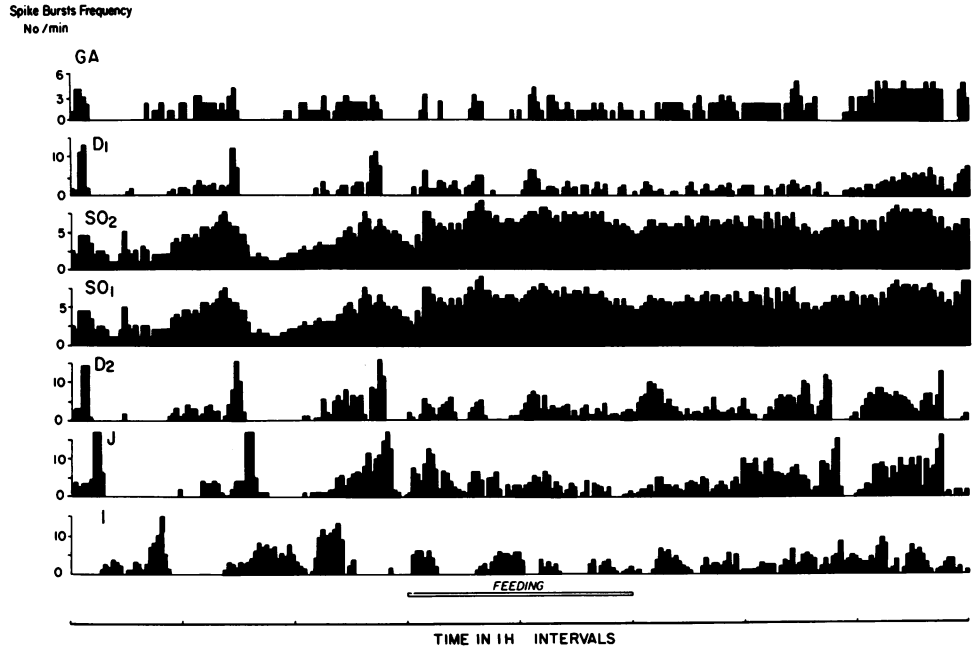

FIGURE 6 Histogram of SO and gastrointestinal myoelectric activity in an opossum before and after feeding. Bipolar electrodes are positioned in the GA, duodenum two sites (D_1 and D_2), SO two sites (SO_1 and SO_2), J, and I. Spike burst frequency is plotted as number per minute. Feeding converts the cyclic MMC pattern of the stomach, duodenum, and small bowel to a continuous fed pattern. Feeding also disrupts the cyclic pattern of spike burst activity in the SO. Within a few min after the onset of feeding SO spike burst frequency increased to approximate the maximal values of 5–6/min observed during fasting. This increase persisted for 5 h at which time the recording session ended. 2 h after feeding cyclic MMC activity is observed at D_2 and J, however, the interval for phase III cycle length is shorter than the cycle length before feeding.

DISCUSSION

Our aim in this investigation was to evaluate SO motor activity in conscious animals during fasting and after feeding. The experimental model developed for the study allowed repeated long-term recordings of SO and gastrointestinal myoelectric activity in a chronic opossum preparation. Although numerous previous reports describe gastrointestinal myoelectric activity in conscious chronic animals, (6–9) we are not aware of any previous study that compares SO myoelectric activity and gastrointestinal myoelectric activity in a chronic animal preparation.

Our study findings demonstrate that: (*a*) A characteristic pattern of gastro-intestinal MMC activity exists in the fasted opossum, (*b*) During fasting the opossum SO demonstrates cyclic changes in spike burst frequency, (*c*) Maximal frequency of the SO spike bursts occurs nearly concurrently with passage of phase III MMC activity through the duodenum, and (*d*) Feeding abolishes the cyclic pattern of spike burst frequency of both the SO and upper gastrointestinal tract.

The opossum gastrointestinal tract exhibits a fasting MMC pattern that is comparable to that recorded in other mammalian species. The average cycle length of 87 min between phase III MMC activity fronts is similar to that reported for the dog (6, 7, 9, 10). The pattern of interdigestive MMC activity in the opossum gastric antrum is of interest. In contrast to phase III of the gastric MMC activity front in the dog and many other animals (7, 8), phase III of MMC activity in opossum gastric antrum did not exhibit a spike burst associated with each antral slow wave. Simultaneous antral recordings with extraluminal strain gauges and electrodes in two animals showed identical frequencies of antral contractile activity recorded by the two different methods. In the opossum the maximal antral spike burst frequency was 1–2 min compared to the antral slow wave frequency of ~4/min.

At present most investigators agree that the SO musculature in most animal species is anatomically distinct from the musculature of the duodenum and contracts independently of duodenal motor activity (1). For this study we selected the opossum because its SO is mainly

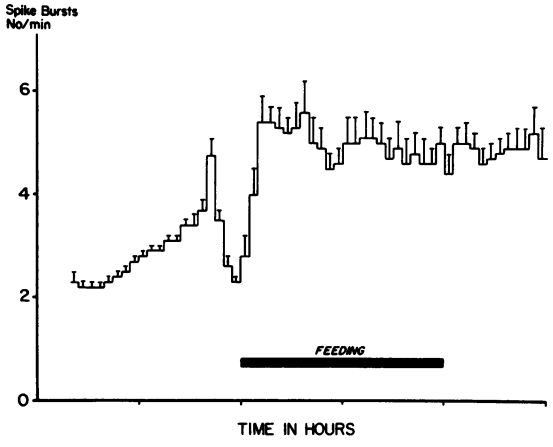

FIGURE 7 SO spike burst frequency before and after feeding. Composite data from six animals. The data is plotted from mean values and the vertical T indicates one SE. Prior to feeding, phases A to D of the interdigestive myoelectric pattern of the SO are observed with the maximal value of ~5/min occurring during phase C. Food was presented to the animal during phase A of minimal SO spike burst activity of ~2/min. Within 5–10 min after presentation of food, the SO spike bursts began to increase in frequency and within 15 min reached a plateau value of 5–6 per min. This high frequency of SO spike bursts continued unchanged for ~5 h at which time the recording sessions ended.

extraduodenal, thereby permitting easy monitoring of SO contractile activity without duodenal interference (3). Additionally, the opossum SO exhibits forceful phasic contractile activity similar to that in the SO of humans (4, 5).

Myoelectric recordings of the opossum SO showed slow waves as well as spontaneous spike bursts. Unlike the obvious regular slow wave activity recorded in the stomach, duodenum, and small bowel, the SO slow waves were difficult to identify, appeared to vary in frequency, and at times were not evident. These difficulties in recording SO slow waves may be related to limitations of our recording method. However, the electrodes we used did give excellent recordings of gastrointestinal slow waves. At present the issue of SO slow waves is under further investigation in our laboratory.

Our study findings demonstrate that the phase C interval of maximal SO activity begins toward the end of phase III of MMC activity in the stomach and virtually coincident with phase III activity in the duodenum. In most respects the interdigestive spike burst activity of the SO is comparable to phases of the MMC that occur in the duodenum. For example, phases B, C and D of SO myoelectric activity correspond closely in timing and character to phases II, III, and IV of MMC activity, respectively, in the duodenum. An important distinction, however, is that the duodenum is quiescent during its phase I, whereas the SO always has repetitive activity during phase A.

Findings from recent studies indicate that biliary and pancreatic secretory function are coordinated with gastrointestinal MMC activity in humans and dogs (11–13). The volume of biliary and pancreatic juices entering the duodenum increases significantly immediately before the phase III activity of the MMC sweeps through the duodenum. Further, the concentration of bile acids and pancreatic enzymes increases during the interval of increased biliary and pancreatic flow. For these reasons, we anticipated that during fasting a relationship might exist between the pattern of SO myoelectric activity and duodenal myoelectric activity. Consequently, the close association between maximal contractile activity of the SO and that of the duodenum was not surprising. Two important unanswered questions, however, merit consideration: (*a*) What is the potential physiological role of enhanced SO activity coincident with phase III activity of the MMC in the duodenum? and (*b*) What mechanism(s) coordinates these two activities?

A recent study from our laboratory indicates that the rhythmic spike burst activity in the opossum SO is peristaltic in character and migrates toward the duodenum (14). The spike burst activity invariably originates near the proximal margin of the SO and generally propagates over the entire length of the Oddi segment. This spike burst activity is the electrical equivalent of a peristaltic pressure wave that on cine recordings actively expels contrast medium from the SO segment into the duodenum (14). During the diastolic interval between SO peristaltic sequences, the sphincter segment refills by passive inflow from the common bile duct. Thus, the opossum SO segment in the opossum appears to act primarily as a peristaltic pump that actively transports SO contents into the duodenum, whereas emptying of the common bile duct into the SO occurs as a passive phenomenon determined by the small positive pressure gradient that exists between the common bile duct and duodenum. An increase in the frequency of SO peristaltic sequences within the physiological range would tend to enhance emptying of the SO segment and perhaps augment protective mechanisms against duodenal-SO reflux.

Although the basic apparatus that generates SO peristaltic contractions is myogenic in origin (14), the mechanism(s) that control contraction frequency are not yet determined. Some preliminary evidence suggests that SO contraction frequency in the opossum is augmented by lumenal distension (unreported observations). Consequently, changes in biliary or pan-

creatic flow could potentially regulate the frequency of SO contractions by producing subtle changes in SO distension.

An increase in hepatic bile flow concurrent with duodenal phase III activity could possibly distend the SO segment sufficiently to produce a maximal frequency of SO contractions. Such an explanation would presume that bile flow increased progressively during phase B of interdigestive myoelectric activity of the SO. Common bile duct outflow might also be augmented by gallbladder contraction. A second possibility is that neural pathways activated during phase III of duodenal MMC activity might excite contractile activity in the SO segment.

Lastly, hormonal influences might possibly coordinate the fasting pattern of myoelectric activity in the duodenum and SO. In the dog and human, motilin has been shown to be an important factor for initiating MMC activity in the stomach and duodenum (15, 16). Motilin also initiates MMC activity in the opossum gastrointestinal tract (unreported observations). The effect of motilin on the opossum SO remains to be determined. CCK increases the frequency of SO contractile activity in the opossum (17), similar to its effect on the rabbit SO (18); however, CCK does not elicit gastrointestinal MMC activity.

Feeding abolished the cyclic pattern of interdigestive myoelectric activity in the opossum SO. Food was presented to the animals during the quiescent phase I interval of the MMC cycle in the duodenum. A significant increase in SO spike burst frequency developed 5–10 min after the animals were given food. The spike burst frequency of the SO reached a maximal value of 5–6/min within 15 min. This rate of spike bursts in the SO was comparable to the maximal frequency of spike bursts that occurred during phase C of the fasting cycle. The rapid increase of SO spike bursts after exposure to food, e.g. within 5–10 min, seems to favor a neural rather than a hormonal mechanism.

Similar to the findings reported in humans, dogs, and pigs (8, 9, 19, 20), feeding disrupted the normal cyclic MMC pattern in the opossum. The MMC pattern began to reappear 2–3 h after eating as activity fronts originating in the mid-duodenum or proximal jejunum, while a fed pattern continued in the stomach, proximal duodenum and SO for at least 3 h at which time the observations were terminated. Duration of MMC disruption after eating has been shown to depend on the volume and composition of food intake (9). In the dog, similar to the opossum, the initial MMC activity that returns after eating occurs as ectopic fronts in the jejunum and is independent of serum motilin concentration (21).

We conclude that the opossum is a suitable experimental model for obtaining long-term recordings of SO myoelectric activity in conscious animals. During fasting the opossum SO shows cyclic changes in spike burst frequency that correlate with passage of the MMC activity through the duodenum. feeding converts SO myoelectric activity to a fed pattern of maximal spike burst frequency. The functional significance of alterations in the frequency of SO spike bursts and the physiological mechanisms that control this activity remain to be determined.

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REFERENCES

1. Boyden, E. A. 1937. The sphincter of Oddi in man and certain repesentative mammals. *Surgery (St. Louis).* **1:** 25–37.
2. Shelhamer, J., and R. K. Goyal. 1973. Physiology of bile transport: manometric studies of common bile duct and sphincter of Oddi. *Gastroenterology.* **64:** 686 (Abstr.)
3. Toouli, J., W. J. Dodds, R. Honda, and W. J. Hogan. 1981. The effect of histamine on the motor function of the opossum sphincter of Oddi. *Am. J. Physiol.* **241:** G122–G128.
4. Csendes, A., A. Kruse, P. Funch-Jensen, M. J. Oster, J. Ornsholt, and E. Amdrup. 1979. Pressure measurements in the biliary and pancreatic duct systems in controls and in patients with gallstones, previous cholecystectomy, or common bile duct stones. *Gastroenterology.* **77:** 1203–1210.
5. Geenen, J. E., W. J. Hogan, W. J. Dodds, E. T. Stewart, and R. C. Arndorfer. 1980. Intraluminal pressure recording from the human sphincter of Oddi. *Gastroenterology.* **78:** 317–324.
6. Code, C. F., and J. A. Marlett. 1975. The interdigestive myo-electric complex of the stomach and small bowel of dogs. *J. Physiol. (Lond.).* **246:** 289–309.
7. Szurszewski, J. H. 1969. A migrating electric complex of the canine small intestine. *Am. J. Physiol.* **217:** 1757–1763.
8. Ruckebusch, Y. and L. Bueno. 1977. Migrating myoelectrical complex of the small intestine—an intrinsic activity mediated by the vagus. *Gastroenterology.* **73:** 1309–1314.
9. De Wever, I., C. Eeckhout, G. Vantrappen and J. Hellemans. 1975. Disruptive effect of test meals on interdigestive motor complex in dogs. *Am. J. Physiol.* **235:** E661–665.
10. Itoh, Z., S. Takeuchi, I. Aizawa, and R. Takayanagi. 1978. Characteristic motor activity of the gastrointestinal tract in fasted conscious dogs measured by implanted force transducers. *Am. J. Dig. Dis.* **23:** 229–238.
11. Peeters, T. L., G. Vantrappen, and J. Janssens. 1980. Bile acid output and the interdigestive migrating motor complex in normals and in cholecystectomy patients. *Gastroenterology.* **79:** 678–681.

12. Keane, F. B., E. P. DiMagno, R. R. Dozois, and V. L. Go. 1980. Relationships among canine interdigestive exocrine pancreatic and biliary flow, duodenal motor activity, plasma pancreatic polypeptide and motilin. *Gastroenterology*. **78:** 310–316.
13. Itoh, Z., and I. Takahashi. 1981. Periodic contractions of the canine gallbladder during the interdigestive state. *Am. J. Physiol.* **240:** G183–189.
14. Toouli, J., R. Honda, W. J. Dodds, W. J. Hogan, S. Sarna, M. S. Egide, and R. C. Arndorfer. 1980. Manometric, electric and flow properties of the opossum sphincter of Oddi. *Dig. Dis. Sci.* **25:** 719 (Abstr.)
15. Itoh, Z., R. Honda, K. Hiwatashi, S. Takeuchi, I. Aizawa, R. Takayanagi, and E. F. Couch. 1976. Motilin-induced mechanical activity in the canine alimentary tract. *Scand. J. Gastroenterol. (Suppl.).* **11** (39): 93–110.
16. Vantrappen, G., J. Janssens, T. L. Peeters, S. R. Bloom, N. D. Christofides, and J. Hellemans. 1979. Motilin and the interdigestive migrating motor complex in man. *Dig. Dis. Sci.* **24:** 497–500.
17. Honda, R., J. Toouli, W. J. Dodds, and W. J. Hogan. 1981. Effect of enteric hormones on sphincter of Oddi (SO) in fasted and fed animals. *Gastroenterology*. **80:** 1179 (Abstr.)
18. Sarles, J. C., J. M. Bidart, M. A. Devaux, C. Echinard, and C. Castaginini. 1976. Actions of cholecystokinin and caerulein on the rabbit sphincter of Oddi. *Digestion*. **14:** 415–423.
19. Vantrappen, G., J. Janssens, J. Hellemans, and Y. Ghoos. 1977. The interdigestive motor complex of normal subjects and patients with bacterial overgrowth of the small intestine. *J. Clin. Invest.* **59:** 1158–1166.
20. Ruckebusch, Y., and L. Bueno. 1976. The effect of feeding on the motility of the stomach and small intestine in the pig. *Br. J. Nutr.* **35:** 397–405.
21. Poitras, P., J. H. Steinbach, G. VanDeventer, C. F. Code, and J. H. Walsh. 1980. Motilin independent ectopic fronts of the interdigestive myoelectric complex in dogs. *Am. J. Physiol.* **239:** G215–220.