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PROPAGATION OF GIANT MIGRATING CONTRACTIONS BETWEEN THE SMALL INTESTINE, CECUM AND COLON DURING RADIATION

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Abstract

Background—Radiation increases the frequency of small intestinal and colonic giant migrating contractions (GMCs). These contractions contribute to the diarrhea and cramping after radiation therapy and are coordinated with one another across the ileocolonic (IC) junction.

Methods—We investigated the coordination of contractile activity between the small intestine, cecum and colon in 5 canines following circumferential myotomy on the ileum at the IC junction and compared it to intact animals. Studies were performed before and during a radiation schedule.

Key Results—Myotomy increased the frequency of small intestinal GMCs prior to irradiation. In intact animals, the duration and amplitude of cecal GMCs decreased when multiple contractions occurred in rapid succession. This is in contrast to small intestinal and colonic GMCs and suggests a different mechanism of propagation for GMCs within the cecum. Ileal myotomy dramatically decreased the frequency of propagating radiation induced colonic GMCs. The total number of colonic GMCs was not altered. Colonic contractile activity was disrupted in intact animals during irradiation. However, after ileal myotomy, irradiation did not affect the pattern of colonic contractile states. Diarrhea in irradiated animals with myotomy started earlier than intact animals. This may be related to the frequency of small intestinal GMCs.

Conclusions & Inferences—Our findings suggest importance of the enteric neural connections at the IC region to contractile disorders of both the small and large intestine. The anatomic relationship between the canine IC junction is similar to the human ileo-appendiceal-colonic region and surgical manipulations of this area may likewise affect human contractile activity.

Keywords

ileocolonic junction; MMC; motility; radiation therapy; surgery

INTRODUCTION

When the gastrointestinal tract is exposed to ionizing radiation, small intestinal (1,2,3) and colonic (4,5) motility are significantly altered and the changes correlate with gastrointestinal symptoms. Increased frequency of GMCs is one manifestation of radiation injury and these contractions are associated with abdominal cramping and diarrhea (6,7,8). At the canine IC junction, the small intestine is in continuity with the proximal colon and the cecum is a diverticulum of the proximal colon, much like the human appendix. The cecum is the only portion of the dog's gastrointestinal tract that exhibits frequent and spontaneous GMCs (9). One to 4 cecal GMCs occur each hour and empty it of feces. Normally, GMCs of the ileum and proximal colon are not synchronized with one another. However, small intestinal GMCs are frequently followed by cecal giant contractions (9). In contrast, most radiation-induced small intestinal GMCs propagate across the IC junction and terminate in the mid colon (4). This contributes to radiation induced diarrhea by rapidly propelling pancreatico-biliary secretions and undigested food into the colon. When GMCs occur in the distal colon, they are associated with defecation (10,11, 12). The functional relationship between the small intestine, cecum and colon during pathologic contractile activity is incompletely understood.

We studied the role of myoneural control of coordination across the IC junction. The objective of our study was to define the relationship between altered small intestinal, cecal and colonic motor activity during irradiation. Our hypothesis was that an intrinsic network exists at the level of the IC junction and coordinates motor patterns between the small intestine, cecum and colon. Interruption of this pathway would interfere with radiation-induced changes in colonic motility.

MATERIAL AND METHODS

Experiments were performed on 10 healthy mongrel dogs (20 – 25 kg). Surgery was performed under general pentobarbital sodium anesthesia (35 mg/kg) through a laparotomy. Five dogs had no surgical alteration of the IC junction and 5 underwent a circumferential myotomy on the ileum at the ileocolonic junction at the time of instrumentation. Myotomy was performed to transect intrinsic myoneural connections between the ileum, cecum and colon while avoiding potential damage to adjacent structures that reanastomosis may have produced.

Strain gauges were oriented to record circular muscle contractions with 2 placed on the ileum (100 cm and 20 cm proximal to the IC junction); 3 on the cecum, (3 – 5 cm apart) and 7 additional gauges equidistant along the colon (intact 8.4 ± 0.7 cm apart, myotomy 7.4 ± 0.5 cm). The first colonic strain gauge was 5 cm distal to the IC junction and the last was at the rectum with no difference in the distance between strain gauges between groups. The lead wires were brought out through a stainless steel cannula in the abdominal wall (2) and dogs were allowed 7 – 10 days to recover.

Myotomy technique was validated on 4 additional dogs using formalin-fixed, H&E transmural sections of the IC junction to confirm completeness. The purpose of the myotomy was to disrupt the myenteric plexus while preserving the submucosal plexus. All

experiments were accomplished within 6 weeks of the myotomy to avoid neural re-growth through the surgical zone.

After an overnight fast, recordings were made for 8 h on a 12-channel polygraph (model 7, Grass, Quincy, MA). The lower and upper cutoff frequencies were set at direct current and 15 Hz, respectively. During 2 control experiments, each dog underwent mock irradiation which involved transporting the animal to the area of radiation, administering 10–15 mg/kg of sodium thiamylal to achieve 5 – 10 min of anesthesia followed by recordings.

Irradiation was administered over 2 weeks and all recordings were made in the fasting state on the days of radiation (2,4). Lightly anesthetized dogs were irradiated using parallel-opposed lateral fields with a 250-kVp orthovoltage x-ray machine filtered to achieve a HVL of 0.9 mm Cu. The focus-to-surface distance was 62 cm, and the field size at the surface of the animal was 21 × 32 cm. The dose rate of the midline of the dog was between 75 and 84 cGy/min, being lowest for the thickest animals. Dogs received 6 doses of 250 cGy in 3 fractions/week on alternate days for 2 successive weeks for a total of 1500 cGy. Radiation dosimetry was based on the measured contours of dogs and ionization chamber measurements were done in a water phantom. The midline absorbed dose was 250 cGy/treatment, with the variation in the dose across the body < ± 7%. Radiation was administered at the same time every morning and transport from radiation to recording was no greater than 30 minutes.

Recordings of small intestinal GMCs (8,14) and migrating motor complexes (MMCs, 14,15) were analyzed visually. MMC cycle length was measured from the end of one phase III activity to the end of the next. Phase III was defined as a group of contractions occurring at maximum frequency, lasting 4 – 8 minutes migrating caudal. Amplitude ratio was defined as the ratio of the amplitude of the GMC to the mean maximum amplitude of contractions during the preceding phase III at the same recording site. In the cecum, since no phase III exists (9), the GMC amplitude was reported as grams of force with each strain gauge calibrated prior to implantation. Cecal contractile activity consisted of alternating quiescence and contractions. A contractile state consisted of a burst of contractions lasting for at least 2 min and 2 contractile states were considered different if there was quiescence of 2 min between them. Cecal GMCs were identified visually (9) as single contractions with an amplitude greater than 1 ½ the maximum amplitude of phase III contractions in the ileum or were immediately preceded by an ileal GMC and possessing a characteristic sharp upstroke.

Colonic motor activity at each recording site consisted of alternating quiescence and contractions. A contractile state consisted of a burst of contractions that lasted for at least 2 min and were considered different if there was a quiescent period of 2 min between them. The onset of a contractile state was compared with the start of the closest contractile state at adjacent recording sites to determine the direction of migration. Contractile states were called a colonic migrating motor complex (CMMC) if it migrated in a caudal or orad direction over at least half the length of the colon (11,12) or 4 consecutive transducers in this study. All CMMCs consisted of propagated contractile states, but not all contractile states produced CMMCs. The total duration/hour was determined by adding up the duration of all contractile states and dividing by the duration of the experiment, 8 hours each (11,12).

Colonic GMCs were identified visually (11,12) as contractions with an amplitude greater than twice the maximum amplitude of colonic phasic contractions, a duration of at least 30 sec and a characteristic sharp upstroke. Like MMCs, the colonic GMC is an all-or-none phenomenon. Occasionally, giant contractions were observed only at a single recording site. Because of the distance between the sites, we assumed that these contractions migrated less than the distance between recording devices. In searching for propagation of GMCs across the IC junction, we arbitrarily set the lag time between a contraction at the last small intestinal strain gauge and the first colonic strain gauge at one minute.

The data were analyzed by Fisher's protected LSD test following ANOVA. Intact dogs were compared to myotomy dogs using unpaired t-tests. All values are expressed as a mean $\pm$ SE. The mean values were determined for each experiment, from which a mean value was determined for each dog. These mean values were then used to determine the overall mean value and the SE. A p value of <0.05 was considered statistically significant. All the studies were done under approval of the Medical College of Wisconsin and Zablocki Veterans Affairs Medical Center Institutional Animal Care and Use Committee review boards and in compliance with the National Research Council's "Guide for the Care and Use of Laboratory Animals."

RESULTS

Histologic Confirmation of Myotomy

Formalin-fixed H&E transmural sections of each of 4 histology dogs revealed the myotomy of the ileum at the IC junction had complete circumferential surgical removal of serosa and muscularis propria which included the myenteric and deep muscular plexus. In addition, the external submucosa displayed minor damage. The internal submucosa, muscularis mucosa, lamina propria and epithelium were intact and unremarkable. Because the submucosa is composed of loose connective tissue, processing artifact occurred and the exact amount of excised submucosa can only be approximated (Figure 1). Although morphology does not infer function, the internal submucosa plexus and mucosa remained intact. No animal developed intestinal perforation due to the procedure and no perimyotomy sepsis was identified at sacrifice.

Small Intestinal, Cecal and Colonic Contractions Prior to Irradiation

Small Intestine—MMC frequency, cycle length, and duration of phase III, were not altered by myotomy (Table 1). Prior to irradiation, animals with ileal myotomy had an increased frequency of GMCs (myotomy $0.263 \pm 0.105/\text{h}$; intact $0.013 \pm 0.013/\text{h}$, $p<0.05$). The amplitude ratio, duration and velocity of migration of the GMCs were not different between the two groups (Table 2). In the intact group, a single small intestinal GMC was identified during 80 recording hours from 5 animals (Figure 2A). That GMC migrated from the small intestine, to the cecum and into the colon. Following myotomy in the preirradiated state, 21 small intestinal GMCs occurred and one migrated into the cecum but not the colon (Figure 2B).

Cecum—In the normal fasted state, the frequency of cecal phasic contractions ($0.74 \pm 0.20/\text{h}$) not associated with a GMC was unchanged by myotomy ($0.91 \pm 0.13/\text{h}$). The mean duration of these phasic contractions was also not altered by myotomy (4.9 ± 0.8 min; myotomy 4.7 ± 0.4 min). GMCs that originate and end within the canine cecum occurred at the same frequency with or without myotomy ($1.3 \pm 0.4/\text{h}$; myotomy $0.8 \pm 0.3/\text{h}$). In the control state prior to radiation, 51% of cecal GMCs occurred in clusters of 2 or more. Following myotomy, this phenomenon accounted for only 35% of the cecal GMCs ($p < 0.05$). The mean duration of GMCs isolated to the cecum was unchanged after myotomy (Table 3).

Colon—Prior to irradiation, the mean duration of colonic contractile states with or without myotomy remained unchanged in the proximal, mid or distal colon. The total duration of colonic contractile states was longer in the distal colon after myotomy but unchanged in the proximal and mid colon. The number of CMMCs in dogs with myotomy was not different from the intact state (intact $0.96 \pm 0.10/\text{h}$, myotomy $1.05 \pm 0.06/\text{h}$). Eleven colonic GMCs originated within the colon and traveled 11.7 ± 1.7 cm in the intact dogs during control experiments. In contrast, 16 GMCs originated in the colon and traveled a significantly shorter distance (7.0 ± 0.4 cm, $p < 0.05$) during control experiments after myotomy.

Small Intestinal, Cecal and Colonic Contractions in Irradiated Dogs

Small Intestine—MMC frequency, cycle length and phase III duration were unaltered by radiation (Table 1). There was a dramatic increase in small intestinal GMCs in all animals after irradiation ($p < 0.05$, see Figure 2). There was a 50% increase in the frequency of irradiation induced small intestinal GMCs in myotomy animals (intact $0.62 \pm 0.15/\text{h}$, myotomy, $0.97 \pm 0.21/\text{h}$). The amplitude ratio, duration and velocity of migration of GMCs were not different between the groups (Table 2). There was a significant difference in the frequency of GMCs that propagate from the small intestine to the colon between the groups. In irradiated dogs with an intact IC junction (Figure 3), 65% of all small intestinal GMCs migrated into the cecum and 60% of those GMCs (58 GMCs) continued into the colon. In contrast, following myotomy, only 1% of small intestinal GMCs migrated from the terminal ileum to the cecum and none continued into the colon (Figure 4).

Cecum—During the days of radiation exposure, the mean duration of cecal phasic contractile states decreased from control values (4.9 ± 0.8 min) to 3.9 ± 0.5 min but was not different from animals with a myotomy (4.5 ± 0.6 min). The frequency of cecal phasic contractions was significantly less in irradiated intact animals than what was observed after myotomy (intact 0.35 ± 0.06 contractile groups/h, myotomy 0.86 ± 0.10 contractile groups/h; $p < 0.05$). The number of cecal phasic contractions in myotomy animals was unchanged when compared to preirradiation values.

As prior to irradiation, myotomy decreased the likelihood of cecal GMCs occurring in clusters of two or more (intact 57.8%, myotomy 28.3%, $p < 0.05$). The number of cecal GMCs/h following myotomy was significantly decreased (Table 3). Prior to irradiation in intact animals, 1/104 cecal GMCs originated in the small intestine, increasing to 97 out of 556 (18%) following radiation exposure. The duration of these GMCs was less than GMCs

originating in the cecum (cecum only, 0.70 ± 0.03 min, origin in small intestine 0.51 ± 0.04 min, $p < 0.05$).

Colon—The frequency of CMMCs during the radiation schedule was not different when myotomized dogs were compared to intact dogs (intact 0.99 ± 0.18 CMMCs/h, myotomy 0.51 ± 0.15 CMMCs/h). Myotomy normalized the mean duration of colonic contractile states following irradiation in the mid and distal colon. (Figure 5). The increased frequency of radiation induced GMCs interrupted normal colonic contractile activity in intact animals while myotomy disrupted the GMCs, allowing normal duration of colonic contractile states. The same pattern was present in the total duration of colonic contractile states.

The frequency of radiation induced colonic GMCs was not different when intact dogs were compared to dogs that had undergone myotomy (Figure 2A–B). In intact animals, 17/128 colonic GMCs (13%) occurred at only one recording site. Myotomy significantly increased the number of colonic GMCs that occurred at a single recording site (43/93 colonic GMCs; 46%). The mean distance of propagation for GMCs was not different between intact and myotomy animals (intact 14.3 ± 2.1 cm, myotomy 14.6 ± 3.3 cm) but significantly fewer GMCs propagated in myotomy animals. In the intact state, 48% of all colonic GMCs terminated in the mid colon. Following myotomy, only 23% of GMCs isolated to the colon propagated ($p < 0.05$).

After myotomy, there was a lag time of 48 sec between the end of the GMC in the last ileal strain gauge and the beginning of the colonic GMC in the first colonic strain gauge in a single GMC. This was greater than 2 standard deviations longer than the time lag of 35.7 ± 4.1 sec between the same points in the irradiated intact animals and may represent a random occurrence. The GMC that migrated from the ileum to the colon in an animal prior to irradiation had a lag time of 34 seconds. This value falls within the range of GMC lag times occurring in the same group of intact animals after irradiation.

Symptoms with Irradiation

Prior to radiation exposure, all of the dog's feces were formed with no significant difference between the intact and myotomy animals. No dogs exhibited watery diarrhea. The incidence of unformed feces was 20% during radiation exposure in intact dogs by week 2 of irradiation but affected 100% of the dogs that had undergone myotomy by the second week of irradiation.

All myotomy and intact dogs exhibited anorexia during radiation exposure. No dog lost greater than 10% of its original body weight. Some dogs experience mild discomfort during GMCs. The frequency of defecation between intact and myotomy animals was not different.

DISCUSSION

The functional relationship between the ileum, cecum and colon has been focused on short bowel syndrome or issues related to bowel transplantation. Our study demonstrates that abnormal contractile patterns within the small intestine may influence motility of the cecum and colon. Previous studies have identified the IC junction and ileocecal valve as a region of

unique contractile events. Quigley et al (17) looked at motor activity in the distal small intestine of a canine model and demonstrated distinct patterns. A high degree of contractile coordination between the distal ileum, IC sphincter, and proximal colon was recognized but these observations were limited to normal contractile activity. These patterns of motor activity suggested a specialized function for this region. Quigley et al (18) suggested that transit of contents from the ileum to the colon had the potential to modulate the colon's ability to compensate for an increasing fluid load. He noted that prolonged propagated contractions or GMCs occurred in the ileum of humans. This study supports a role of GMCs in radiation induced diarrhea.

Thompson et al (19) noted that extensive resections of the distal small intestine are associated with motor disruption in the proximal remnant. They hypothesized that resection of the distal ileum through loss of the receptor site for either inhibitory reflexes or bile salt absorption determined the motor response to resection. Our data suggests that alteration of the inhibitory reflexes is a likely cause. We avoided the use of resection to simplify the study of the intrinsic myoneural link between the ileum, cecum and colon. Thus, although the absorptive surface and luminal contents remain the same, the motor pattern of the colon and small intestine are altered by separating their myoneural connections with a myotomy. With myotomy the small intestine exhibited an increased frequency of GMCs, while the colon displays fewer propagating GMCs. In addition, the characteristics of colonic contractile states after myotomy were normalized.

Myotomy alone increased the frequency of small intestinal GMCs prior to radiation exposure. Interruption of the IC junction may interfere with feedback inhibition of small intestinal GMCs from the colon similar to what occurs within the small intestine (7) as the "ileal brake". Wen et al (20) have suggested that PYY, and possibly GLP-1, participate in the ileal brake. This negative feedback loop affects both the proximal and distal small bowel. The proximal colon also triggers the feedback inhibition of gut motility (colonic brake, 20). In a followup paper (21), Wen et al also suggested that increased volumes and unabsorbed nutrients in the proximal colon alter proximal small bowel motility. Volume-induced effects were mediated via extrinsic nerves, whereas nutrient-induced effects may be mediated by humoral factors, such as plasma PYY. Our previous experiments show that serum PYY is depressed with irradiation and suggests a separate mechanism (22). GMCs produce ascending inhibition within the small intestine that is dependent upon intrinsic innervation (7) while descending inhibition required extrinsic neurons. Ascending inhibition generated by colonic GMCs may influence motility of the distal small intestine via a similar pathway. These experiments cannot define the mechanism of possible colonic inhibition of small intestinal GMCs.

Sarna et al (9) reported that ileal and cecal GMCs were associated with one another but did not investigate these contractions during abnormal motility. Our radiation model exposes both small and large intestine to equal doses of radiation. The majority of small intestinal GMCs that propagate into the colon migrate through the cecum but GMCs may also originate within the cecum. The pathway of propagation for GMCs from the terminal ileum is to the tip of the cecum and then into the proximal colon.

Cecal GMCs, unlike small intestinal and colonic GMCs are not an all or none phenomenon. When multiple GMCs propagate from the small intestine through the cecum, there is a progressive decrease in the amplitude and duration of these contractions suggesting desensitization or fatigue. Since the amplitude and duration of the colonic GMCs did not decrease, there may be different mechanisms for contraction initiation or propagation between the cecum and the colon. Because the canine cecum anatomically resembles the human appendix (9), the propagation of these contractions suggests that the human appendix may possess similar neural circuitry.

Pathways controlling contractile activity in these experiments may apply to other pathologic processes with altered motility including inflammatory bowel disease or infectious diarrhea. Since the coordination of contractile activity occurs through the small intestinal wall to the cecum and colon, resection or interference with that pathway may alter manifestations of disease. Interruption of the ileal-cecal-colonic network may increase ileal contractile activity that is associated with diarrhea and abdominal pain. This may be a potential etiology of abdominal pain in some patients. The logistical difficulties in recording from human distal small intestine contribute to a lack of information regarding this region.

In searching for propagation of GMCs across the IC junction, we arbitrarily set a window of one minute to identify all potential propagating GMCs. The lag time of 36 seconds in intact animals is significantly shorter than that observed in the single myotomy animal that had one GMC migrate across the ileal myotomy. That single GMC may have been a random occurrence. The average velocity of radiation induced GMCs across the IC junction is 0.7 cm/sec, slower than the velocity of small intestinal GMCs.

Radiation induced colonic GMCs were less likely to propagate in animals with ileal myotomy. The frequency of propagation of colonic GMCs as well as the pattern of mid colonic termination was dependant upon an intact IC junction. Our study demonstrated that myotomy disrupted the pattern of colonic GMCs following irradiation. There was no evidence of recovery of intact contractile patterns to suggest that re-growth of myoneural contacts occurred within the 6 week experiment.

Irradiation altered the pattern of colonic contractile states in intact animals but myotomy returned irradiated patterns to those found in normal, nonirradiated controls. The enteric nervous system plays a prominent role in the control of colonic contractions. Interruption of the ileal circular and longitudinal muscle layer, with its intrinsic myenteric network, changed the mean and total duration of colonic contractile states in the distal colon. Normal animals exhibit a progressive increase in the mean duration of colonic contractile states along the length of the colon. Irradiation eliminates this pattern so that all regions of the colon have the same mean contractile duration. Colonic contractile patterns that were altered in intact animals undergoing abdominal irradiation were normalized after an ileal myotomy. The high frequency of propagating GMCs influenced the duration of colonic contractile states. While the contractile patterns of the colon are controlled by intrinsic neurons, they were altered by the excessive frequency of GMCs.

Diarrhea experienced by animals developed earlier following myotomy when compared to intact irradiated animals in spite of nearly normal colonic motor recordings with fewer propagating colonic GMCs. During this time, the frequency of small intestinal GMCs nearly doubled in the myotomy animals compared to those with an intact IC junction. Presuming that the absorptive and secretory changes of radiation would be similar with or without myotomy, increased diarrhea is likely due to the increased frequency of small intestinal GMCs. Our data suggests that processes associated with diarrhea and abdominal cramping, often attributed to the colon, may originate within the small intestine.

Our previous experiments have identified abnormal patterns of intestinal contractile activity after abdominal irradiation. We compared these patterns following disruption of the myenteric plexus by myotomy at the IC junction. Myotomy increased the frequency of small intestinal GMCs prior to irradiation. While small intestinal and colonic GMCs were an all-or-none phenomena, cecal GMCs progressively decreased in duration and amplitude during multiple sequential contractions, suggesting different mechanisms of propagation within the cecum. Radiation induced colonic GMCs were less likely to propagate within the colon following ileal myotomy. This finding suggests that in intact animals, ileal contractile abnormalities may influence colonic motor activity. Colonic contractile states were altered by irradiation in intact animals but were not different from control in myotomy animals following irradiation, suggesting that the large numbers of propagating colonic GMCs interfered with colonic contractile states. Finally, there is a difference in the onset and severity of diarrhea in myotomy animals that correlates with the frequency of small intestinal GMCs. The anatomic relationship between the canine IC junction is similar to the human ileo-appendiceal-colonic region and similar neural connections may affect human contractile activity (23). Our findings suggest the importance of the IC region to contractile disorders of both the small and large intestine. Other disease processes may produce abdominal cramping and diarrhea via similar neural circuitry.

Acknowledgments

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Abbreviations

GMC	Giant Migrating Contraction
MMC	Migrating Motor Complex
CMMC	Colonic Migrating Motor Complex
cGy	Centigray
H&E	hematoxylin and eosin
HVL	half-value layer

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Figure 1.

This H & E section of the ileum shows complete circumferential removal of serosa and muscularis propria including the myenteric and deep muscular plexus. In addition, a portion of the external submucosa was excised. The internal submucosa, muscularis mucosa, lamina propria and epithelium were intact and unremarkable.

Figure 2.

A. This scattergram shows the point of origin and length of propagation of each giant migrating contraction in a composite of all 5 animals that were irradiated but had not undergone an ileal myotomy. Across the x – axis, at the top of the scattergram, the experimental state is identified (control recordings, radiation fraction 1 through 6). The y – axis identifies the distance traveled by location. The distal small intestine, cecum and proximal, mid and distal colon are noted. If a giant migrating contraction occurred at only a single recording site, it is identified as a dot. If a giant migrating contraction propagated from the small intestine directly into the colon and was not recorded in the cecum, it is noted by a dotted line through the cecum. If the animal defecated in association with a giant migrating contraction, the letter **D** and an asterisk was used to identify the event. Note that the majority of GMCs originate in the small intestine and end in the mid colon.

B. The point of origin and length of propagation of each giant migrating contraction in 5 animals that had undergone an ileal myotomy. See A. legend. Note the increased frequency

of small intestinal GMCs in the control state and the disordered propagation of colonic giant migrating

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Figure 3.

This tracing illustrates small intestinal, cecal and colonic GMCs propagating across the IC junction following the second dose of 250 cGy in an intact dog. Along the left margin of the tracing, SI refers to small intestine, CE is cecum and C is colon. The numbers refer to the distance in centimeters from the IC junction. Note the regular propagation of GMCs across the IC junction. Also, each successive giant migrating contraction results in a decreased amplitude and duration of the contraction within the cecum. This is not true within the small intestine or colon.

Figure 4.
In this post myotomy animal following the first dose of 250 cGy, five GMCs reach the terminal ileum and none propagate across the IC junction. See Figure 3 legend.

Figure 5.

This bar graph represents the mean duration of colonic contractile states of intact control, intact irradiated, myotomy control and myotomy irradiated animals. In the mid and distal colon in irradiation intact animals, the increased frequency of propagated colonic GMCs interrupts colonic contractile states. This results in significantly decreased mean duration of colonic contractile activity.

Table 1

Small Intestinal Migrating Motor Complexes

	MMC frequency (#/h)	Phase III duration (min)	MMC cycle length (min)
Intact	0.39 ± 0.05	8.3 ± 3.1	123.5 ± 14.7
Myotomy	0.35 ± 0.04	6.7 ± 0.4	133.1 ± 9.8
<i>Radiation Intact</i>	<i>0.45 ± 0.08</i>	<i>7.6 ± 2.0</i>	<i>132.0 ± 9.7</i>
<i>Radiation Myotomy</i>	<i>0.39 ± 0.04</i>	<i>6.6 ± 0.2</i>	<i>114.5 ± 13.8</i>

Table 2

Small Intestinal Giant Migrating Contractions

	Velocity of Migration (cm/sec)	Amplitude Ratio (GMC/MMC)	Duration (sec)
Intact	1.6 ± 0.00	2.3 ± 0.00	19.2 ± 0.00
Myotomy	1.1 ± 0.20	2.6 ± 0.21	20.1 ± 2.40
<i>Radiation Intact</i>	<i>1.5 ± 0.10</i>	<i>2.6 ± 0.20</i>	<i>16.4 ± 1.00</i>
<i>Radiation Myotomy</i>	<i>1.0 ± 0.10</i>	<i>2.6 ± 0.10</i>	<i>17.4 ± 0.20</i>

Table 3

Cecal Giant Migrating Contractions

	Frequency of GMCs Isolated to cecum/h	Amplitude (grams)	Duration (sec)
Intact	1.3 ± 0.4	192.9 ± 29.0	43.2 ± 2.4
Myotomy	0.8 ± 0.3	258.7 ± 55.3	44.4 ± 2.4
<i>Radiation Intact</i>	1.6 ± 0.4	167.9 ± 24.9*	42.0 ± 1.8
<i>Radiation Myotomy</i>	0.7 ± 0.1*	266.4 ± 59.1	40.8 ± 1.2*