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Analysis of Fasting Antroduodenal Manometry in Children

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Antroduodenal manometry has been used to determine the pathophysiology associated with signs and symptoms of gastrointestinal motility disorders. The diagnostic value of antroduodenal manomentry has been limited by the paucity of data from normal children. In this study, we compared antroduodenal manometry findings from 95 patients with symptoms suggesting a gastrointestinal motility disorder to 20 control children. Phase III of the migrating motor complex (MMC) was less frequent in patients ($P < 0.05$), especially in those who required total parenteral nutrition ($P < 0.001$), than in controls. Abnormal migration of phase III and short intervals between phase IIIs were more frequent in patients than in controls ($P < 0.01$ and $P < 0.05$, respectively). During phase II, persistent low-amplitude contractions and sustained tonic-phasic contraction were found only in parenteral-nutrition-dependent children. Short or prolonged duration of phase III, absence of phase I following phase III, tonic contractions during phase III, low amplitude of phase III contractions in a single recording site and clusters of contractions or prolonged propagating contractions during phase II were not more frequent in patients than in controls. We conclude that there are five manometric features having a clear association with pediatric gastrointestinal motility disorders: (1) absence of phase III of the MMC, (2) abnormal migration of phase III, (3) short intervals between phase III episodes, (4) persistent low-amplitude contractions, and (5) sustained tonic-phasic contractions.

KEY WORDS: manometry; gastrointestinal motility; migrating motor complex.

Antroduodenal manometry measures the intraluminal pressure in the distal stomach and the duodenum. There are many reports describing abnormal contraction patterns in symptomatic patients studied with antroduodenal manometry (1–7). In several studies, attempts have been made to differentiate various patients groups from each other and from normal subjects by defining "abnormal manometric findings" (1–3, 8–11). In one study, abnormal manometric findings occurred with similar frequencies in healthy and symptomatic adults (11).

In children, antroduodenal manometry has been used in the evaluation of chronic intestinal pseudoobstruction, nonulcer dyspepsia, gastroesophageal reflux disease, recurrent abdominal pain, toddler's diarrhea, and diabetic gastroparesis (5, 8, 12–19). Intestinal manometric findings have been reported from healthy newborns and premature infants (20–22). Except for the absence of the MMC predicting a requirement for special nutritional support (19), the clinical implications of discrete manometric patterns remained largely speculative. The total number of children studied is small, and there is only a rudimen-

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tary understanding of the sensitivity and the specificity of manometric findings.

In this study, we attempted to define normal and abnormal antroduodenal manometric findings by comparing the data from children with upper gastrointestinal motility disturbances to the data from children with no upper gastrointestinal disease.

MATERIALS AND METHODS

Subjects. We studied 95 patients, 52 males and 43 females, 0–20 years of age (mean ± SD = 4.6 ± 4.7). All patients had been referred to our institutes (Harbor UCLA Medical Center, Torrance, California, and Gunma University School of Medicine, Gunma, Japan) from other hospitals for antroduodenal motility studies to clarify the physiology responsible for their signs and symptoms. Mechanical obstruction was ruled out in all cases by either x-ray or laparotomy. Patients with known secondary causes of functional obstruction (3), such as diabetes mellitus, systemic sclerosis, or amyloidosis, were excluded from the present study. In order to evaluate the manometric findings in relation to the severity of the symptoms, we divided these patients into three groups based on the symptom severity assessed by means of nutritional support.

Group 1 consisted of 29 patients (17 boys and 12 girls, mean age 7.6 ± 6.9 years old) with abdominal distension, nausea and vomiting, loss of appetite, and abdominal pain, but no nutritional support besides their oral food intake. Group 2 consisted of 27 patients (16 boys and 11 girls, 3.5 ± 2.7 years old) who required occasional intravenous infusion and/or gastrostomy or jejunostomy feeding to relieve their gastrointestinal symptoms but never needed central parenteral nutrition (CPN) to maintain their nutrition. Group 3 consisted 39 patients (19 boys and 20 girls, 3.1 ± 2.4 years old) who required CPN occasionally or continuously to fulfill their daily nutritional requirements.

The control group consisted of 20 subjects (10 boys and 10 girls, mean age 7.5 ± 6.9 years old) without upper gastrointestinal disease. Although it is ideal to have age-matched healthy controls, we believed that intestinal manometric study, because of its inherent discomforts, should not be done in children who might not benefit from testing. As a consequence there are no data from asymptomatic healthy children. We included as controls nine children with severe constipation who underwent both colonic and antroduodenal manometry to assess the possibility of a generalized motility disorder, involving colon and small intestine. Each was diagnosed conclusively as having functional fecal retention based on normal colonic manometry and resolution of all symptoms after effective medical management (23). Seven other children with histories of chronic illness requiring special nutritional support were finally diagnosed as having Munchausen syndrome-by-proxy proven by the resolution of all symptoms with separation from the primary caretaker. In these seven cases, manometric studies were carried out when the symptoms were absent. Two adolescents, 18 and 19 years old, were student volunteers. In two term infants with cholestasis, the parents allowed us to record antroduodenal motility prior to infusing magnesium sulfate into the duodenum to collect bile. (In Japan, intraduodenal infusion of magnesium sulfate is a common diagnostic technique to diagnose congenital biliary atresia, which was subsequently excluded in these two cases.)

TABLE 1. PRESENCE OF PHASE III

	Group 1 (N = 29)	*Group 2 (N = 27)*	*Group 3 (N = 39)*	*Control (N = 20)*
Antral phase III	11 (37.9%)	14 (51.9%)	6a† (15.4%)	10 (50.0%)
Duodenal phase III*	28 (96.6%)	23 (85.2%)	15b† (38.5%)	19 (95.0%)

* Including both phase III that started from the stomach and phase III that started from duodenum.

† a and b indicate significant difference from the control ($P < 0.01$ and $P < 0.001$, respectively).

Drugs affecting gastrointestinal motility were stopped three days prior to manometry.

Methods. We used a six-lumen polyvinyl catheter to measure intraluminal pressures in the gastric antrum and duodenum. In infants, a three-lumen catheter was used, instead. The details of the technique has been reported elsewhere (15, 20). After sedation of the patients, the catheter was positioned with either fluoroscopic or endoscopic guidance. One or two proximal side holes were positioned in the gastric antrum and the other four or five in the duodenum. The next day, recording was carried out. The catheter was perfused with distilled water by a low-compliance, pneumohydraulic, capillary-infusion system (Arndorfer Medical Specialties, Inc., Greendale, Wisconsin) at a rate of 0.4 ml/min. In young subjects, including infants, the catheter was perfused by a piston pump (Harvard Apparatus, South Natrick, Massachusetts) connected to a stainless steel capillary at a rate of 0.1 ml/min. Amplitudes recorded by these two methods were previously shown to be equal (20). Pressures were recorded on a polygraph equipped with pressure couplers. We studied fasting motility in each patient for at least 4 hr after at least 12 hr of fasting, only the first 4 hr of which we evaluated in each subject for the fair comparison. We recorded postprandial motility for an hour after a meal in most of the patients. In the present study, however, postprandial motility was not analyzed, because the volume of the meals varied depending on the condition of the patient, and some patients had symptoms so severe that they could not ingest any food. This study was approved annually by the Harbor-UCLA Human Subjects Committee beginning in May 1985 and by Gunma University (Department of Pediatrics) Human Investigation Committee on May 1984. Informed written consent was obtained from the parents of every patient.

Data Analysis. All records were inspected visually. We analyzed data according to the items appearing in Tables 1–3. We chose these items based on the data published on normal and abnormal antroduodenal motility in children and adults (4, 16, 25–38).

Precise definition of phase III was particularly important when analyzing the data, because in clinical practice it was often difficult to decide whether the contraction band was a normal phase III episode, an abnormal phase III, or non-

TABLE 2. MANOMETRIC FINDINGS OF PHASE III

	Group 1 (N = 28)	*Group 2 (N = 23)*	*Group 3 (N = 15)*	*Control (N = 19)*
Short duration (<3 min)	18* (64.3%)	13 (56.5%)	6 (40.0%)	7 (36.8%)
No aboral migration	9b† (32.1%)	8b (34.8%)	3 (13.0%)	0 (0.0%)
Not followed by phase 1	8 (28.6%)	11a (47.8%)	7 (30.4%)	3 (15.8%)
Short interval (<30 min)	9 (32.1%)	9a (39.1%)	5 (21.7%)	2 (10.5%)
With tonic component	3 (10.7%)	6 (26.1%)	5 (21.7%)	3 (15.8%)
With prolonged contraction	2 (7.1%)	3 (13.0%)	0 (0.0%)	0 (0.0%)
Low amplitude (<20 mm Hg)	7 (25.0%)	6 (26.1%)	5 (21.7%)	3 (15.8%)

* Figures are the number of patients in whom each finding was recorded in the phase III.

† a and b indicate significant difference from the control ($P < 0.05$ and $P < 0.01$, respectively).

propagating contraction clusters. In the present study, phase III was defined as bands of regular repetitive contractions occurring at a maximal rate for the part of the alimentary tract (3–4/min in the antrum and 10–12/min in the small intestine) lasting >30 sec, propagating faster than 3 cm/min more than 6 cm along the duodenum or small intestine (Figure 1). Only phasic pressure rises >10 mm Hg were regarded as contractions, except in patients with a myopathic pattern. In children with intestinal myopathy, it was possible to recognize phase III-like patterns consisting of pressure changes less than 10 mm Hg when there was a stable baseline. Propagation velocity in the small intestine was calculated by dividing the distance (centimeters) between the most proximal and distal side holes by the time interval (minutes) between the time when phase III started in the most proximal site and when in the most distal site. The phase IIIs in the most proximal duodenum were often short in duration and started after the phase IIIs in the second proximal site in normal individuals. In these cases, the second proximal site, instead of the most proximal site, was used to calculate migration velocity. The bands of repetitive contractions which did not satisfy the above definition were regarded as clusters.

Each phase III was evaluated according to the following criteria.

1. Duration. When phase III in the duodenum or jejunum (except the most proximal site) lasted less than 3 min, phase III was regarded as short (2, 24).
2. Aboral Migration. When the calculated propagation velocity was greater than 25 cm/min, the phase III was regarded as nonmigrating or stationary (3, 10, 25–27) (Figure 2). When the velocity was less than 0, the migration was regarded as retrograde.
3. Not followed by Phase I. When contractions occurred within 5 min after the phase III, the following phase I was regarded as absent (2, 7, 28, 29).
4. Short Interval. When the interval between the phase III and the preceding phase III was less than 30 min, the interval was regarded as short (6, 30–32) (Figure 3).
5. With Tonic Component. Tonic component referred to an elevation of the baseline during phase III by more than 10 mm Hg for longer than 1 min (1, 3, 5) (Figure 4).
6. With Prolonged Phasic Contraction (PPC). PPC was defined as a contraction that occurred in the duode-

TABLE 3. MANOMETRIC FINDINGS OF PHASE II

	Group 1 (N = 29)	*Group 2 (N = 27)*	*Group 3 (N = 39)*	*Control (N = 20)*
Clustered contractions	13* (44.8%)	14 (51.9%)	21 (53.8%)	8 (40.0%)
Prolonged propagating contraction	11† (37.9%)	4 (14.8%)	1 (2.6%)	2 (10.0%)
Discrete clusters of contractions	0 (0.0%)	0 (0.0%)	1 (2.6%)	0 (0.0%)
Low peak amplitude	0 (0.0%)	2 (7.4%)	8† (20.5%)	0 (0.0%)
Sustained phasic-tonic contraction	0 (10.7%)	1 (3.7%)	3 (7.7%)	0 (0.0%)

* Figures are the number of patients in whom each finding was recorded in the phase II.

† Significant difference from the control ($P < 0.05$).

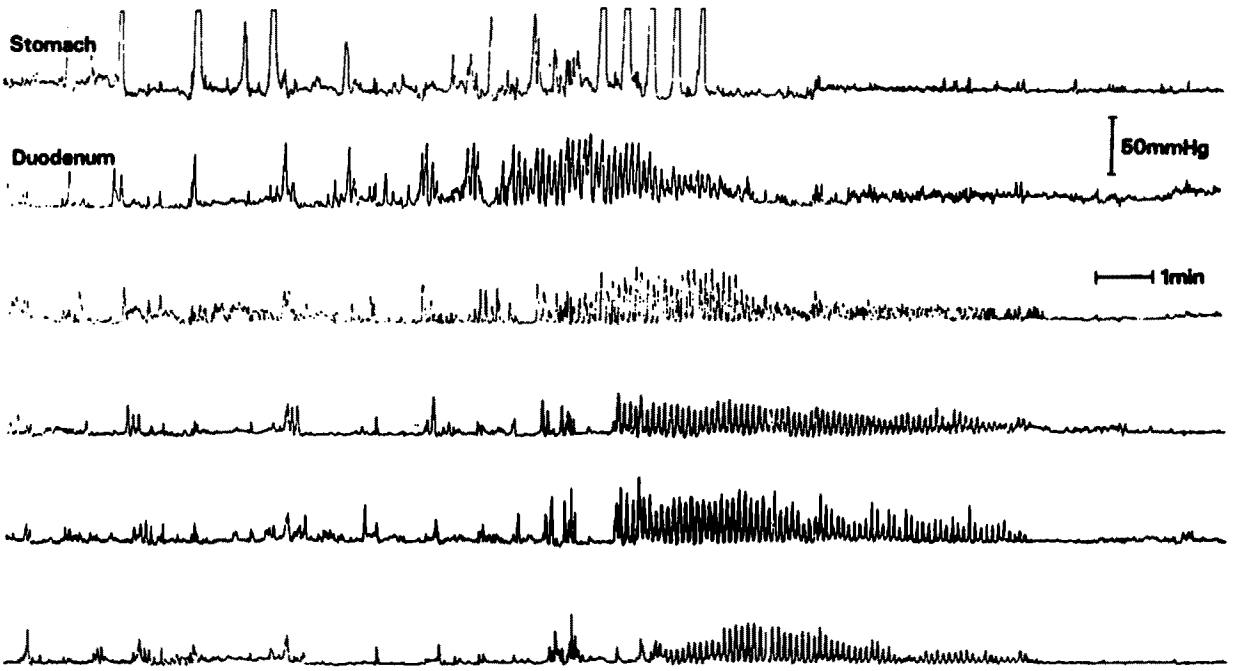

Fig 1. Normal phase III. Pressure changes in the gastric antrum (top tracing) and the duodenum were recorded by means of an infused catheter. Note the band of repetitive contractions starting in the antrum and migrating aborally along the duodenum.

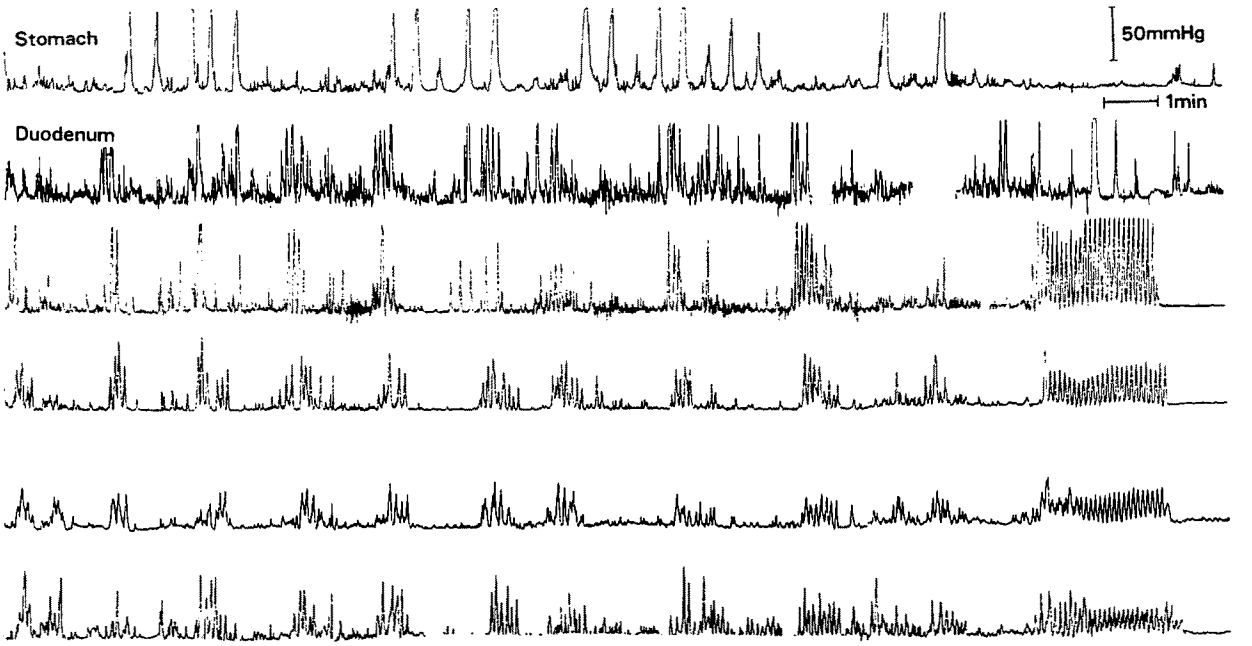

Fig 2. Abnormal migration of phase III. A phase III occurred in duodenal recording sites and jejunum simultaneously (underline) [From Tomomasa (40) with permission].

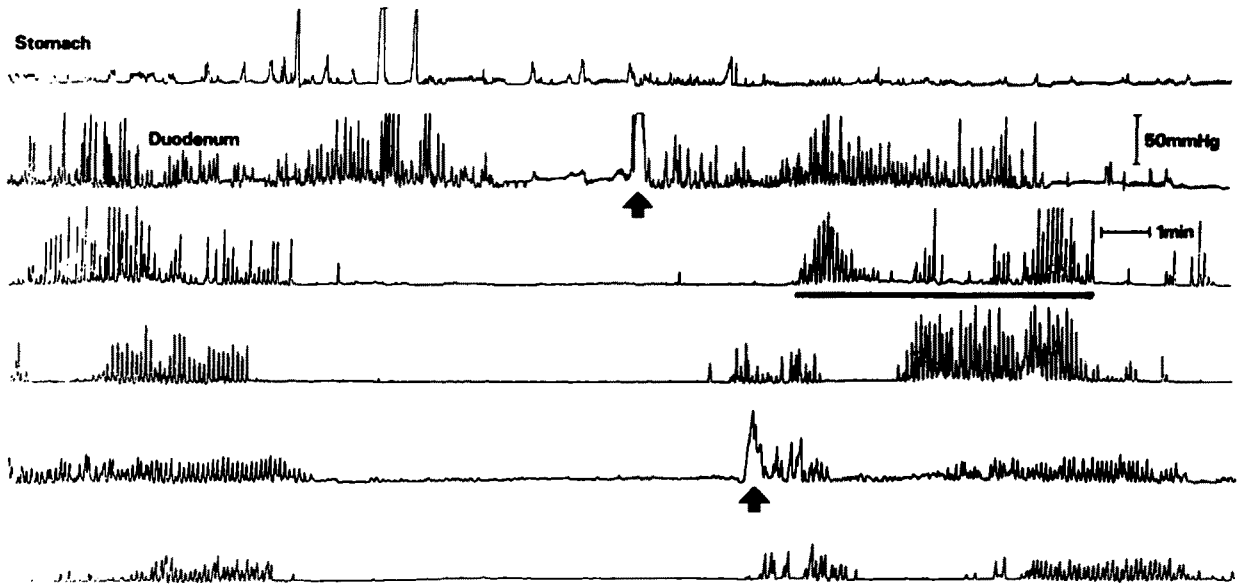

Fig 3. Short interval of phase IIIs and prolonged phasic contractions (arrows) during phase III. Two phase IIIs occurred separated by an interval of 10 min.

num or in the small intestine, had a peak amplitude of more than 30 mm Hg, and a duration longer than 6 sec (4, 25, 33).

7. Low Peak Amplitude. A mean amplitude of less than 20 mm Hg in one or more recording sites was regarded as low (3, 7).

Phase 2 was evaluated according to the following criteria:

1. Tonic Component. Elevation of the baseline more than 10 mm Hg lasting more than 1 min with or without phasic contractions (3, 34–36).
2. Prolonged Propagating Phasic Contraction (PPPC). PPPC was defined as a contraction that propagated along the duodenum or the small intestine more than 6 cm within 6 sec, had a mean peak amplitude of more than 30 mm Hg, and had a duration longer than 6 sec at more than one site (6, 37, 38).
3. Clusters of Contractions. A band of repetitive contractions that occurred at the maximal frequency for the site (same as phase III), lasted more than 30 sec, and did not satisfy the above criteria for phase III (1–5, 27, 35, 37, 38).
4. Discrete Clustered Contractions (DCC). A series of clusters each of which lasts longer than 30 sec during the total period of longer than 5 min.
5. Sustained Phasic and Tonic Contractions (SPTC): A cluster of contractions which had tonic components and lasted more than 10 min in only one recording site, with normal motility patterns at the other sites (4, 5, 35) (Figure 5).
6. Low Amplitude. When the peak amplitude of more than 90% of contractions was less than 20 mm Hg at all sites (7) (Figure 6).

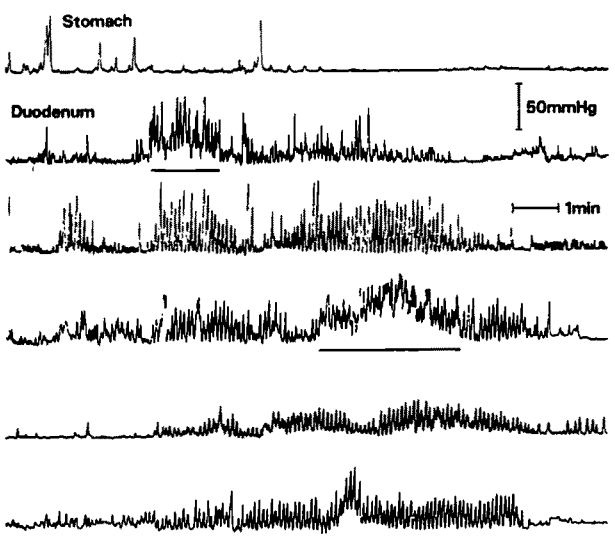

Fig 4. Tonic component within the phase III. Tonic component was defined as an elevation of the base line of phase III by more than 10 mm Hg for longer than 1 min.

RESULTS

During a 4-hr test session with the children fasting, phase III of the MMC appeared in 95% of control children, 69.5% of all patients ($P < 0.05$), and 38.5% of the subset of CPN dependent patients ($P < 0.001$) (Table 1).

The characteristics of phase III are summarized in Table 2. Phase III was free from limitations in 8 of 19 control children with phase III, but in only 8 of 66 symptomatic children with phase III ($P < 0.05$). Abnormal migration of phase III was seen in 20 of 66 patients and in none of the control cases ($P < 0.01$).

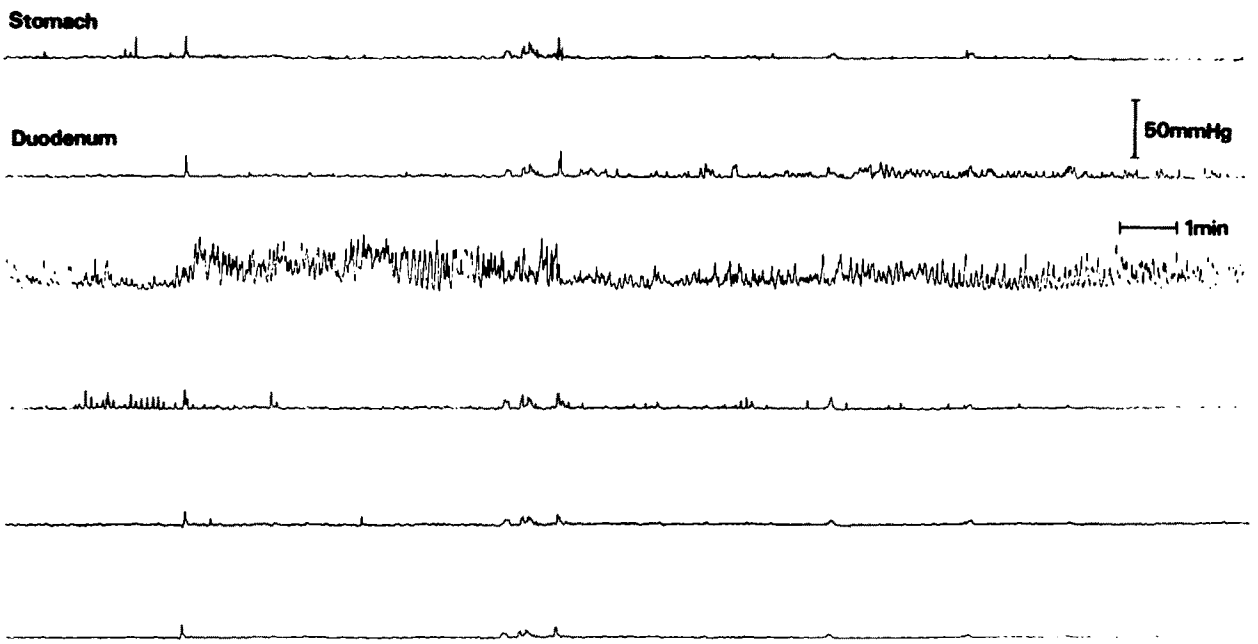

Fig 5. Sustained phasic and tonic contractions (SPTC). SPTC was defined as a cluster of contractions that had tonic components and lasted for longer than 10 min in only one recording site with a normal motility pattern in the other sites [From Tomomasa (40) with permission].

Lack of phase I and a short interval between phase IIIs were more common in group 2 patients when compared with control ($P < 0.05$). They were also more frequent in whole patients than in controls ($P < 0.05$). Phase IIIs with short-duration, and low-amplitude, tonic, and prolonged phasic contractions were not significantly more frequent in patients than in controls ($P > 0.05$).

Characteristics of phase II are summarized in Table 3. Prolonged propagating contractions were more

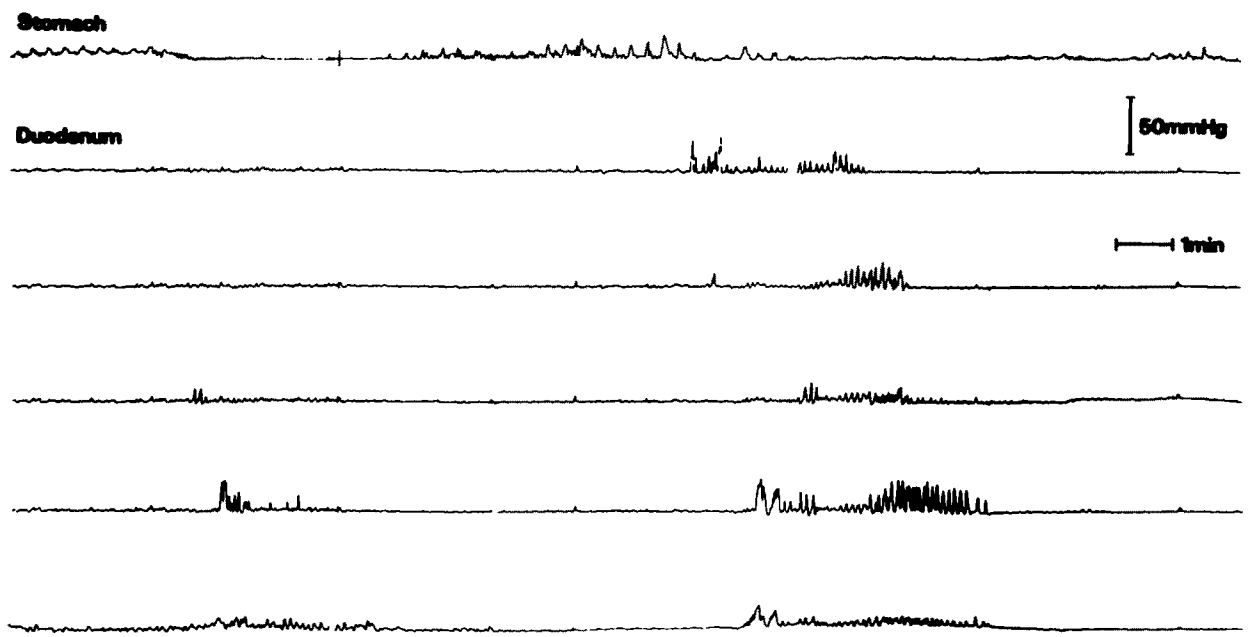

Fig 6. When the peak amplitude of more than 90% of contractions throughout the record was less than 20 mm Hg, it was regarded as persistent low amplitude [From Tomomasa (40) with permission].

common in group 1 patients than in controls ($P < 0.05$). Persistently low-amplitude contractions (myopathic pattern) were observed only in group 2 ($P > 0.05$) and group 3 patients ($P < 0.05$). SPTC were noted only in severe cases. Clusters of contractions, DCC, and SPTC were not more frequent in patients than in controls.

Three children in the control group (15%), 16 in group 1 (55%), 20 in group 2 (74%), and 32 in group 3 (82%) had one or more of the five following abnormalities: the absence of phase III during 4 hr of fasting, abnormal migration of phase III, short intervals between phase IIIs, persistently low amplitude contractions at all recording sites, and SPTCs [$P < 0.001$ (ANOVA)].

DISCUSSION

Within the last decade, antroduodenal manometry has moved from research laboratories to referral centers, where it can be applied to investigate patients with unexplained upper gastrointestinal symptoms. Fasting intestinal motor activity consists of cycles of phase III patterns of contractions from the stomach to the ileum (10), known as the migrating motor complex (MMC). Most easily identified is phase III, which consists of 3–5/min bursts of rhythmic, repetitive, high-amplitude contractions that are propagated slowly from stomach to ileum, sweeping undigestible luminal contents and bacteria out of the small bowel. This is followed by phase I of quiescent activity and is preceded by phase II or irregular activity, consisting of random, intermittent contractions of variable amplitude. After meals the MMC is replaced by intermittent continuous contractions of variable amplitude, some of which are propagated.

In this study, we attempted to discriminate normal from abnormal fasting antroduodenal contraction patterns by comparing data from 95 children with symptoms of gastrointestinal motility disorder to 20 children with no upper gastrointestinal symptoms. Phase III of the MMC occurred less frequently in patients than in controls during a 4-hr recording period. The presence of the phase III appears to be a marker for neuromuscular integrity, at least in childhood (1, 5, 10, 11). Phase III is absent in preterm infants <34 weeks postconception (20). The appearance of phase III may signal a developmental milestone. Thus, the absence of phase III in the presence of normal amplitude contractions may indicate a persistent neurodevelopmental delay or an acquired neuropathy. In adults, there is a wide variability in MMC cycling time between individuals and within the same individual on separate days (9), and the absence of phase III during a 3- or 4-hr study is not a diagnostic abnormality (1).

In our study, patients without a phase III during a 4-hr test session were more likely to require parenteral nutrition. We previously showed that the absence of phase III during a 4-hr recording period was correlated with a requirement for special nutritional support and a poor response to cisapride (19). This larger retrospective analysis confirmed the smaller prospective study demonstrating that phase III was an important predictor of the clinical course in children.

One possible way to increase the discriminative power of the absence of phase III to diagnose functional motility disturbance may be to make a manometric recording for a prolonged period of time utilizing nonperfused catheters and an ambulatory recording. Short studies in the motility laboratory have the advantage of facilitating direct observation by the clinician, so that symptoms correlated with manometric events can be documented precision. Instead of the 24-hr ambulatory manometric recording, we currently use erythromycin in an attempt to induce phase III in children if they have no spontaneous phase III MMCs during the 4-hr fasting period (13).

We observed an abnormal migration of phase III only in patients, but not in controls. Nonpropagating and retrograde phase IIIs were reported in patients with functional upper gastrointestinal symptoms (3, 39), myotonic dystrophy (7), and chronic intestinal pseudoobstruction (CIP) (1, 5, 15). Abnormal migration of the phase III is a discrete abnormality and suggests a gastrointestinal motility disorder.

Absence of phase I and short intervals between phase IIIs were more common in our patient group than in controls. Phase I is absent in some patients with CIP (40) and short in patients with myotonic dystrophy (7). Short intervals between phase IIIs have been described in patients with CIP (40), functional gastrointestinal disorders (6, 8, 39), diabetic motility disorders, and postvagotomy diarrhea (32). In our studies, the differences between the frequency of these findings in patients and controls were not very impressive. Therefore, the significance of these findings are not very clear.

Prolonged propagating phasic contractions were more frequent in group 1 patients than in controls. These contractions are a normal, although infrequent feature of ileal motility in humans (41). PPPCs have been recorded from the small intestine of patients with irritable bowel syndrome (6) and a novel type of

chronic diarrhea (38). At present, the occasional occurrence of PPPC's should not be regarded as an indicator of disturbed intestinal motility.

In our study, clustered contractions were seen in patients and in controls with similar frequencies. Nonmigrating clusters of contractions have been described in the upper small intestine of the normal newborns (20) and healthy adults during fasting (2). Frequent, nonmigrating clusters of contractions in the small bowel have long been interpreted as an abnormal finding indicating CIP (5), mechanical bowel obstruction (2), diabetic gastroparesis (42), functional bowel disorder (3), or systemic sclerosis involving the gastrointestinal tract (34). DCCs have been observed in the small bowel of patients with irritable bowel syndrome and some healthy controls (6). In our study DCC was seen in only one patient. Based on our findings, we suggest that the presence of clustered contractions or DCC in the small bowel should not be interpreted as an abnormal manometric finding. SPTCs were noted only in patients with severe symptoms. It has been described in functional upper-gut symptoms (3), and CIP (1).

The most important diagnostic role for antroduodenal manometry in clinical practice may well be the discovery of normal physiology in children and adolescents with apparent intestinal failure. Illness dyssynchronous with the physiology should prompt the clinician to consider alternative explanations such as Munchausen's syndrome-by-proxy (43) and visceral pain disorder (44). Indeed, overinterpretation of manometric findings may reinforce illness-related behaviors or provide inadvertent collusion with perpetrators of child abuse (45).

In conclusion, we describe five manometric features having a clear association with intestinal motility disorders: (1) absence of phase III of the MMC, (2) abnormal migration of phase III, (3) short intervals between phase III episodes, (4) persistent low-amplitude contractions, and (5) sustained tonic-phasic contractions. This analysis was extensive, yet incomplete. We hope that this study will be a step forward towards better understanding normal gastrointestinal physiology and will be useful for the clinical applicability of the manometric techniques.

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