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TECHNICAL NOTE

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The antroduodenal transition time is prolonged in adults with type 1 diabetes

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ABSTRACT

Background: The gastroparetic syndrome encompasses antral hypomotility, gastric dysrhythmia, impaired antroduodenal coordination, pyloric dysfunction, and abnormal duodenal motility; the last three collectively referred to as pylorospasms. We hypothesized that antroduodenal motility is diminished and transition time is prolonged in adults with type 1 diabetes (T1D) and polyneuropathy.

Methods: This cross-sectional study included 124 participants, of which 21 were healthy, 53 had T1D and 50 had T1D with distal symmetrical polyneuropathy (T1D + DSPN). We used the wireless motility capsule to assess antroduodenal transition time, gastric emptying time, gastric and small bowel motility indices (MI), and numbers of alkalic/acidic exposures.

Results: In comparison with controls, patients with T1D had prolonged antroduodenal transition time (1.85±1.5 vs. 6.6±4.8 minutes; p=0.02), which was even more pronounced in patients with T1D+DSPN (1.85±1.5 vs. 17.8±28.5 minutes; p<0.008. T1D+DSPN tended to have diminished gastric MI (11.9±2.4 vs. 12.7±1.0, p=0.07) and small bowel MI (13.1±1.4 vs. 13.6±0.6, p=0.05) and experienced more antral/pyloric alkalic episodes (1.2±1.3 vs. 2.0±2.1, p=0.02) compared with controls.

Conclusion: The current method may assess a proxy for severity of pylorospasms in patients with diabetes and other diseases associated with upper gastrointestinal motility disorders, which ultimately may optimize future management.

KEYWORDS
antroduodenal passage, antroduodenal transisition, diabetic enteropathy, diabetic gastroparesis, pylorospasm, pyloric antrum, pyloric passage

1 | INTRODUCTION

Delayed gastric emptying in the absence of mechanical obstruction is referred to as gastroparesis, which is a common complication to long-term diabetes.[1] Gastroparesis is accompanied by symptoms including nausea, vomiting, upper abdominal pain, postprandial fullness, and bloating.[2,3] It has been suggested that poor glycemic control and associated autonomic neuropathy may lead to 1) antral dilation and hypomotility, 2) gastric dysrhythmia, 3) lack of antroduodenal coordination, 4) pyloric dysfunction, and 5) abnormal duodenal motility, which in concert leads to dysmotility and increased tonic pyloric activity, referred to as pylorospasm.[4-6]

Hitherto, invasive, and resource-demanding diagnostic methods have been used to evaluate the antroduodenal and pyloric sphincter

function including scintigraphy,[7] antroduodenal manometry,[8] and the endoFLIP,[9] providing detailed categorization of abnormal segmental motility patterns.[10]

In addition to established measures such as gastric transit times, the wireless motility capsule (WMC) may, however, provide an attractive and less invasive alternative, allowing assessment of antroduodenal transition time, antral and duodenal motility indices, (MI) and numbers of alkalic/acidic exposures as it traverses the pyloric region.[11,12] We hypothesized that the adults with type 1 diabetes (T1D) and T1D with polyneuropathy (T1D+DSPN) experienced dysmotility and pylorospasms. Consequently, the aims were to use the WMC in healthy and in T1D with/without polyneuropathy and compare 1) antroduodenal transition time, 2) gastric and small bowel MI, and 3) numbers of alkalic/acidic exposures across the pyloric region.

2 | METHODS

2.1 | Study population

This cross-sectional study included 124 participants, of which 21 were healthy (median 52 (IQR 45–55)), 53 had T1D without DSPN(42 (IQR 28–57)) and 50 had T1D+ DSPN (50 (IQR 45–56)) according to the Toronto criteria.[13] Patients were recruited from the Department of Endocrinology at Aalborg University Hospital, Denmark and received stable antidiabetic medication minimum 1 month prior to study entry. The cross-sectional data presented in this technical note are based on secondary analysis of existing data.[14] All participants completed the self-reported Gastroparesis Cardinal Symptom Index (GCSI), wherefrom three subscales can be derived nausea/vomiting, bloating and postprandial fullness. The study was approved by The North Denmark Region Committee on Health Research Ethics, Denmark: N-20170045 and N-20130077, the latter is a centrally registered clinical trial EUDRA-CT:2013–004375–12 and NCT02138045.

2.2 | The wireless motility capsule

The WMC (SmartPill Monitoring System, Medtronic, Minneapolis, USA) records pH, pressure, and temperature as it transverses the GI tract and measures of gastric emptying time, gastric, and small bowel motility indices (MI) are described in details elsewhere.[15] The gastric MI was calculated during 1 hour before T1, and the small bowel MI was calculated during1 hour after T2 suggested by Camilleri et al.[16] We defined antroduodenal transition time as the duration from T1 (last point with stable acidic pH measure) to T2 as (first point with stable alkalic pH measure) and numbers of alkalic/acidic episodes. Furthermore, T1 is characterized by a sudden increase from pH<1 until T2, where the pH has increased minimum 5-pH units from pH at T1. The number of alkalic exposures is acquired by counting sudden increase in antral pH, followed by drops in pH more than 0.5-pH-units. The number of acidic exposures occurring after T2 was counted when a sudden decrease in duodenal pH was followed by a minimum 0.5-pH units increase. Data were analyzed semi-automatically by customized MATLAB subroutines (MATLAB R2017b, MathWorks), but all traces were independently confirmed by 2 observers (LD and CB) and if any disagreement, consensus was reached, before further analyses.

2.3 | Statistics

Descriptive data are reported as mean ±standard deviation if not otherwise stated. The antroduodenal transition time in the three cohorts was compared using one-way ANOVA with Tukey´s test for post hoc comparisons. The gastric and duodenal MI between healthy and T1D was compared with Student t test. A number of participants who experienced >1 episodes with alkalic/acidic exposures were compared with those who experiencing <=1 episodes by use of a chi-square test. Associations between antroduodenal transition time, gastric, and duodenal MI´s and nausea, HbA1c, and gastric emptying time were analyzed using Pearson's correlations. IBM SPSS Statistics (Version 26, IBM) was used for all. A p-value <0.05 was considered statistically significant.

3 | RESULTS

We analyzed the following numbers of traces: Healthy: 19/21; DM1: 43/53 and DM1+DSPN: 44/50. Recordings were missing either because they were not undertaken, or extensive signal loss existed in the pyloric region. Clinical information of the T1D patients are presented in Table 1

TABLE 1 Clinical data from the two diabetes cohorts.

	Patients with Type 1 diabetes	Type 1 diabetes with polyneuropathy
Disease duration (year)	20 (13–31)	31 (25–40)
HbA1C*	61.1±11.2	66.5±14.0
Gastric emptying time (minute)*	206±136	356±461
Gastroparesis Cardinal Symptom Index	0.4±0.5	0.5±0.6
Nausea	0.1 ± 0.2	0.2 ± 0.6
Bloating	0.5±1.0	0.8±1.0
Postprandial fullness	0.5±0.6	0.6±0.7

Note: Data are presented as median (IQR) or mean±SD, depending on data distribution.
*p = 0.04

3.1 | Antroduodenal transition time

Patients with type 1 diabetes have a significantly prolonged antroduodenal transition time (F = 6.13, p = 0.003). Post hoc testing revealed difference between HP and T1D: (1.85 ± 1.5 minutes vs. 6.6 ± 4.8 minutes; p = 0.02) and even more pronounced between HP and T1D+DSPN (1.85 ± 1.5 minutes vs. 17.8 ± 28.5 minutes; p = 0.008). A typical example of the antroduodenal transition time is shown in Figure 1. There was no association between transition time and symptoms, HbA1C nor with gastric emptying time.

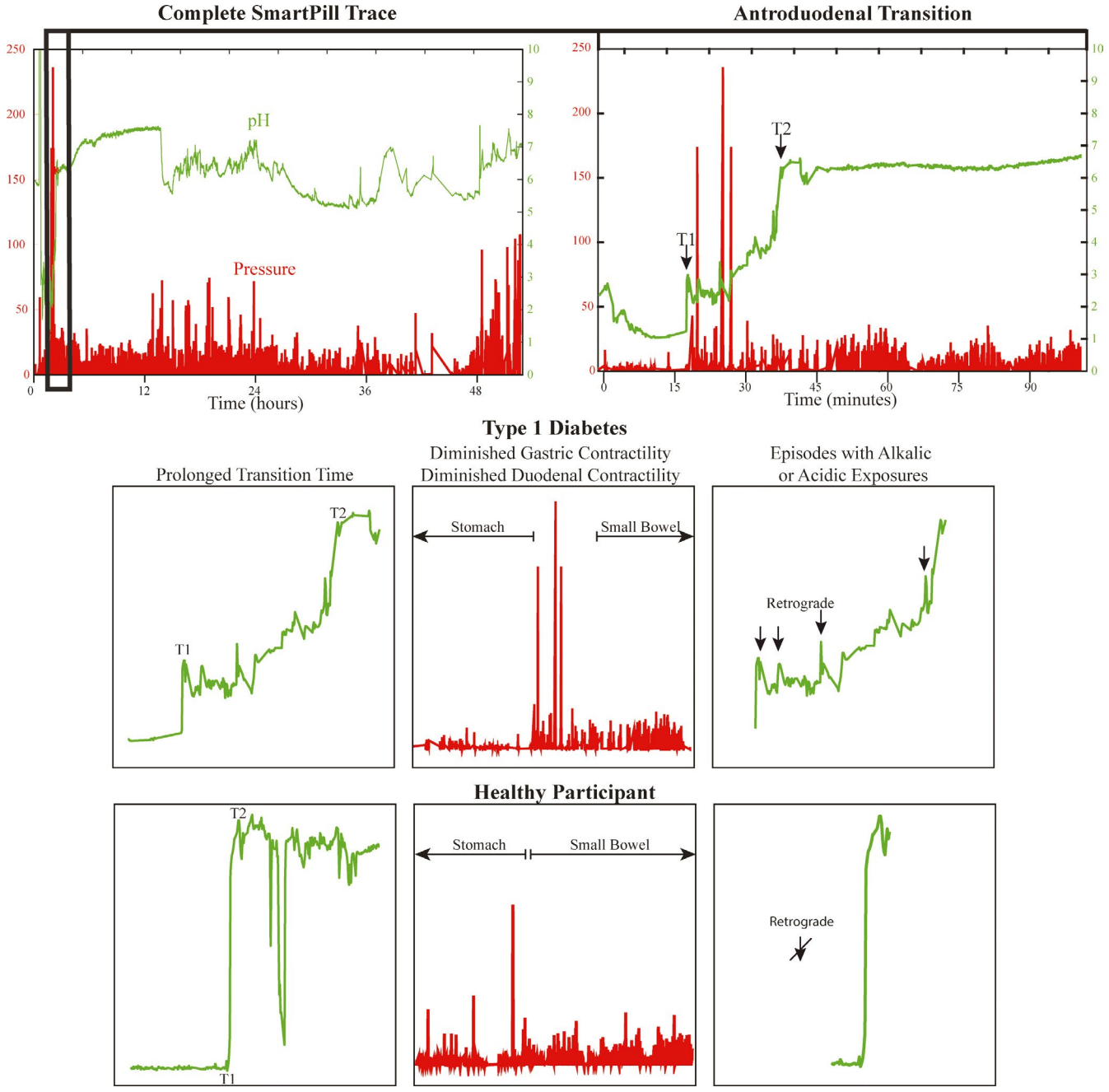

FIGURE 1 A representative wireless motility capsule trace of an adult with type 1 diabetes mellitus and polyneuropathy revealing the presence of pylorospasms. The upper row shows the full trace on the left with the time (hours) on the x-axis, while a zoomed-in view of the antroduodenal transition is shown on the right with the time (minutes) on the x-axis. The y-axis on both represents the pressure in mmHg (red, left y-axis) and pH units (green, right y-axis). The lower row illustrates the transition time between T1 and T2, where T1 is characterized by a sudden increase from pH<1 until T2, where the pH increases minimum 5-pH unit from pH at T1. The antroduodenal transition time was calculated as the time duration between T1 and T2. The downward pointing arrows illustrate four episodes of antral alkalic exposures, plausibly representing duodenogastric reflux. Furthermore, one episode of acidic exposure to the WMC after entering the duodenum is seen representing either gastric content spill-over or a true episode of retro peristaltic movement of the capsule. The motility indices were diminished in the antrum and duodenum, however, the prominent increased pressure spikes likely reveal enhanced pyloric pressure/spasm.

3.2 | Motility index before and after the antroduodenal passage

In comparison with the healthy, patients with T1D+DSPN tended to have diminished gastric motility index (11.9 ± 2.4 vs. 12.7± 1.0; p = 0.07) and duodenal motility index (13.1± 1.4 vs. 13.6 ± 0.5; p = 0.05). Furthermore, in T1D+DSPN, nausea and gastric emptying time were highly significantly associated with the motility index of the stomach (r = −0.835, p = 0.001for nausea; r = −0.585, p<0.001 for gastric emptying time) and duodenum (r = −0.914, p < 0.001 for nausea, r = −0.47, p = 0.001 for gastric emptying time).

3.3 | Episodes with alkalic or acidic exposures

A typical example of four antral alkalic and one duodenal acidic episode is shown in Figure 1. The number of alkalic exposures for HP was 1.2±1.3; for T1D: 1.7±1.7 and T1D+DSPN: 2.0±2.1. In comparison with the HP, a number of alkalic exposures >1 were more frequently observed in T1D+DSPN: (χ^2 = 5.38, p = 0.02). There was no association between these and symptoms or gastric emptying time. The number of acidic exposures for healthy was 3.0±2.1; for T1D: 2.9±2.3 and T1D+DSPN: 2.7±2.7, (χ^2 =2.86, p =0.24).

4 | DISCUSSION

We showed that in comparison with healthy the antroduodenal transition time was prolonged in patients with T1D with and without concomitant DSPN. The latter group also had diminished MI and increased numbers of antral or pyloric alkalic episodes, representing duodenogastric reflux of bile and bicarbonate. Finally, we showed that diminished MI´s in the stomach and duodenum was associated with nausea and gastric emptying time in those with DSPN.

During normal physiological conditions, intestinal neurohormonal control of gastric emptying is slowed in response to the duodenum being exposed to nutrients, causing inhibition of the gastric MMC-phase 3 driving force.[14] The underlying dissociative mechanism of the proposed physiologic reflux of duodenal contents and the reflux of potentially harmful bile acids is, however, incompletely understood.[17,18-21] Nevertheless, antroduodenal transition in healthy is well-coordinated and fast, which is also confirmed in our data. In contrast, electro-gastrography has revealed disturbances of the gastric electrical rhythm in patients with diabetes, leading to impairment of the ileal break mechanism.[22] Different sophisticated research methods including high-resolution manometry, scintigraphy, and entrocapsules such as PillCam or endoFLIP have been used/proposed to unravel the underlying mechanisms. The clinical availability of these methods is unfortunately limited.[5,8,9,11] Thus, the minimally invasive WMC may offer an intuitively easy interpretable and warranted clinical method. Antroduodenal transition is characterized by pH alterations from stable gastric acidic environment to stable duodenal alkalic environment, and the duration of this, including, for example, unstable pH measures, represents to the best of our knowledge a proxy for severity of pylorospasms. In fact, if the suggested measure can be validated further, it may serve as a future clinical endpoint to targeted treatments for the gastroparetic syndrome.

Furthermore, the WMC provides the ability to measure antral and duodenal motility indices. In addition, it can reveal numbers of antral/pyloric alkalic exposures representing duodenogastric reflux caused by incomplete function of the pyloric sphincter, juxta pyloric dyscoordination, and exposure of duodenal content to an "entrapped" "capsule" caused by pylorospasms or it may be MMC-phase 3 related. Previous observations of increased antral pH in dogs [23] and in humans [24] support our findings.

On the other hand, quantification of duodenal acidic exposures represents likely gastric content spill-over caused by incomplete function of the pyloric sphincter, juxtapyloric dyscoordination or true retrograde movements of the capsule.

The physiological inhibitory ileal break mechanism is dissociated from the physiological inhibitory effects of acute hyperglycemia, however, in diabetes the two mechanisms co-exist and may exaggerate the duodenal response of each other, leading to duodenogastric reflux.[25,26]

Although frequently overlooked in the clinic, partly due to its silent nature diabetic autonomic neuropathy coexists with diabetes induced CNS-alterations and peripheral somatic or visceral polyneuropathy. It has been shown that the systemic neuronal impairment is associated with generation and maintenance of GI symptoms in diabetes.[27-29] The reported nausea was milder than reported by others,[30-32] conceivably reflecting that gastroparesis was not an inclusion criteria. Nonetheless, in patients with concomitant verified DSPN, nausea, and gastric emptying were consistently associated with diminished gastric and duodenal MI, which reasonably reflects involvement of pylorospasms prior to gastroparesis *per se*. Furthermore, hyperglycemic exposure of the GI tract, negatively impacts the enterocytes and the enteric nervous system, leading to decreases enteric specialized functions such as hormonal secretion and uncoordinated motility and regulation.[33,34] Taken together, it is not a surprise that the more severe findings of pylorospasms shown as remarkably prolonged antroduodenal transition time, diminished MI, and more frequent episodes of duodenogastric reflux in the diabetes patients with concomitant DSPN.[35]

In conclusion, the WMC may offer a clinical applicable alternative to evaluate a proxy for severity of pylorospasms in patients with diabetes or other neurogastroenterological dysfunctions. If successful, these findings may ultimately optimize future pharmacological therapy and management. The future development of the concurrent measurements of the WMC with the capsule trajectories could further explore the antroduodenal dissociation of the underlying mechanisms including tonic pyloric pressure, lack of antroduodenal coordination, pyloric dysfunction, and/or abnormal duodenal motility.

CONFLICT OF INTEREST

No competing interests declared

AUTHOR CONTRIBUTION

CB and AMD designed the study as a concept of diabetic enteropathy. CB wrote first draft; DL analyzed data, AW analyzed and collected the experimental data, All authors contributed to interpretation of data, intellectual discussion and reviewed the manuscript.

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