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DAY-TO-DAY REPRODUCIBILITY OF PROLONGED AMBULATORY COLONIC MANOMETRY IN HEALTHY SUBJECTS

Satish SC Rao, Siddharth Singh, and Ranjit Mudipalli

Division of Neurogastroenterology, Department of Internal Medicine, University of Iowa Carver College of Medicine, Iowa City, Iowa

Abstract

Background—Although colonic manometry provides useful information regarding colonic physiology, considerable variability has been reported both for regional motility and manometric patterns. Whether colonic manometry is reproducible is not known.

Methods—Seven healthy volunteers (m/f= 3/4, mean age = 34 yrs) underwent two studies of 24-hour ambulatory colonic manometry, each two weeks apart. Manometry was performed by placing a 6-sensor solid-state probe, up to the hepatic flexure and anchored to colonic mucosa. Colonic motility was assessed by the number and area-under-curve (AUC) of pressure waves and motility patterns such as high-amplitude propagating contractions (HAPC). Waking and meal-induced gastrocolonic responses were also assessed. Paired t-test was used to examine the reproducibility and intra- and interindividual variability.

Results—The number of pressure waves and propagating pressure waves and HAPC, and AUC were similar between the two studies. Diurnal variation, waking and meal-induced gastrocolonic responses were also reproducible. There was some variability in the incidence of individual colonic motor patterns.

Conclusions—Colonic manometry findings were generally reproducible, particularly for the assessment of key physiologic changes, such as meal-induced gastrocolonic, HAPC and waking responses.

Keywords

Ambulatory colonic manometry; reproducibility; intraindividual and interindividual variation

INTRODUCTION

The motor activity of the colon is complex, and shows temporal and spatial variations.^{1–4} Previous studies of colonic motility have reported inconsistent results both, in humans^{5,6} and in animal models.⁷ These discrepancies may be due to methodological shortcomings such as the use of non-ambulatory studies,^{8,9} recordings from either limited colonic segments^{10–13} or for short periods,^{14,15} use of different recording systems^{8,9,12} and studies performed after extensive cleansing.^{16,17} Over the last decade, the use of prolonged 24-hour ambulatory colonic manometry, with multiple sensors, and recordings obtained from multiple colonic segments, has provided a better understanding of overall colonic motor activity^{1,13}. Using this technique, we and others have identified seven different

Address Correspondence to: Satish SC Rao, M.D., Ph.D., FRCP (Lon), Department of Internal Medicine, Division of Gastroenterology/Hepatology, University of Iowa Hospitals and Clinics, 4612 JCP, Iowa City, Iowa 52242, Telephone: (319)-353-6602, FAX: (319)-353-6399, Satish-rao@uiowa.edu.

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patterns of colonic phasic pressure activity in normal healthy adult subjects.^{1,5,13} Also, previous studies using this technique have provided a better understanding of the pathophysiology of slow-transit constipation and has identified features suggestive of colonic neuropathy versus colonic myopathy.^{18,19} This technique has also been used in patients with irritable bowel syndrome²⁰, where abdominal pain may be related to specific colonic motor patterns as well as in fecal incontinence²¹ and inflammatory diarrhea.²² In pediatric practice, this technique has been used to select medical and surgical treatment options when conventional management of constipation has failed.^{23,24}

However, considerable intersubject and intraindividual variation has been reported in colonic motor activity.^{6,25} A significant day-to-day variability in colonic manometry may pose significant problems regarding the accuracy of findings, particularly when comparing different groups, or when studying the influence of therapy on clinical outcome. It is unknown if colonic manometry is a valid and reproducible technique.

Our hypothesis was that studies of colonic manometry are reproducible and stable over time. Therefore, our aim was to investigate the intra-individual and day-to-day reproducibility of prolonged 24-hr colonic manometry in healthy individuals.

METHODS

Subjects

Seven healthy volunteers (3 men, 4 women, median age 34 yrs, range 22 – 47 yrs) were recruited through a hospital advertisement. All subjects gave written informed consent, and the study protocol was approved by our Human Investigation Review Board. The volunteers were in good health, had no previous history of gastrointestinal symptoms or surgery, were not taking any medications other than multivitamins or oral contraceptive pills, and had normal physical examination. They all reported normal bowel function.

Manometry assembly

We used a 6-mm diameter flexible probe containing six strain-gauge pressure transducers (Koningsberg Instruments, Pasadena, CA). The transducers when correctly positioned, were approximately located at 10, 20, 35, 50, 70 and 90 cm from the anus, with the most proximal sensor located around the hepatic flexure. We chose variable distances between the transducers in order to optimize their location in the rectum, sigmoid colon, descending colon, and transverse colon. The probe was attached to an ambulatory recorder (Type 7-MPR, Gaeltec Ltd., Isle of Syke, UK). The recorder had a sampling frequency of 8 Hz and was engineered to compress data and store one megabyte of information using a selective data acquisition algorithm. This allowed us to record colonic motility continuously in all subjects. After completing the prolonged ambulatory recording, the data was transferred to an IBM PC and was stored on a hard drive for future analysis.

Experimental Design

After an overnight fast, the subjects were admitted to the Clinical Research Center. At 7:00 AM, they received a 750ml tap water enema. Thereafter, using the following technique, the manometry probe was placed in the colon. A silk thread was tied to the tip of the probe and at three other sites along the shaft of the probe. The thread at the tip was grasped by a snare introduced through the biopsy channel of a pediatric colonoscope (Olympus GIFC10, Japan). The snare was pulled back so that its tip lay 2–3 cm inside the distal end of the instrument. Using minimal sedation (Midazolam 3 to 5mg IV), the probe and the colonoscope were advanced under direct vision up to the hepatic flexure, with minimal air insufflation. Once the location of the probe was confirmed by fluoroscopy, the silk thread

was released, freeing the probe. Subsequently, a clip-fixing device was introduced through the biopsy channel and the silk sutures located at the tip and at three other sites on the probe were clipped to the colonic mucosa using mucosal clips (Olympus America Inc, Melville, NY).^{26,27} The snare was removed. The colonoscope was withdrawn, after decompressing the colon. The probe was then taped securely to the gluteal region. Fluoroscopy was repeated the next morning and again at the end of the study to assess the location of each sensor. The total radiation exposure for each individual did not exceed 1144 μ rads.

The recorder was placed in a shoulder bag and the subjects were free to ambulate throughout the study. At 9:00 AM, all subjects received a 400-Kcalorie snack. At 6:00 PM and again the next morning at 11:00 AM, they received a standardized 1000-Kcalorie meal. The subjects were allowed free access to water (maximum 1.5 L/24 h) but were prohibited from drinking alcohol. The subjects slept overnight at the Clinical Research Center from 10:00 PM onward. The next morning, they were awakened at 6:00 AM. The motility recording was continued until 2:00 PM, and thereafter the probe was removed. An event marker was attached to the recorder, and the subjects were encouraged to use this and mark the time of events such as eating, walking, and sleeping or to indicate the occurrence of symptoms such as abdominal pain, passing flatus, etc. They were also provided with a diary, in which they described the event(s) or the symptom(s) and recorded its time and duration. During the course of the study, if the tip of the probe had shifted by more than 15 cm from its original location and could not be repositioned under fluoroscopy or the probe was expelled, the data from that subject was excluded. After the recording was completed, by gently tugging on the probe it was removed.

Two weeks later, all 7 subjects underwent a repeat procedure, using an identical protocol.

Data analysis

The pressure activity that was recorded from each transducer was analyzed by observing the manometry tracings on a monitor and by using a software analysis program [AMBB, Gaeltec Ltd, Dunvegan, Isle of Skye]. Pressure waves with an amplitude ≥ 8 mm Hg and a duration ≥ 3 s were included in the analysis. Physical activity such as walking or coughing was associated with movement artifacts that were confirmed with the help of the subject's diary, and these segments were excluded from the analysis. In order to exclude the potential effects of instrumentation, sedation and probe placement on colonic motility, we discarded the manometric data from 9:00 AM to 2:00 PM on day one. The 24 hr recording obtained from 2:00 PM on day 1 until 2:00 PM on day 2 was analyzed.

Generally, colonic pressure activity was complex and variable. We were able to identify all seven colonic manometry patterns as previously described.¹³ We were especially interested in propagating pressure waves, defined as pressure events that migrated aborad across three or more consecutive channels with a velocity > 0.5 cm/s (16), periodic rectal motor activity (PRMA), defined as periodic motor activity seen in the rectosigmoid colon, which consisted of discrete bursts of phasic and tonic pressure waves with a frequency of ≥ 3 per min and a cycle duration of ≥ 3 per min and high amplitude propagating contracting (HAPC) (also known as specialized propagating pressure wave¹³ or high amplitude propagating sequences¹), defined as pressure wave sequences that migrated aborad across three or more consecutive channels with an amplitude ≥ 105 mm Hg and duration ≥ 14 s.¹³ These waves are most likely involved in moving gas and/or feces along the colon.^{2,13,28} We measured the incidence and the maximum amplitude for each sequence of pressure waves. For each hour and at each pressure sensor, we measured the number of waves and the area under the curve (AUC) of pressure waves, as a means of quantifying colonic pressure activity.¹³ For the purposes of examining the diurnal variation, we divided the 24-hour recording into three eight-hour epochs. The pressure activity that was recorded between 2:00 PM and 10:00 PM

was taken as an index of Day 1 motor activity. The motor activity that was recorded between 10:00 PM and 6:00 AM was taken as an index of nighttime motor activity. The following day, the pressure activity that was recorded between 6:00 AM and 2:00 PM was taken as an index of Day 2 motor activity. For each of these epochs, we calculated the incidence of the aforementioned patterns and the various other parameters of pressure activity. Because previous studies had suggested that the early morning waking response and gastocolonic motor response are important parameters for assessing colonic neuromotor integrity,¹⁸ we analyzed the colonic pressure activity for a one hour period before and after waking as well as 1-h preprandially and 1-h and 2-h post-prandially.

Statistical analysis

The data are expressed as the mean $\pm$ standard error of mean (SEM). The statistical differences within a group for colonic pressure activity (number of pressure waves/hr, mean AUC/hr, mean number of propagating waves/hr and mean number of HAPCs/hr) between day 1, nighttime and day 2 were compared by using repeated measures ANOVA.

Reproducibility of colonic manometry was assessed by comparing the colonic pressure activity between the two studies, using the paired t-test. Waking and gastrocolonic response were also assessed in the two studies independently, using Student's t-test, to assess reproducibility. Intra-individual variability was summarized as the mean difference and corresponding 95% confidence intervals. The coefficient of variation (%) for intra-individual variability was calculated by dividing the standard deviation (SD) of differences between the initial and repeat studies by the overall average of both studies. Interindividual coefficient of variation was calculated by averaging the coefficient of variation for initial and repeat studies. A p value of <0.05 was taken as significant. All analyses were performed using the SPSS software (SPSS 14.0, Illinois, Chicago).

RESULTS

Subjects and Symptoms

All of our patients tolerated the colonic manometry probe placement and completed the studies without any adverse event. We analyzed 336 hours of colonic motility recording, from 2 sets of data from 7 healthy subjects. There were 8 events of defecation during the course of the 2 studies and these were observed in 4 subjects. Of these, 2 subjects had 2 episodes of defecation during each study and two other subjects had two episodes each. All defecation episodes were preceded by HAPCs. There was no difference in the number of defecation episodes during study 1 when compared to study 2. At the end of the study, in all of these subjects, the most proximal sensor was located at the hepatic flexure or in the proximal transverse colon and without significant migration. No significant adverse events were noted during probe placement or its removal.

Colonic pressure activity

The total number of pressure waves/hr during study 1 was similar to those observed in study 2 (Table 1). Likewise, the AUC for pressure waves for the two studies during the 24-hr period were comparable and similar (Table 1). There was a significant diurnal variation with a reduction in the number of pressure waves and AUC of pressure waves during nighttime when compared to daytime, both during study 1 and during study 2, and was similar for both studies (Figure 1,2).

Propagating pressure waves, PRMA and HAPC

Seven colonic motor patterns were identified during both studies as described previously¹³ but our analyses were limited to propagating pressure waves and high amplitude propagated contraction waves.

The total number of propagating pressure waves/hr was comparable between the two studies (Table 1). Propagating pressure waves also demonstrated the normal circadian rhythm with fewer waves at nighttime when compared to daytime, particularly on day 2. Also, this pattern was reproducible during both studies (Figure 3). The incidence of PRMAs (no. of cycles per 8-hr epoch) in the rectosigmoid region was comparable between the 2 studies [Study 1 vs Study 2, 10.0 vs. 6.4; $p=NS$]. There was trend towards nocturnal predominance of PRMAs in both studies, although the difference was not statistically significant [Study 1, 9.6 vs. 10.9 (daytime vs. nighttime PRMAs), Study 2, 5.4 vs. 8.3 (daytime vs. nighttime PRMAs); $p=NS$]

Similarly, high amplitude propagated contractions were detected in all subjects and during both studies. The total number of high-amplitude propagated contractions/hr was comparable between the two studies (Table 1). The number of HAPCs was significantly less during nighttime when compared to daytime in both studies [Study 1, 0.9 vs. 0.4 (daytime vs nighttime HAPCs/hr), $p<0.05$; Study 2, 1.0 vs. 0.6 (daytime vs nighttime HAPCs/hr), $p<0.05$]. The amplitude of HAPCs was comparable in both the studies [Mean(SEM) mmHg, Study 1 vs Study 2, 151.3(10.3) vs. 160.6(8.6); $p=NS$]. Most HAPCs originated at the most proximal sensor, i.e., at or proximal to the transverse colon, in both studies (Study 1 vs. Study 2, 75% vs. 78%; $p=NS$).

Regional variation

There were no consistent differences in the colonic pressure activity or colonic motor pattern between the transverse colon, descending colon and rectosigmoid colon in both studies (data not shown). In study 2, mean number of pressure waves was lower in transverse colon than the descending colon, on day 1 (Table 2).

Colonic motor response to waking

Colonic pressure activity showed ~3 fold increase in number of pressure waves and AUC within 1-h of waking, as compared to 1-h immediately pre-wake, and was similar and reproducible for the two studies. There was also an increase in the number of propagating pressure waves and HAPCs, although this was not statistically significant (Table 3).

Gastrocolonic response

Ingestion of a meal was associated with a significant ~2-fold increase in the number of waves and in the AUC of pressure waves, both at 1-h and 2-h postprandially and were similar and reproducible for the two studies. There was a consistent, though statistically insignificant, trend in post-meal increase in number of propagating pressure waves and HAPC in both studies (Table 3).

DISCUSSION

Studies of colonic manometry have previously shown significant temporospatial variation in colonic motility.¹⁻⁴ Also, several factors have been identified that account for the variability in colonic motor activity, which could be considered under two broad categories - intrinsic or “colon-dependent” and extrinsic or “technique-dependent”.

Intrinsic sources of variability include the variability of colonic motor patterns, with as many as seven different identifiable motor patterns that occur without a clear cyclical/recurring pattern,¹³ the diurnal variation and the changes after awakening^{13,29} and after a meal or type of meal.^{3,14,30,31} Also, gender,^{13,32} age,³³ sleep pattern,¹¹ stress,^{3,34} and differences in colonic contents may influence intrinsic activity of the colon.⁵ Extrinsic factors include sensor localization^{10,21} and migration,^{26,27} non-physiological conditions (sedation, prolonged immobilization, stressful laboratory environment, bowel preparation with complete evacuation of stool)^{16,17} and use of different instrumentation (solid-state manometers vs. perfused tube manometry).^{8,9,12}

In order to minimize these, we performed our study in healthy individuals, with almost equal sex distribution. Both studies were performed in identical, physiological conditions with use of minimal sedation, in ambulatory subjects. We excluded the initial 5-hr of recording, to minimize the effects of sedation and tap water enema preparation on colonic motility. In addition, all subjects received standardized meals and were instructed to go to bed and wake up at the same time. Thus, we sought to minimize the influence of extrinsic factors on colonic motility recording.

We found that the number of pressure waves and the area-under-curve of pressure waves were similar during two separate studies in the same individual. Also, when the data was examined in 8-hr time periods, separately, there was remarkable similarity in the corresponding periods for the two studies. There was, however, some intra- and interindividual variability in the two studies most likely related to intrinsic colonic variation.

Furthermore, assessment of colonic motor patterns as evidenced by high-amplitude propagated contractions and propagating pressure waves, demonstrated that the overall incidence of these patterns were similar between the two studies, although there was some intra- and interindividual variability. Previously, a wide intersubject variation in HAPCs has been reported in normal subjects, with a range 6–25 HAPCs per 24-hr and 0–40 HAPCs per 24-hr, respectively.^{2,21} This could be due to intrinsic colonic variability as well as the use of varying definitions of HAPCs (for example, HAPCs defined by pressure waves >50mm Hg vs. >100mm Hg amplitude),²⁵ as well as the sensitivity of the transducer.¹³

Circadian variation in colonic motility has emerged as a consistent feature of 'normal' colonic motility.^{13,29,35,36} Such a variation is also identifiable in patients with slow-transit constipation, although with generalized blunting of response. Circadian variation was reproducible within our subjects.¹⁸ Also, in our study, we found that both the waking and gastrocolonic responses were reproducible, with no variability between studies. These responses are most likely mediated by neurohormonal mechanisms as well as by the central nervous system and enteric nervous system.^{5,30,31}

Colonic manometry has been utilized in adults with slow-transit constipation to identify manometric patterns suggestive of colonic neuropathy and myopathy.¹⁸ Absence of HAPCs, meal-induced gastrocolonic response and/or waking response, i.e., absence of any 2 of these 3 physiological responses, has been proposed as a manometric basis for identifying colonic neuropathy. In contrast, the presence of any 2 of these 3 responses, but with a magnitude of response that is less than 2 standard deviations of the normal range has been proposed as evidence of colonic myopathy. It has been shown that patients with colonic neuropathy are unlikely to respond to conservative pharmacological management, and are more likely to benefit from surgical intervention, whereas patients with colonic myopathy show significant benefit from prolonged medical management.¹⁸ Hence, these key elements of colonic motor function, namely, HAPC response, meal-induced gastrocolonic response

and waking response help us to identify subsets of patients with slow-transit constipation suitable for surgical intervention.

The limitations of our study include the small number of subjects who were examined, but were felt to be adequate to explore whether there was significant day to day variability within subjects during a prolonged 24 hour invasive and ambulatory study. Furthermore, given the limitations of commercial solid state technology, we could only examine motility at 6 sites in the colon, which may have reduced the spatial resolution of propagating sequences. Finally, our manometry probes were designed to reach the hepatic flexure and hence cecal and ascending colon motility could not be evaluated.

In conclusion, prolonged ambulatory colonic manometry appears to be a valid and robust measurement of colonic motility and its findings are generally reproducible in the assessment of motility, although there is some intra- and interindividual variation. The technique reproducibly identifies circadian variation, gastrocolonic and waking responses and high amplitude pressure contractions, which are key elements of this investigation and have been shown to be altered in several colonic disorders. Thus, our findings, in conjunction with recent findings on reproducibility of colonic barostat for assessment of colonic compliance, tone and sensory function³⁷ suggests that together, colonic manometry and barostat studies provide a robust, comprehensive technique of assessing colonic sensorimotor function in health and disease.

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Figure 1.

Number of pressure waves/hr for the three 8-hr epochs (Day 1, Night and Day 2) in studies 1 and 2 for each individual subject with variability.

*-Study 1, Day 1 and Day 2 vs. Night, $p < 0.05$; ‡-Study 2, Day 1 and Day 2 vs. Night, $p < 0.05$; †-Study 1, Day 1 vs. Day 2, $p < 0.05$

Figure 2. Mean Area Under Curve (AUC) for the three 8-hr epochs (Day 1, Night and Day 2) in studies 1 and 2 for each individual subject with variability.

*-Study 1, Day 1 and Day 2 vs. Night, $p < 0.05$; ‡-Study 2, Day 1 and Day 2 vs. Night, $p < 0.05$; †-Study 1, Day 1 vs. Day 2, $p < 0.05$

Figure 3.

Mean number of propagating pressure waves/hr for the three 8-h epochs (Day 1, Night and Day 2) in studies 1 and 2 for each individual subject with variability.

*-Study 1, Day 1 and Day 2 vs. Night, $p < 0.05$; #-Study 2, Day 1 and Day 2 vs. Night, $p < 0.05$

Table 1

Reproducibility, with intra- and interindividual variability, for Number of waves/h and Area Under the Curve (mm Hg*s), throughout the colon (P1–P6). Please note there was no significant difference between studies 1 and 2.

	Study 1 Mean (SEM)	Study 2 Mean (SEM)	Mean difference (95% CI for difference between studies 1 and 2)	Coefficient of variation (%)	
				Intra- individual	Inter- individual
No. of pressure waves (per hr)	84.8 (9.2)	83.8 (4.8)	0.9[49.1, -47.3]	28	22
AUC (mmHg*s×10 ³) (per hr)	11.7 (1.2)	11.3 (1.1)	0.4[10.0, -9.2]	41	26
No. of propagating pressure waves (per hr)	4.0 (0.4)	3.9 (0.5)	0.1[2.0, -1.8]	24	30
No. of HAPCs (per hr)	0.7 (0.1)	0.8 (0.4)	-0.1[0.5, -0.7]	39	25

Table 2

Number of waves/h and Area Under the Curve (mm Hg*s $\times 10^3$) for the Transverse Colon (P1–P2), Descending Colon (P3–P4) and Rectosigmoid Colon (P5–P6) in healthy controls in 2 distinct studies, for the three 8-h periods of Day 1, Night and Day 2. Please note there was no significant difference between studies 1 and 2 in the corresponding 8-h periods.

		Study 1			Study 2		
		Day 1	Night	Day 2	Day 1	Night	Day 2
Mean No. of waves	Transverse	94.3(13.2)*	52.3(9.3)	115.7(11.1) [†]	75.5(9.6) ^{††}	47.8(10.7)	82.2(13.3)
	Descending	93.2(13.7)*	43.6(8.6)	101.6(6.6) [†]	105.1(9.7) [§]	54.4(7.0)	109.4(4.0) [¶]
	Rectosigmoid	82.5(15.0)	58.9(9.6)	119.9(14.6) [†]	105.7(12.6)	69.6(15.5)	90.3(11.9)
AUC (mm Hg $\times$ s) $\times 10^3$	Transverse	8.7(1.2)	5.6(1.3)	13.1(1.8) [†]	8.4(1.7) ^{††}	4.9(1.3)	9.7(1.9) [¶]
	Descending	12.0(1.6)*	5.8(1.2)	15.1(1.2) [†]	13.9(1.2) [§]	7.1(1.1)	16.4(1.3) [¶]
	Rectosigmoid	10.6(1.9)	8.3(1.5)	19.1(2.5) ^{†‡}	15.0(2.4) [#]	9.0(1.9)	15.8(2.1) [¶]

* Study 1, Day 1 vs. Night, $p < 0.05$;

[†] Study 1, Day 2 vs. Night, $p < 0.05$;

^{†‡} Study 1, Day 1 vs. Day 2, $p < 0.05$;

[§] Study 2, Day 1 vs. Night, $p < 0.05$;

[¶] Study 2, Day 2 vs. Night, $p < 0.05$;

^{††} Study 2, Day 1, Transverse colon vs. Descending colon, $p < 0.05$;

[#] Study 2, Day 1, Transverse colon vs. Rectosigmoid colon, $p < 0.05$.

Table 3

Waking and gastrocolonic response in healthy controls in 2 distinct studies. Please note there was no significant difference between studies 1 and 2.

	Study 1			Study 2		
	Pre-waking	Post-waking		Pre-waking	Post-waking	
WAKING RESPONSE						
	No. of pressure waves					
	55.1(11.0)	133.2(19.9) [‡]		52.5(8.4)	136.3(14.2) [#]	
	AUC (mmHg*s)×10 ³					
	7.7(1.5)	20.9(3.3) [‡]		5.7(1.2)	20.2(3.9) [#]	
	No. of propagating pressure waves					
	3.4(0.6)	5.3(1.1)		4.6(0.8)	5.3(1.4)	
	HAPCs					
	0.4(0.2)	1.1(0.7)		0.4(0.3)	0.7(0.3)	

		Study 1				Study 2			
		Preprandial	1-h postprandial	2-h postprandial	Preprandial	1-h postprandial	2-h postprandial		
GASTROCOLONIC RESPONSE	No. of pressure waves	83.0(6.4)	162.9(13.2) [*]	144.2(8.7) [‡]	84.0(7.5)	142.6(12.6) [§]	128.9(9.3) [¶]		
	AUC (mmHg*s)×10 ³	11.0(1.7)	22.4(2.3) [*]	18.4(1.8) [‡]	9.1(2.0)	17.9(3.0) [§]	15.1(2.8) [¶]		
	No. of propagating pressure waves	3.3(0.8)	4.4(0.8)	3.8(1.1)	3.5(0.7)	5.6(0.8)	6.4(1.0) [¶]		
	HAPCs	1.0(0.4)	1.4(0.6)	1.2(0.2)	0.6(0.2)	0.8(0.4)	0.7(0.2)		