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# Resistance of postprandial gastric functions and autonomic balance to taste stimulation

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Submitted October 13, 2021; accepted in final form November 8, 2021

## Abstract

Exposure to unpleasant tastes leads to disturbances of interdigestive gastric myoelectrical activity (GMA) and may affect sympathetic/parasympathetic balance (SPB). We made a careful study to determine whether taste stimulation modulates the postprandial GMA, SPB, and gastric emptying (GE) of a solid meal. Eighteen healthy volunteers (9F/9M) entered the study. On six separate days, we recorded a four-channel electrogastrogram from each volunteer during a 35-min fasting period, then for 90 min after ingestion of a solid test meal of 300 kcal. GE was measured using a <sup>13</sup>C-octanoic acid breath test. Heart rate variability (HRV) analysis was simultaneously performed. At the start of the 21st min after the test meal, subjects received an agar cube delivering either a sweet, salty, sour, or bitter taste, which they kept in the mouth for 35 min. Control procedures involved sessions performed with a tasteless agar cube, and without any stimulation. There was no effect of the experimental intervention upon the relative power share of particular GMA rhythms. Stimulation with the salty and the bitter taste evoked a statistically significant increase in the dominant frequency, whereas the sweet and sour taste did not affect it. Taste stimulation did not interfere with the meal-induced rise in the dominant power, nor affect slow wave coupling. The kinetics of the solid GE remained unchanged by the intervention. None of the taste stimulations affected the postprandial SPB. Taste stimulation elicited after ingestion of a meal, in contrast to that during a fast, did not adversely modify the postprandial pattern of either the GMA or SPB, nor affect the GE of solids.

Key words: electrogastrography, gastric emptying, gastric myoelectrical activity, heart rate variability, taste stimulation

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# Introduction

Apart from breathing, sleeping and possibly thinking, intake of nourishment is one of the basic activities of the *Homo sapiens* species. Considering the basal instinctive exigencies of our species, the need to sate hunger is in second place, just behind our procreative needs. The intake of food triggers a complex set of physiological processes such as myoelectrical, motor, and secretory events, as soon as our senses detect the presence of an appetizing (or may be at times unappetizing) dish. With regard to motility, the cardinal change consists in a switch from the migrating myoelectrical and motor complex, governing during the interdigestive phase, to a characteristic postprandial pattern of gastrointestinal electrical and contractile activity. In a previous paper we reported on investigations aimed at determining if taste stimuli may influence the gastric myoelectrical activity during the interdigestive phase. A principal finding of that research was that stimulation with tastes subjectively perceived as disgusting (bitter, salty, and sour) evokes prominent disturbances of the gastric myoelectrical activity (GMA), which may be considered an evolutionary archetype of a warning reaction of the human organism towards noxious food (1). The present study addresses the effect of taste stimulation upon the postprandial GMA and gastric emptying of a solid meal, in combination with the effect on autonomic balance.

# Methods -

The study was conducted in 18 non-smoking volunteers, 9 women and 9 men (age:  $24.7 \pm 1.8$  years, body mass index:  $22.00 \pm 1.51$  kg/m<sup>2</sup>) fulfilling the World Health Organization criteria of good health (2). Everyone had a negative <sup>13</sup>C-urea breath test result for *Helicobacter pylori* infection. Exclusion conditions comprised current use of any drugs, history of surgery affecting the anatomy of the digestive tract (except for an appendectomy), and pregnancy. Informed, written consent was obtained from all subjects and the study was performed in accordance with the Declaration of Helsinki. The project was approved by the Bioethics Committee of the Medical University of Silesia.

#### Initial gustatory examinations

Individual taste recognition thresholds of the sweet, bitter, sour, and salty taste were determined following the sip and spit approach described in detail in the International Standard ISO3972 (3) with one modification consisting in the replacement of caffeine with quinine hydrochloride as a bitter taste standard. In the latter case a set of eight concentrations was used: 0.0656; 0.1166; 0.2075; 0.369; 0.628; 1.146; 2.071 and 3.699 mg/l.

#### Preparation of agar cubes for taste exposure

Aqueous solutions of saccharose, NaCl, citric acid or quinine hydrochloride were prepared *ex tempore* at a concentration greater than 100-fold of the taste recognition threshold of the individual. Then 100 ml aliquots were heated until boiling before adding 2 g of agar (Arche Naturprodukte GmbH, Hilden, Germany) while stirring intensely. When a solution was ready, it was poured into a form, cooled steadily and finally put into a refrigerator to set.

# Study protocol

The subjects came to the laboratory in the morning after a 12-h overnight fast. For electrogastrographic recording six Red Dot class Ag/AgCl electrodes (type 2222, 3M Canada, London, ON, Canada) were placed on the abdomen and connected to Polygraf ID (Synectics Medical, Denmark) (4). Seven Ag/AgCl electrodes (type

R-LLL-510, Bio Lead-Lok, Józefów, Poland) were fixed to the thorax for the purpose of a continuous electrocardiographic recording with an AsPEKT 702 recorder (Aspel, Zabierzów, Poland) (5).

According to the randomization scheme, each of the volunteers participated in six research sessions held on separate days. A numbered list of predefined sequences of interventions (exposure to the four tastes, a tasteless cube, and a no-cube session) was prepared before commencement of investigations. A given sequence was then allotted by the laboratory staff to subjects consecutively entering the study.

The examination started with a basal 35-min record of GMA and electrocardiogram (ECG) accomplished in a sitting position. At the end of this epoch two samples of expiratory air were collected into glass vials (Exetainer<sup>®</sup>, Labco Ltd., Lampeter, UK) for determination of the basal <sup>13</sup>CO<sub>2</sub> content.

At time "0" the subjects were served a solid test meal—a 1,255 kJ (300 kcal) pancake containing within the solid phase 68 mg of <sup>13</sup>C-octanoic acid (code INC610P, Euriso-Top, Saint Aubin, France; the manufacturer certifies its  $\geq$ 99% enrichment in <sup>13</sup>C). The detailed description of the preparation procedure is given elsewhere (6). Ten minutes were allowed for consumption. The volunteers received 200 ml tap water to wash down the meal.

After elapse of 20 min from time "0", a 35-min exposure intervention took place. A subject was given an agar cube containing one of the four taste delivering substances. Moreover, two control routines were used: administration of a tasteless cube (made of agar only) and a session without administration of a cube. During a session with a cube, it was placed into the oral cavity between the tongue and the palate. The volunteers had to keep it in place as motionless as possible, so as to avoid mastication movements. Moreover, instruction was given not to swallow the saliva but to spit it into a provided container. A disintegrated/melted agar cube was immediately replaced by another one. The GMA and ECG recording was performed during the exposure and for 35 min following it.

After the completion of each session the volunteers rated the sensations experienced during the exposure from three aspects: displeasure/pleasure (score ranging from -10 to 10 points), intensity (0 to 10 points), and nausea feeling (0 to 10 points) with the use of visual analogue scales. In order to check if the sensations perceived during taste stimulation differed between the postprandial exposures and those applied in the fasted state, we used data obtained in our former study (1). Such comparison is valid because in the former study an almost identical group of subjects was examined: 10 women and 8 men (age:  $22.3 \pm 0.7$  years, body mass index:  $22.26 \pm 0.87$  kg/m<sup>2</sup>) (1).

Expiratory air samples were collected for a total of six hours counted from time "0": every 10 min during the first hour and then at 15-min intervals for five hours.

#### Analysis of the recordings

The GMA records and the ECGs were analysed off-line by an operator blinded to the experimental interventions.

Electrogastrograms were analysed with the use of dedicated software (Polygram Net<sup>™</sup> EGG 311224, Medtronic A/S, Skovlunde, Denmark). The following parameters were derived: dominant frequency (DF), dominant power (DP), relative share of the normogastria-range (2.0–4.0 cycles per minute, cpm), bradygastria-range (0.5–2.0 cpm), and tachygastria-range (4.0–9.0 cpm) power within the power density vector, average percentage of slow wave coupling (APSWC) (7).

ECGs were subjected to a spectral heart rate variability (HRV) analysis within the frequency domain performed with the HolCARD 24W v5.10 software (Aspel S.A.). The power within the low frequency (LF: 0.04–0.15 Hz) and high frequency (HF: 0.15–0.40 Hz) band was expressed as a percentage of the total power

of the whole frequency range being analyzed. The LF/HF ratios were calculated (5).

The above analyses comprised the 30-min basal fasted period, as well as the 30-min exposure epoch and the 30-min post-exposure period during the postprandial phase.

## Gastric emptying

The  ${}^{13}\text{CO}_2$  content in the expiratory breath samples was determined with the use of isotope ratio mass spectrometry (ABCA, Automated Breath  ${}^{13}\text{C}$  Analyser, SerCon Ltd., Crewe, UK). A quality control procedure involved a run of the measurement on a standard gas (5% CO<sub>2</sub> within N<sub>2</sub>) of a certified  ${}^{13}\text{CO}_2$  content of -31.33 ‰ before and after every series of breath air samples (8).

Duration of the lag phase (T\_Lag) and gastric half emptying time (T1/2) were taken as the measures of the gastric emptying of the solid meal (6).

## Statistics

Depending on the distribution, the results obtained were subjected either to a repeated measures analysis of variance (ANOVA) followed by Tukey's honest significant difference (HSD) test, or to Friedman's ANOVA followed by a Wilcoxon signed rank test. If applicable, Mood's median test was used. Statistical significance was set at the P<0.05 level, two-tailed. Results are presented as means ± SE or as medians with interquartile ranges (9).

#### Results

The median taste recognition thresholds amounted to (data in brackets are interquartile ranges)—sweet: 4.32 g/l saccharose (3.02; 4.32), salty: 0.24 g/l NaCl (0.16; 0.32), sour: 0.16 g/l citric acid (0.13; 0.20), and bitter: 0.89 mg/l quinine hydrochloride (0.37; 1.15). The data concerning the subjective ratings of sensations experienced during taste stimulation and statistical comparisons are assembled in Table 1. The sweet taste was perceived as pleasant. Also, a low positive score on the pleasure scale was obtained with the stimulation by a tasteless cube. The other three tastes were scored as definitely unpleasant, with the disgust order: salty>sour>bitter. In accordance with the methodological assumption, the median taste intensity score for the tasteless cube amounted to zero. The perception of the sweet, salty, and sour taste was more intense than of the bitter taste. Nausea was reported infrequently, yet in the case of the salty and the bitter taste the score thereof was significantly higher in comparison to the tasteless cube.

In addition, using the data from our former study (1), comparison of perception of the taste stimulation between the postprandial and the interdigestive state was performed. This analysis revealed that in the case of sweet taste the perception of pleasure was lower during the postprandial compared to the interdigestive state

 Table 1. Subjective ratings of sensations experienced by the volunteers during taste stimulation in the postprandial state

	Tasteless cube	Sweet cube	Salty cube	Sour cube	Bitter cube
Pleasure (score range: -10 to 10)	1 (0; 2)	3.5 (2.25; 7)	-4 <sup>a,b</sup> (-5.75; -2.25)	-2 <sup>a,b</sup> (-3.75; 1)	-1 <sup>a,b</sup> (-2; 1)
Intensity (score range: 0 to 10)	0 (0; 1)	7.5 <sup>a,c</sup> (7; 8)	8 <sup>a,c</sup> (7; 10)	7.5 <sup>a,c</sup> (7; 8)	3.5 ° (3; 4.75)
Nausea (score range: 0 to 10)	0 (0; 0)	0 (0; 1)	0.5 <sup>a</sup> (0; 2)	0 (0; 0)	0 <sup>a</sup> (0; 1)

Data in the table are medians with interquartile ranges (in brackets). Statistically significant of differences were found: a vs. the tasteless cube, b vs. the sweet cube, c vs. the bitter cube.

(Fig. 1, upper panel). Moreover, bitter taste caused significantly less displeasure during the postprandial stimulation. On the other hand, stimulation with salty taste evoked much more unpleasantness in the postprandial compared to the interdigestive state, and the pertinent difference was highly statistically significant (Fig. 1, upper panel). Regarding the intensity score, either salty or sour taste were perceived stronger during the postprandial compared to the interdigestive state; the latter difference was highly statistically significant (Fig. 1, lower panel). The only difference concerning the nausea score was disclosed in the case of the salty taste: 0.45  $\pm$  0.10 fasted vs. 4.61  $\pm$  1.09 postprandially, *P*=0.04.

The numeric data reflecting the relative power share of the particular electrogastrographic rhythms are put together in Table 2. ANOVA did not reveal any significant main effect of the intervention (with three levels: basal fasted—stimulation in fed state—post-exposure period in fed state) on these parameters. Stimulation with the salty and the bitter taste evoked a statistically significant increase in the DF, whereas the sweet and sour taste did not affect it (Fig. 2). Significantly greater than in the fasted state appeared to be also the DF during the post-exposure period on the day of application of the tasteless cube (Fig. 2). Ingestion of the solid test meal brought about a statistically significant augmentation of the DP on every of the six examination sessions (Fig. 3). Taste stimulation did not interfere with this rise (Fig. 3). Nor has the taste stimulation any effect on the slow wave coupling reflected by the APSWC (Fig. 4).





**Fig. 1.** Comparison of the perception of taste stimulation between the postprandial vs. the interdigestive situation; data pertaining to the interdigestive period were taken from Waluga et al. (1).

Table 2. Effect of tast	e stimulatio.	n in the pos	tprandial st	ate upon the	relative por	wer share o	f electrogası	rographic r	hythms witl	hin the pow	er density v	/ector
		Basal fasted	l observation		Sti	mulation pei	riod in fed st	ate	Post	-exposure pe	eriod in fed	state
	Chan 1	Chan 2	Chan 3	Chan 4	Chan 1	Chan 2	Chan 3	Chan 4	Chan 1	Chan 2	Chan 3	Chan 4
No cube session Normogastria (%)	$52.4 \pm 2.9$	44.1 ± 2.9	$46.9 \pm 3.6$	$51.1 \pm 3.3$	$50.4 \pm 3.1$	$52.6 \pm 3.1$	$54.3 \pm 3.8$	$43.7 \pm 2.8$	$53.1 \pm 3.2$	$49.9 \pm 3.2$	$56.1 \pm 3.4$	44.3 ± 2.8
Tachygastria (%)	$21.6 \pm 1.7$	$26.2 \pm 2.5$	$24.6 \pm 2.3$	$20.7 \pm 1.4$	$25.6 \pm 1.6$	$25.1 \pm 1.5$	$22.5 \pm 2.3$	$27.5 \pm 2.0$	$23.7 \pm 1.9$	$25.1 \pm 2.1$	$21.8 \pm 1.6$	$27.9 \pm 1.8$
Bradygastria (%)	$26.0\pm2.4$	$29.8\pm3.0$	$28.6\pm3.5$	$28.2 \pm 2.8$	$24.1\pm2.3$	$22.2\pm2.3$	$23.2\pm2.9$	$28.9 \pm 2.6$	$23.2\pm2.4$	$25.0 \pm 2.9$	$22.1\pm2.6$	$27.8 \pm 2.5$
Tasteless cube session												
Normogastria (%)	$47.2 \pm 2.9$	$43.4 \pm 2.5$	$43.0\pm3.2$	$45.6 \pm 2.5$	$49.8 \pm 3.2$	$50.7 \pm 4.1$	$50.4\pm3.6$	$44.0\pm2.8$	$53.4\pm3.6$	$50.8 \pm 3.7$	$53.3\pm3.7$	$43.6 \pm 2.5$
Tachygastria (%)	$21.9 \pm 1.6$	$24.4\pm1.6$	$26.0\pm1.3$	$23.8 \pm 1.3$	$25.4 \pm 1.6$	$22.5\pm1.6$	$24.3\pm2.1$	$25.9\pm1.6$	$23.7\pm1.7$	$22.6\pm1.6$	$24.6 \pm 1.9$	$26.5\pm2.1$
Bradygastria (%)	$30.9\pm2.9$	$32.1\pm2.2$	$31.0\pm3.0$	$30.6 \pm 2.7$	$24.9 \pm 2.2$	$26.8\pm3.1$	$25.4 \pm 2.5$	$30.1\pm2.6$	$22.9 \pm 2.4$	$26.6 \pm 2.8$	$22.1\pm2.6$	$29.9\pm2.5$
Sweet taste session												
Normogastria (%)	$49.8\pm3.5$	$41.6\pm3.4$	$44.7 \pm 3.1$	$49.8\pm3.0$	$51.2\pm4.3$	$55.7 \pm 3.5$	$50.6 \pm 4.0$	$44.5\pm2.9$	$52.9 \pm 4.1$	$51.8\pm3.3$	$55.0 \pm 4.3$	$42.8 \pm 3.2$
Tachygastria (%)	$22.3\pm2.5$	$26.2\pm2.4$	$24.5\pm1.5$	$22.8\pm1.8$	$22.2\pm2.2$	$19.4\pm1.5$	$22.4\pm1.8$	$24.3\pm1.5$	$22.7 \pm 2.1$	$22.2\pm1.8$	$21.3\pm2.0$	$27.0\pm2.5$
Bradygastria (%)	$27.9 \pm 3.6$	$32.2\pm3.8$	$30.9 \pm 3.9$	$27.4 \pm 2.7$	$26.6\pm3.4$	$24.9\pm3.0$	$27.1\pm3.2$	$31.2\pm3.2$	$24.4 \pm 3.0$	$26.0 \pm 3.2$	$23.8 \pm 3.6$	$30.1\pm3.6$
Salty taste session												
Normogastria (%)	$52.6\pm3.2$	$45.2 \pm 3.4$	$46.5\pm2.5$	$45.5 \pm 3.0$	$50.9 \pm 4.1$	$51.8\pm4.2$	$51.1 \pm 4.0$	$45.2\pm2.9$	$50.8 \pm 3.5$	$48.7\pm2.7$	$54.8 \pm 4.1$	$44.9\pm3.3$
Tachygastria (%)	$19.2\pm1.2$	$22.4\pm2.4$	$22.7\pm1.9$	$23.1\pm1.9$	$23.4\pm1.9$	$23.1\pm2.3$	$22.8\pm1.6$	$26.9\pm2.3$	$25.0 \pm 2.1$	$25.1\pm2.3$	$23.7\pm2.2$	$29.4 \pm 2.5$
Bradygastria (%)	$28.3 \pm 3.0$	$32.4\pm3.3$	$30.8 \pm 3.4$	$31.3\pm3.4$	$25.7\pm2.5$	$25.1\pm3.0$	$26.1\pm3.6$	$28.0 \pm 2.1$	$24.2 \pm 2.4$	$26.2\pm3.5$	$21.6\pm2.7$	$25.7 \pm 2.4$
Sour taste session												
Normogastria (%)	$49.9\pm3.4$	$46.6\pm3.2$	$47.8\pm3.8$	$47.6\pm2.8$	$49.7\pm3.8$	$51.0 \pm 4.1$	$50.3\pm3.4$	$46.6\pm3.0$	$51.8\pm3.9$	$54.6\pm4.0$	$53.1\pm3.9$	$44.6 \pm 3.5$
Tachygastria (%)	$21.6\pm1.7$	$23.2\pm2.1$	$22.6\pm2.1$	$22.9\pm1.8$	$23.8 \pm 2.2$	$23.4\pm2.2$	$24.3\pm2.8$	$25.5\pm2.1$	$26.9\pm2.9$	$23.0\pm2.1$	$22.4 \pm 2.4$	$27.3 \pm 2.1$
Bradygastria (%)	$28.4 \pm 2.9$	$30.2\pm2.7$	$29.6\pm3.3$	$29.6 \pm 2.5$	$26.6 \pm 2.7$	$25.6 \pm 3.3$	$25.4 \pm 2.3$	$28.0 \pm 2.4$	$21.3\pm2.5$	$22.4 \pm 3.2$	$24.5 \pm 3.0$	$28.1\pm2.9$
Bitter taste session												
Normogastria (%)	$49.4\pm3.2$	$43.4 \pm 3.4$	$50.1 \pm 3.1$	$46.7\pm2.9$	$48.9 \pm 3.4$	$50.3\pm3.4$	$51.9\pm3.8$	$45.1\pm2.7$	$52.2 \pm 3.4$	$49.0\pm3.1$	$53.9 \pm 4.5$	$45.3 \pm 3.2$
Tachygastria (%)	$24.7 \pm 1.7$	$28.6 \pm 2.2$	$24.8 \pm 1.6$	$24.6\pm1.9$	$24.3 \pm 2.1$	$23.8 \pm 1.6$	$22.0\pm1.7$	$24.6\pm1.5$	$24.7 \pm 1.7$	$25.5 \pm 2.1$	$22.0 \pm 1.9$	$25.2\pm1.9$
Bradygastria (%)	$25.9\pm2.7$	$28.1\pm2.8$	$25.1\pm2.5$	$28.7\pm2.7$	$26.8\pm2.4$	$25.9\pm2.6$	$26.0\pm3.1$	$30.3\pm2.4$	$23.1\pm2.2$	$25.5\pm2.5$	$24.2 \pm 3.3$	$29.5\pm2.4$
Chan: channel; ANOVA	did not revea	al any signific	cant main eff	fect of interve	ention.							

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Fig. 2. Effect of taste stimulation on the dominant frequency (DF) of the postprandial gastric myoelectrical activity. The following segments of electrogastrograms were considered: F=30-min basal fasted period, as well as S=30-min stimulation epoch and P=30-min post-exposure period during the postprandial phase. Statistical significance of differences: a P=0.023, b P=0.026, and c P=0.010 vs. basal fasted.



**Fig. 3.** Effect of taste stimulation on the dominant power (DP) of the postprandial gastric myoelectrical activity. The following segments of electrogastrograms were considered: F=30-min basal fasted period, as well as S=30-min stimulation epoch and P=30-min post-exposure period during the postprandial phase. The asterisks indicate statistically significant differences vs. the basal fasted situation but for clarity of presentation exact *P* values are omitted.



**Fig. 4.** Effect of taste stimulation on the average percentage of slow wave coupling (APSWC) of the postprandial gastric myoelectrical activity. The following segments of electrogastrograms were considered: F=30-min basal fasted period, as well as S=30-min stimulation epoch and P=30-min post-exposure period during the postprandial phase.

Table 3. Effect of taste stimulation upon the gastric emptying of a solid meal

	No cube	Tasteless cube	Sweet cube	Salty cube	Sour cube	Bitter cube
T_Lag (min)	$125\pm 6$	$134\pm 6$	$135\pm 6$	$127\pm 6$	$137\pm7$	$130\pm5$
T½ (min)	$174\pm8$	$185\pm8$	$186\pm7$	$175\pm7$	$184\pm8$	$178\pm 6$

T\_Lag: lag phase, T<sup>1</sup>/<sub>2</sub>: the gastric half emptying time; ANOVA did not reveal any significant main effect of intervention.

ANOVA did not disclose any significant main effect of intervention upon the gastric emptying of the solid test meal. Hence the lengths of T Lag and T1/2 were similar across the six examination sessions (Table 3).

Ingestion of the solid test meal brought about typical shifts of the HRV parameters: the HF decreased and the LF increased, hence the LF/HF ratio also rose. According to ANOVA, taste stimulation did not affect the postprandial shift of the autonomic balance (Table 4).

# Discussion

While searching through databases, such as *Medline* and *Scopus*, for studies which could be somehow related to our examinations we came across two papers only. Stern et al. (10) examined electrogastrographically the GMA while healthy volunteers were subjected to two sessions of sham feeding of an appetizing or unappetizing food. Wicks et al. (11) registered an electrogastrogram and measured the gastric emptying of a liquid low-calorie meal and had their subjects chew and spit Slim-Fast bars coated with a glaze of bitter or strawberry taste. Thus, our study protocol appears to be different.

The hypothesis we wanted to test was whether stimulation with four tastes would affect the postprandial GMA, gastric emptying, and autonomic balance. Therefore, our stimulation session commenced 20 min after

	No cube		Tasteless cube		Sweet cube	
	Stimulation	Post-exposure	Stimulation	Post-exposure	Stimulation	Post-exposure
ΔHF (%)	$-5.5\pm1.2$	$-6.4 \pm 1.1$	$-3.5\pm1.2$	$-6.7 \pm 1.1$	$-5.2\pm1.7$	$-5.5\pm1.4$
ΔLF (%)	$4.8 \pm 1.3$	$4.9\pm1.1$	$2.4\pm1.4$	$6.4\pm1.2$	$2.4\pm1.3$	$4.1\pm1.4$
$\Delta$ LF/HF (%/%)	$0.19\pm0.04$	$0.21\pm0.04$	$0.10\pm0.05$	$0.26\pm0.04$	$0.12\pm0.05$	$0.18\pm0.06$
	Salt	y cube	Sour cube		Bitter cube	
	Stimulation	Post-exposure	Stimulation	Post-exposure	Stimulation	Post-exposure
ΔHF (%)	$-3.2\pm1.5$	$-4.9\pm0.9$	$-5.1\pm1.2$	$-3.7\pm1.0$	$-2.7\pm1.0$	$-3.6\pm0.9$
ΔLF (%)	$1.8\pm1.8$	$3.9\pm 1.3$	$5.0\pm1.4$	$4.0\pm1.3$	$2.8\pm1.1$	$3.8\pm 1.0$
ΔLF/HF (%/%)	$0.08\pm0.06$	$0.18\pm0.04$	$0.21\pm0.05$	$0.15\pm0.05$	$0.10\pm0.04$	$0.15\pm0.04$

Table 4.	Effect of taste stimulation upon the meal-induced changes in the sympathetic/parasympathetic
	activity balance

Examination periods: a 30-min stimulation period was started 20 min from time "0" i.e. from the beginning of the test meal consumption, and was followed by a 30-min post-exposure observation.  $\Delta$ : difference versus the basal interdigestive period in the heart rate variability parameters: HF: the normalized high frequency (0.15–0.40 Hz) power, LF: the normalized low frequency (0.04–0.15 Hz) power, LF/HF: the ratio LF to HF. ANOVA did not reveal any significant main effect of intervention.

the start of ingestion of the test meal. Accordingly, a transition from the fasted to fed pattern of gastric motility and HRV pattern was assured. In the first place, this was confirmed by a significant rise of the DP of the gastric slow waves. In the second place, a typical meal-induced diminution of the HF power and the increase in the LF power, resulting in the augmentation of the LF/HF ratio has been observed (5). Exposure to one only taste per examination session was assumed. For this purpose, a special taste-delivering system was invented, consisting in preparation of agar cubes containing a taste substance at a concentration adjusted individually to the taste recognition threshold multiplied by a factor of 100 (1). Instruction was given to the subjects to keep the cube motionless between the tongue and the palate because according to Ohmure et al. (12) mastication movements may modulate the GMA and gastric emptying. Two control sessions involved examination with a tasteless cube, and without administration of a cube.

Interestingly, perception of the taste stimulation during the postprandial state was found to be different from that observed in the fasted subjects. Sweet taste was perceived less pleasant, and bitter was felt less unpleasant postprandially. Perception of sour taste appeared to be stronger after a meal than during the interdigestive state. The most pronounced difference against the fasted state was found, however, in the case of salty taste, which postprandially was perceived more unpleasant and more intensely, as well as was ranked higher on the nausea score.

According to the results obtained, the postprandial GMA pattern, gastric emptying of a solid meal, as well as the meal-induced shifts in the HRV appeared to be "resistant" to the taste stimulation because it did not bring about any essential changes thereof. It should be pointed out that this finding is different from our former observations carried out during the interdigestive state. Then a clear impact of unpleasant tastes (bitter>salty>sour) upon the GMA with a less pronounced, but discernible, influence on the HRV was demonstrated (1).

A similar discordance was encountered previously while examining the effect of noise exposure upon the GMA. When accomplished during the interdigestive state, a 45-min exposure to a wide-band industrial noise of 91 dB(A) intensity evoked in healthy volunteers a pronounced disorganization of the gastric myoelectrical activity, characterized by a decrease in the relative time share of normogastria, an increase in the duration of tachygastria, and a deteriorated stability of the dominant frequency and dominant power of the gastric slow

waves (13). On the other hand, the postprandial GMA, and the gastric emptying of either a semiliquid or a solid test meal remained unchanged irrespective of what noise type (pink tonal, pink band, blue tonal, blue band) was emitted (5). One would therefore suspect that ingestion of a meal may provide a protective effect on the digestive tract against a deranging effect of stress, whether it would be in the form of exposure to noise or taste stimulation. Results obtained by Prof. Robert Stern's research group support the hypothesis outlined. Namely they demonstrated a protective effect of intake of a meal against disorganization of the GMA by such a powerful laboratory stressor, like illusory rotation provoking motion sickness (14, 15).

A question arises why ingestion of a caloric meal elicits a somehow 'protective' influence against derangements of the GMA and autonomic balance evoked otherwise by exposure to disgusting tastes during a fast. The most likely explanation which springs to mind is that ingestion of a solid caloric meal switches the stomach from a quiescent interdigestive condition into an active postprandial state wherein taste stimuli do not play such an incisive role as during a fast. After all, as was outlined earlier in the Discussion, two of the four tastes examined-salty and sour-were perceived even more intensely when administered postprandially than during the interdigestive state. As a matter of fact, in their comprehensive review on the role of gastric motility in the control of food intake, Janssen et al. (16) describe the switch from the interdigestive to the postprandial functioning as 'drastic' (16). During the interdigestive phase, the proximal stomach muscle tone is high, while the distal stomach is engaged in a recurrent contraction pattern known as the migrating myoelectrical (or motor) complex (MMC) (16). Upon transition to the postprandial state, the proximal stomach relaxes to accommodate the increasing amounts of food, whereas the distal stomach mixes and grinds the food by exerting powerful and regular peristaltic contractions. The stretch of both the proximal and antral part of the stomach trigger mechanosensitive receptors which convey information via vagal and splanchnic nerves to specialized structures in the central nervous system (16). As a result, sensation of satiety evolves, which in turn may make the gastric myoelectrical activity less responsive to taste stimulation than during the interdigestive state during which the sensation of hunger prevails.

Summing up, we conclude from our results that taste stimulation elicited after ingestion of a meal, in contrast to that performed during a fast, does not adversely modify the postprandial pattern of the gastric myoelectrical activity or sympathetic/parasympathetic balance, and that neither affects the gastric emptying of solids.

# Author Contribution -

MW, AK-J and KJ conceived the study and its protocol, analyzed the data and wrote the manuscript. MD, MK, and MB directed the experiment, analyzed the data and wrote the manuscript. JL, JP, DJ and JR were involved in pursuing the experiment, collecting the data and writing the manuscript.

# **Conflict of Interest**

None.

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