

COMPARISON BETWEEN PRE-PRANDIAL AND POST-PRANDIAL HEART RATE VARIABILITY (HRV).

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Abstract : After food ingestion, peptides are released in GIT, which cause local vasodilatation. Therefore, after meals, redistribution of blood occurs because of shifting of large amounts of blood into GIT. In normal individuals, this is well compensated and does not lead to post-prandial hypotension. The mechanism of post-prandial hypotension is well known. We hypothesized that there may be a decrease in parasympathetic activity (tone) after meals to compensate for the change in blood distribution. We carried out the study to find out the changes in the autonomic tone before and after meals (lunch) in normal individuals, using Heart Rate Variability (HRV). From the series of RR intervals marked, the time domain and frequency domain measures of HRV were obtained using Nevrokard software (version 6.4). Continuous ECG was recorded in 15 healthy adult subjects (mean age 29.06 ± 6.2 ; 13 males and 2 females). The ECG was recorded in pre-prandial and post-prandial state for a period of five minutes each as follows: (1) just before the subjects had lunch, (2) 15 minutes after lunch, (3) 1 hour after lunch, and (4) 2 hours after lunch. Time domain and frequency domain measures of HRV were compared between pre-prandial state and rest of post-prandial states. The autonomic tone parameters did not show a significant change between the pre-prandial state and the immediate post-prandial state. [Range, i.e., the difference between the maximum and minimum RR intervals (406 ± 161.14 vs. 416.66 ± 125), standard-deviation of normal to normal RR interval

Synopsis : Ambarish et al studied the changes in autonomic activity (tone) using heart rate variability (HRV) after feeding. Parasympathetic and sympathetic tone was studied in 15 healthy adult subjects before and after lunch. No significant change was seen in both when the autonomic tone parameters were compared. But the authors propose that HRV can be used to detect autonomic neuropathy in diabetics at a very early stage, by comparing pre-prandial and post prandial HRV.

(56.33 ± 22.72 vs. 67.63 ± 26.50), RMSSD (55.02 ± 35.85 vs. 63.87 ± 32.60), NN50 (42.13 ± 29.43 vs. 51.86 ± 29.83), PNN50 (12.67 ± 10.29 vs. 15.27 ± 9.71), HF (49.53 ± 15.10 vs. 47.07 ± 16.88), LF (41.41 ± 13.18 vs. 46.49 ± 15.99), LF/HF (0.98 ± 0.53 vs. 1.26 ± 0.90), total power (148.27 ± 37.78 vs. 137.61 ± 37.10)]. No significant change was seen in the above parameters between the pre-prandial state and the later phases of post-prandial state. Since there is no significant decrease in the time domain measures and the HF value between the pre-prandial and the post-prandial states, we conclude that the parasympathetic tone is not altered. The parameters denoting sympathetic tone, ie, LF and LF/HF, also do not show a significant change. This indicates that the cardiovascular autonomic tone is not affected by ingestion of meals in normal individuals. Thus we refute our hypothesis. In conclusion, the HRV parameters do not alter significantly after meals in normal individuals.

Key words : heart rate variability pre-prandial postprandial
time domain frequency domain

INTRODUCTION

The gastro-intestinal tract (GIT) is supplied by the enteric nervous system (ENS) comprising the myenteric plexus of Auerbach, the external and the internal sub mucous plexuses. This apart, the GIT comes under the influence of the autonomic nervous system (ANS) that has the sympathetic and the parasympathetic components (1). Ingestion of food is a visceral stimulus that leads to various physiological adjustments which include metabolic and cardiovascular changes such as increased blood flow to GIT, and a decreased skeletal muscle blood flow (2). Food intake causes peptides to be released in the GIT, which leads to vasodilatation locally. This leads to redistribution of blood, i.e. more blood being supplied to GIT (3). The enteric nervous system that controls the pacemaker and motor activity of GIT, communicates with the central nervous system, interacting with the heart through the autonomic nervous system.

The measured heart rate is modulated

by the two main components of autonomic nervous system. These are parasympathetic and sympathetic nervous system. An increase in sympathetic activity increases the heart rate and increased parasympathetic activity decreases the heart rate, thus balancing each other. Vagal activity is dominant in resting conditions.

In healthy individual, the interval between successive heartbeats is constantly varying. The variations in heart rate are cyclic and non-cyclic. The non-cyclic variations are frequent, sudden large beat to beat changes in R-R intervals superimposed on the cyclic changes, and occur throughout the day and night. These are abolished by atropine but not affected by beta-blockers, and absent in subject with parasympathetic neuropathy. The cyclical variations are associated with various physiological functions like respiration, baroreceptors reflex activity, thermoregulatory mechanisms, and changes in peripheral chemoreceptor activity and renin-angiotensin activity. These functions, in general are normally exhibits cyclic oscillations. Each of these

oscillations occurs at particular frequency, which gets reflected as predominant peak in frequency spectrum. The frequency components are divided in three major bands starting from the very low frequency (VLF) range between 0-0.03 Hz. This frequency range is associated with thermoregulatory mechanisms, changes in peripheral chemoreceptor activity and/or fluctuation in the activity of renin-angiotensin. There are cyclical variations occurring in association with changes in baroreceptors activity. These changes occur at low frequency of 0.10 Hz (range 0.03–0.15) and are significantly modified by sympathetic blockade. There is strong correlation between this low frequency variation in the heart rate and direct measures of sympathetic nerve activity. However, vagal blockade produces small reduction in this low frequency component in HRV spectrum, suggesting that there is also parasympathetic component to this cyclic activity. The ratio of high and low frequency components in HRV spectrum is commonly used to denote sympathovagal balance. The respiratory related high frequency changes occur at 0.25 Hz (range 0.18–0.4). These can be abolished by vagal blockade (4). These two factors suggest that this particular type high frequency component in HRV is parasympathetically mediated. Thus, the study of Heart Rate Variability (HRV), gives an insight into the heart rate control mechanisms. HRV gives a fairly accurate idea about the individual's sympathetic and parasympathetic tone (5).

HRV can be analyzed by several methods. Two approaches, namely, the time domain and the frequency domain methods are commonly employed. Both these approaches are complementary to each other. The time

domain parameters give overall fluctuations in the heart rate. From ECG strip, a series of R-R intervals are calculated and then various indices are computed by statistical analysis. Among these indices the standard deviation of normal-to-normal RR intervals (SDNN) is an index that quantify the total amount of variability present in the recording and information on sympathovagal balance. Other indices like RMSSD (the square root of the mean of the sum of the squares of the differences between adjacent RR intervals), NN50 count (the number of pairs of adjacent RR intervals differing by more than 50 ms during the selected time interval), and PNN50 [percentage of NN50 counts of all RR intervals, i.e. (NN50 count/Total count of RR intervals) × 100] quantify parasympathetic activity.

The frequency domain analysis involves plotting of R-R interval duration against number of R-R intervals (tachogram). The frequency domain method dissects out the entire range of fluctuations into different frequencies; it can separate the sympathetic (low frequency) and parasympathetic (high frequency) contribution to the heart rate regulation. This information is usually presented graphically by plotting the vertical axis against the frequency at which it occurs on the horizontal axis. By measuring area under curve at different frequency a numerical measure of the amount of high and low frequency cyclical variability present in the recordings is obtained.

HRV is a simple method to quantify cardiovascular autonomic neural input, in normal as well as to evaluate patients with acute Myocardial Infarction, Cardiac Failure, Obesity and Irritable Bowel

Syndrome (1). Studies on elderly subjects has shown that blood pressure falls post prandial (6), but adequate studies have not been done as yet on the effect of food intake on HRV in normal subjects.

We hypothesized that food intake may result in a transient decrease in parasympathetic activity. Probably by the decreased control of parasympathetic nervous system, the heart rate may be increased to compensate for the pressure profile in the non-gut areas. Since the changes demand mild change, the sympathetic activation may not be called for. Thus, the sympathovagal tone is reflected and quantified in the sino-atrial node of heart (1).

MATERIAL AND METHODS

Subjects :

The subjects were 15 healthy adults; 13 males and 2 females in the age group of 29.06 ± 6.2 years working in the Physiology department of All India Institute of Medical Sciences, New Delhi, India. All the subjects gave their informed consent prior to their inclusion in the study. History was recorded for each of the subject before starting the test to screen for any evidence of autonomic dysfunction and any other disorders, as well as for alcohol abuse. None of the subjects had abdominal symptoms like abdominal pain, abdominal discomfort, diarrhea, constipation, feeling of distension, flatulence, etc. They did not have any heart related complaints either. Just before and during the study, the subjects did not consume tea or coffee and did not smoke cigarettes.

They were not on any kind of medical therapy.

Procedure :

Each of the subjects were allowed to have breakfast in the morning at 8.30 AM. No dietary restrictions were imposed before the study. The procedure was explained to each subject before the test. The HRV was recorded using Nevrokard software version 6.4 (Medistar, Slovenia). The HRV was recorded with subject in supine posture. Three electrodes of clip type were applied – one to right forearm, at the wrist. The second electrode was applied to the right leg at the ankle and the third electrode was applied to the left leg at the ankle. A non-reactive electrode gel was applied to the skin where electrodes were applied for good conduction. The recordings were taken at controlled room temperature ($22 \pm 1^\circ\text{C}$) in the Autonomic Function Test Laboratory. For each subject all recordings were taken in one day. It was seen to it that the surroundings were calm and quiet. The subjects were restricted from active movements and they rested in sitting position in-between the recordings.

HRV was recorded continuously for a period of 5 minutes (300s) in each of the recordings. HRV was recorded once before the subject had meals (lunch). The 2nd HRV recording was taken 15 minutes after lunch. The 3rd HRV recording was taken 1 hour after lunch and the 4th HRV recording was taken 2 hours after lunch. The composition of food in lunch was similar in all subjects in quantity and quality. Food was provided in the lab and the subjects consumed food

in sitting position and adequate time was allowed to consume food. HRV was recorded in each subject between 12.30 and 18.00 hours to minimize the circadian effects. The timings confirmed to their normal lunch timings.

The autonomic tone parameters:

The time domain parameters taken into consideration were; range (the difference between the maximum and the minimum RR intervals), standard deviation of normal-to-normal RR intervals (SDNN), RMSSD (the square root of the mean of the sum of the squares of the differences between adjacent RR intervals), NN50 count (the number of pairs of adjacent RR intervals differing by more than 50 ms during the selected time interval), and PNN50 [percentage of NN50 counts of all RR intervals, i.e. (NN50 count/ Total count of RR intervals) \times 100]. The frequency domain parameters taken into consideration were; LF/HF ratio, LF, HF and total power. The normalized LF, HF and total power were also calculated (mentioned as normalized units/nu). For spectral analysis, 0 to 256 samples from the total number of samples in each subject were taken for analysis.

Statistical analysis:

The above-mentioned time domain and frequency domain parameters were compared between the pre-prandial state and the rest of the postprandial states. The Wilcoxon signed ranks test (non-parametric test for 2 related samples) was employed to analyze and compare the data between the meals.

RESULTS

None of the subjects experienced any adverse symptoms after consumption of meals. The time domain measures did not show a significant change between the pre-prandial state and the immediate postprandial state (i.e. 15 minutes after lunch): [Range: (406 \pm 161.14 msec vs. 416.66 \pm 125 msec), SDNN (56.33 \pm 22.72 vs. 67.63 \pm 26.50), RMSSD (55.02 \pm 35.85 vs. 63.87 \pm 32.60), NN50 (42.13 \pm 29.43 vs. 51.86 \pm 29.83), PNN50 (12.67 \pm 10.29 vs. 15.27 \pm 9.71)].

The frequency domain parameters also did not show a significant change between the pre-prandial and the immediate postprandial state: [HF (49.53 \pm 15.10 vs. 47.07 \pm 16.88), LF (41.41 \pm 13.18 vs. 46.49 \pm 15.99), LF/HF

TABLE I

	<i>Pre-lunch</i>	<i>Post lunch 1 15 min</i>	<i>Post lunch 2 1 hours</i>	<i>Post lunch 3 2 hours</i>
Range	406.00 \pm 161.15	416 \pm 125	406.73 \pm 226.79	426.53 \pm 278.88
Std. Deviation	55.34 \pm 22.73	67.63 \pm 26.50	54.36 \pm 26.15	53.55 \pm 22.91
RMSSD	55.02 \pm 35.85	63.87 \pm 32.60	46.39 \pm 28.71	45.54 \pm 26.76
NN50 Count	42.13 \pm 20.43	51.86 \pm 29.83	36.40 \pm 31.91	37.73 \pm 30.36
HF (nu)	49.53 \pm 15.10	47.07 \pm 16.88	46.91 \pm 12.06	42.99 \pm 17.91
LF (nu)	41.41 \pm 13.18	46.49 \pm 15.99	44.88 \pm 13.40	48.72 \pm 14.29
LF/HF	0.98 \pm 0.53	1.26 \pm 0.90	1.11 \pm 0.73	1.46 \pm 0.93
Total Power(nu)	148.27 \pm 37.78	137.61 \pm 37.10	160.29 \pm 60.75	205.80 \pm 129.45

(0.98 ± 0.53 vs. 1.26 ± 0.90). Total power (148.27 ± 37.78 vs. 137.61 ± 37.10).

No significant change was seen in the above parameters between pre-prandial state and the rest of the postprandial states also. Thus parasympathetic tone did not alter significantly between meals with time (Table-I).

DISCUSSION

There is increase in heart rate after a meal in young healthy and older persons. There is splanchnic blood pooling after a meal, which reduces systemic vascular resistance. This leads to postprandial reduction in the return of blood to the heart. But, meal ingestion in young persons is followed by a slight increase in systolic blood pressure because of an increase in heart rate and cardiac output. The activity of sympathetic nervous system increases after meals. There is increase in heart rate, plasma norepinephrine levels and sympathetic nerve activity of muscle recorded by microneurography. There is also increase in plasma renin activity, cardiac output, and forearm vascular resistance after meal ingestion in healthy young persons, which compensate for splanchnic blood pooling and result in a stable blood pressure after meal ingestion (7).

In healthy young persons, it has been shown that beta-adrenergic blockade with propranolol does not attenuate the increase in heart rate in response to food ingestion, which suggests that the change in heart rate is independent of the sympathetic nervous system and it could be due to vagal

withdrawal. Also other factors might be involved like insulin, somatostatin, vasoactive peptides and also intra-vascular fluid status. It is seen that in patients with autonomic failure, postprandial hypotension is associated with the absence of a cardio-acceleratory response. Thus alteration in blood volume by meal is adjusted by compensatory mechanisms in healthy persons but not in disease states. In our study since there is no raise in the time domain measures and the HF value between pre-prandial and the postprandial states, we conclude that the cardiac parasympathetic tone is not altered. The parameters denoting the sympathetic tone i.e. LF and LF/HF ratio, also did not show a significant change. There is no change in cardiac sympathetic activity in postprandial period. This indicates that the cardiovascular autonomic tone is not affected by ingestion of meals in normal young adults. Thus, we refute our hypothesis. In conclusion, HRV parameters do not alter significantly after food ingestion in normal young adults.

Postprandial regulation of central hemodynamics is highly dependent on the autonomic nervous system (8). The probable reason for the insignificant alteration in cardiovascular autonomic tone after food intake is that in young and healthy subjects, the redistribution of blood volume does not lead to any systemic changes in blood pressure. The sympathetic activity very quickly compensates for any effects of the parasympathetic activity after food intake (6, 9). Food ingestion causes postprandial hypotension in patients with autonomic failure (3). An increase in postprandial parasympathetic tone may

be seen in these patients as the autonomic regulation is compromised. Depressed HRV can be used as an early warning sign of diabetic autonomic neuropathy (5). In these cases; HRV can be used as a robust and non-invasive method to assess the status of the cardiovascular autonomic tone.

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