Research Paper

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Influence of pre & post –prandial states on measures of heart rate variability in healthy subjects

Medical Science

KEYWORDS : Heart rate variability / Autonomic nervous system / Pre-prandial state / Postprandial state.

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ABSTRACT

Objective: Heart rate variability refers to variations of heart beat intervals of sinus rhythm and is used for the noninvasive investigation of cardiac autonomic function & also used as prognostic factor in life threatening cardiac disease. Further study is done to compare the changes in heart rate variability in pre & post - prandial states in young healthy individuals .

Material & methods: Frequency domain measures of heart rate variability were obtained using medical analyzer module of Nivomon Software. ECG was recorded in twenty healthy male subjects (mean age 23.45 + 2.86) in pre-prandial & post-prandial states continuously for a period of five minutes (300 seconds) each time as follows :- 1) Pre lunch (just before lunch), 2) Post lunch 1 (15 minutes after lunch), 3) Post lunch 2 (1 hour after lunch), 4) Post lunch 3 (2 hours after lunch). Frequency domain measures of heart rate variability were compared between pre-prandial state & different post-prandial states.

Results: There was no significant decrease in high frequency values of heart rate variability (which shows parasympathetic activity) between pre-prandial & the post-prandial states.

Conclusions: The parasympathetic activity is not altered. The parameters denoting low frequency values of heart rate variability (which shows sympathetic activity) also do not show a significant change.

INTRODUCTION

Heart rate variability is an indicator of autonomic function and physiological coherence (Tulppo et al, 2004). Specifically, heart rate variability is a measurement of the "interaction between sympathetic (i.e. "fight or flight" energy mobilization) and parasympathetic (i.e. opposite of sympathetic activity or "relaxation" response) activity in autonomic function (Callahan et al, 2000).

The gastro-intestinal tract (GIT) is supplied by the enteric nervous system comprising the myenteric plexus of Auerbach, the external and the internal sub mucous plexuses. This , apart the GIT , comes under the influence of the automatic nervous system that has the sympathetic and the parasympathetic components (Adeyemi et al, 1999). 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 (Elsenbruch et al, 2001). 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. 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 (Polinsky et al, 1995).

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 are 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 as well as renin-angiotensin activity (Malliani et al, 2005).

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 (LF) 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 (HF) changes occur at frequency of 0.25 Hz (range 0.18–0.4). These can be abolished by vagal blockade (Jiri et al, 2002). These two factors suggest that this particular type high frequency component in HRV is parasympathetically mediated. Thus, the study of HRV gives an insight into the heart rate control mechanisms. HRV gives a fairly accurate idea about the individual's sympathetic and parasympathetic tone (Malik 1994).

MATERIAL & METHOD

The study was carried out in the Department of Physiology,

S.M.S. Medical College, Jaipur (India) . Twenty healthy male subjects with age ranging of 23.45±2.86 years were selected from among the hospital staff and their relatives. All the subjects gave their informed written consent prior to their inclusion in the study. None of the subjects had any abdominal symptoms like abdominal pain, abdominal discomfort, diarrhoea, 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.

PROCEDURE

(i) Patient's preparation was done after explaining exact recording procedure and then following instructions were given to the subjects :

(a) To avoid food preceding two hours of heart rate variability testing.

(b) No coffee, nicotine or alcohol intake 24 hours prior to the testing.

(c) To wear loose and comfortable clothing.

(ii) Recording

For short term analysis of heart rate variability, impedance peripheral pulse was recorded in the supine position for five minutes. The study was performed between 12.00 noon to 6.00 P.M. after a supine resting period of more than thirty minutes. Room ambient temperature was maintained between 24-25°C. Subject was instructed to breathe quietly during the entire recording period with closed eyes and was advised to avoid :

(a) Talking
(b) Moving hands, legs and body
(c) Coughing during test
(d) Sleeping

(iii) Acquisition

The impedance peripheral pulse wave signals were continuously amplified, digitized, and stored in the computer for offline analysis in frequency domain.

(iv) Processing

The detection of 'C' wave (major positive upward deflection in the impedance plethysmograph in normal subjects) was done by software developed indigenously by Bhabha Atomic Research Center (BARC) incorporated in the medical analyzer module of NIVOMON and was taken to obtain the cardiac interval (RR interval) as 'C' wave occurs within 150 milli-second of the R wave (Jindal et al, 2004). All recordings were visually examined and manually corrected if required. Abnormal beats and areas of artifacts were automatically and manually identified and excluded from the study.

Heart rate variability was evaluated in accordance with guidelines previously set forth (Task force , 1996). Power spectral analysis for the frequency domain methods, based on the FFT (Fast Fourier Transform) algorithm was used. The resultant power density spectrum was then analyzed for following bandwidths: very low frequency (0.001 to 0.04 Hz), low frequency (>0.04 to 0.15 Hz) and high frequency (>0.15 to 0.4 Hz). Low frequency and high frequency were further normalized [LF (n.u., normalized units) and HF (n.u., normalized units)] to better quantity sympathovagal balance. Heart rate variability was recorded continuously for a period of five minutes (300 seconds) in each of the recordings as follows :

(a) Pre lunch (just before lunch)
(b) Post lunch (15 minute after lunch)
(c) Post lunch (1 hour after lunch)
(d) Post lunch (2 hours after lunch)

Heart rate variability was recorded once just before the subject had meals (pre lunch). The 2nd heart rate variability recording was done fifteen minutes after lunch. The 3rd heart rate variability recording was done one hour after lunch and the 4th heart rate variability recording was done two 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.

Quantification of Heart Rate Variability

Power spectral densities (PSD) are plotted in ms^2/Hz against preset frequencies. Power of the spectral bands are calculated in ms^2 (absolute power) and in normalized units (n.u.).The normalized units were calculated as (Pichon et al, 2005) :

H.F. (n.u.) = HF (absolute units) / LF (absolute units) + HF (absolute units) x 100

L.F. (n.u.) = LF (absolute units) / LF (absolute units) + HF (absolute units) x 100

The L.F / H.F. ratio was calculated to assess sympathetic / parasympathetic modulation.

STATISTICAL ANALYSIS

The above-mentioned 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

The frequency domain parameter did not show a significant change between the pre -prandial and the immediate postprandial state [LF (44.92±12.61 n.u. vs. 49.52±17.72 n.u.), HF (53.56±12.35 n.u. vs. 50.32±13.90 n.u.) and LF/HF ratio (0.94±0.50 vs. 1.08±0.50)]. No significant change was seen in the above parameters in pre-prandial state and rest of the postprandial states also (Table no.1).

Table No.1

Absolute Units	Prelunch	Post Lunch 1 (15 minutes)	Post lunch 2 (1 hour)	Post lunch 3 (2 hours)
Low frequency (Normalized units, n.u.)	44.92±12.61	49.52±14.72	46.99±13.57	51.39±14.75
High frequency (Normalized units, n.u.)	53.56±12.35	50.32±13.90	48.80±16.48	45.36±14.17
LF/HF ratio	0.94±0.50	1.08±0.50	1.16±0.76	1.32±0.78

DISCUSSION

There is increase in heart rate after a meal in young healthy 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 (Ambarish et al, 2005). 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 (Rene et al, 1995).

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 (Kelbaek et al,1987).

In our findings, heart rate variability parameters do not alter significantly after food ingestion in normal young adults. There

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have been few studies that have examined changes in autonomic modulation in response to a meal. Ambarish et al (2005) compared heart rate variability parameters in fifteen healthy adults (thirteen males and two females) in preprandial and postprandial states and his result also showed no changes in low frequency and high frequency values .

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