POINCARE PLOT OF HEART RATE VARIABILITY: AN APPROACH TOWARDS EXPLAINING THE CARDIOVASCULAR AUTONOMIC FUNCTION IN OBESITY

KRISHNAN MURALIKRISHNAN1*, KABALI BALASUBRAMANIAN1, SAJJADH M. JAWAHAR ALI1 AND BADANIDIYUR VISHWANATHA RAO2

Department of Physiology, Stanley Medical College, Chennai, India and Institute of Physiology and Experimental Medicine, Madras Medical College, Chennai, India

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Abstract: Obesity has been shown to affect cardiovascular function. Heart rate variability (HRV) has been an accepted method of measuring cardiovascular autonomic function. The aim of the study is to evaluate the impact of obesity on HRV using Poincaré plot (POP) analysis. A finding of sympathovagal imbalance in pre-obese adults in respiratory sinus arrhythmia (RSA) could provide important diagnostic information about early subclinical autonomic dysfunction in obesity. Thirty one obese (BMI 26.84±2.47) adult males (25.42±7.86 years) were compared with 31 normal subjects (25.38±4.61 years). In all participants, anthropometric and blood pressure (BP) measurements were performed. After rest at supine position for 5 minutes, they were asked to do control deep breathing for 1 minute. HRV was measured in terms of POP analysis. Differences in Resting heart rate (RHR) (P≤0.025), Pulse pressure (PP) (P≤0.048), SD1 (P≤0.042) and SD2 (P≤0.039) of the POP between the two groups were significant. Correlation between Body mass index (BMI) and (PP) (p=0.19); SD1 (p=0.47) and SD2 (p=0.39) of the POP were significant in obese groups. Obesity is related to sympathovagal imbalance characterized by depressed parasympathetic tone and increased sympathetic activity. Nonlinear methods like POP permit simple assessment of autonomic function, despite measuring different aspects of HRV.

Key words: poincaré plot obesity heart rate variability

INTRODUCTION

Obesity is a well-known risk factor for coronary heart disease in adults (1, 2). It is reported to increase the risk of a high blood pressure (BP) and a subsequent overt...
hypertension in middle age (3). Various studies have shown that salt intake, blood volume, and cardiac output are higher in obese than normal subjects (4). Recent research has applied methods for quantifying sympathetic nervous system (SNS) activity to the study of the SNS pathophysiology in obesity-related hypertension (5, 6). In fact, rapid weight gain is associated with increased cardiac sympathetic tone in humans (7). However, there has been a lack of autonomic imbalance studies that involve obese individuals of younger age group. HRV is a noninvasive measure of heart rate fluctuations. R-to-R interval variations on electrocardiograms represent beat-to-beat control mechanisms of the heart. Sympathetic and parasympathetic activities directed to the sinus node characterized by each cardiac cycle can be modulated by central and peripheral stimulators. These stimulations generate rhythmic fluctuations in efferent neural discharge that manifest as oscillations in the heart beat period. HRV denotes the variability's of both instantaneous HR and consecutive RR intervals. Cardiac autonomic function can be quantified by short term and long term HRV analysis (8).

The Poincaré plot in HRV is a scatter plot of the current R-R interval plotted against the preceding R-R interval. The synonyms of Poincaré plot are Scatter plot or scattergram, Return map or phase delay map and Lorenz plot. Poincaré plot analysis is a simple quantitative visual technique compared to conventional fast Fourier transform indices (FFT) (9, 10). The plot provides summary information as well as detailed beat-to-beat information on the behavior of the heart (11). Points above the line of identity indicate R-R intervals that are longer than the preceding R-R interval, and points below the line of identity indicate a shorter R-R interval than the previous. Accordingly, the dispersion of point’s perpendicular to the line of identity (the “width”) reflects the level of short-term variability. The points along the line of identity (the “length”) reflect the long-term variability (12).

Tulppo et al (13) fitted an ellipse to the shape of the Poincaré plot and defined two standard descriptors of the plot, SD1 and SD2, for quantification of the Poincare plot geometry. These standard descriptors represent the minor axis and the major axis of the ellipse respectively as shown in Figure 1. The description of SD1 and SD2 in terms of linear statistics, given by Brennan et al (14) shows that the standard descriptors guide the visual inspection of the distribution. It reveals a useful pattern of the RR interval data by representing both short and long term variations of the signal (13, 14). SD1 shows the standard deviation of the short-term variability of the data. The SD2 shows standard deviation of the continuous long-term R-R intervals (SD2 or major axis) is measured along the horizontal axis. The point where both axes intersect corresponds to the total mean of the R-R intervals. Various authors have shown that varying lags of Poincaré plot. But it does give better understanding about the autonomic control of the heart rate (9, 15).

MATERIALS AND METHODS

All the subjects gave written informed consent prior to participation in the study. The subjects included MBBS students and
also individuals screened under master health check-up scheme (age, 19 to 44 years) of Stanley Medical College & Hospital, Chennai. They were screened clinically for cardiovascular diseases, neural or endocrine disorders and diabetes by a standard protocol. The subjects were non-smokers, non-alcoholics and were not on any other medication for the past 3 months. For all participants, a clinical examination was performed followed by anthropometric and BP measurements. They were divided into two groups based on new guidelines issued by Government of India for obesity: Normal (BMI: 18.5-23 kg/m²) and obese (BMI ≥25 kg/m²) (16).

The test was performed in the morning two hours after a light breakfast. All subjects were asked to void urine before testing and made to sit in the Neurophysiology Lab of Department of Physiology, Stanley Medical College for 20 minutes. The subjects were instructed to remove their footwear and the height was measured using a right-angle ruler placed on the head against a tape measure secured to the wall. Current weight (in kg) was measured using an electronic scale. The body mass index was calculated as weight divided by height squared (in kg/m²). Baseline BP (measured with a sphygmomanometer) and HR were measured after 10 min of rest in supine position. Electrocardiogram (ECG Lead II) and respiratory movements were acquired using RMS Polyrite D Hardware, India and instantaneous HR and RR intervals were plotted continuously using Finland v1.1 software for HRV analysis, in supine position, at rest and in controlled deep breathing (inspiration 5 sec, expiration 5 sec) for 1 minute.

### Poincaré plot analysis

The recommendations of Task Force on HRV were followed (8). Briefly a 10 min ECG (Lead II) was acquired at the rate of 200 Hz at rest followed by the same while controlled deep breathing. An RR series was extracted using maximum amplitude and sharpness of the peaks for R wave detection. These are RMS proprietary algorithms and validated with Fluke Biomedical, USA, after exclusion of artifacts and ectopics. This algorithm is fed to the Finland v1.1 software for HRV analysis. The SD1 and SD2 were calculated for the controlled deep breathing segment of one minute and the report sheet was generated.

### Statistical analysis

The Data are expressed as mean ± Standard Deviation. The variation in parameters between the two groups was tested using student’s independent t-test. A correlation between normally distributed indices was determined using the Karl Pearson correlation coefficient. P value was less than 0.05 was considered significant. SPSS software was used for the statistical analysis.

### RESULTS

The baseline characteristics of the normal and the obese subjects are recorded in the Table I. The subjects in both the groups were age matched (p=0.98). The BMI varied significantly among the normal and the obese subjects (p=0.001). The resting heart rate (p=0.025) and pulse pressure (PP), (p=0.048) varied significantly between the two groups, meaning thereby obesity reflects
TABLE I: General parameters, Basal heart rate (BHR), Blood pressure during supine rest and Heart rate variation.

<table>
<thead>
<tr>
<th></th>
<th>Normal (n=31)</th>
<th>Obese (n=31)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>25.38±4.61</td>
<td>25.42±7.86</td>
<td>0.988</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>21.71±2.99</td>
<td>26.84±2.47</td>
<td>0.001***</td>
</tr>
<tr>
<td>Resting HR (bpm)</td>
<td>70.48±10.06</td>
<td>76.4±6.4</td>
<td>0.025*</td>
</tr>
<tr>
<td>SBP (mm Hg)</td>
<td>104.97±8.71</td>
<td>104.57±7.2</td>
<td>0.861</td>
</tr>
<tr>
<td>DBP (mm Hg)</td>
<td>68.21±7.76</td>
<td>67.19±7.51</td>
<td>0.641</td>
</tr>
<tr>
<td>PP (mm Hg)</td>
<td>35.12±5.68</td>
<td>39.62±9.97</td>
<td>0.048*</td>
</tr>
<tr>
<td>MAP (mm Hg)</td>
<td>80.46±7.64</td>
<td>80.4±8.88</td>
<td>0.986</td>
</tr>
<tr>
<td>SD1 (ms)</td>
<td>62.71±22.37</td>
<td>50.12±20.54</td>
<td>0.042*</td>
</tr>
<tr>
<td>SD2 (ms)</td>
<td>154.83±54.64</td>
<td>125.24±50.96</td>
<td>0.039*</td>
</tr>
</tbody>
</table>

All data are expressed as Mean±Standard Deviation. P≤0.05, P≤0.01, P<0.001 compared with both groups. BP: Blood Pressure; BMI: Body Mass Index; HR: Heart Rate; bpm: beats per minute; SBP & DBP: systolic and diastolic BP respectively; PP: pulse pressure; SD1: short-term variability; SD2: long-term variability.

TABLE II: Correlation between BMI, resting HR, Pulse pressure (PP), HRV during rest and controlled deep breathing.

<table>
<thead>
<tr>
<th></th>
<th>Normal (n=31)</th>
<th>Obese (n=31)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Correlation between BMI and Resting HR</td>
<td>0.02</td>
<td>0.961</td>
<td>0.12</td>
</tr>
<tr>
<td>Correlation between BMI and PP</td>
<td>0.50</td>
<td>0.037*</td>
<td>0.30</td>
</tr>
<tr>
<td>Correlation between BMI and SD1</td>
<td>0.37</td>
<td>0.063</td>
<td>0.21</td>
</tr>
<tr>
<td>Correlation between BMI and SD2</td>
<td>-0.47</td>
<td>0.078</td>
<td>-0.36</td>
</tr>
<tr>
<td>Correlation between Resting HR and SD1</td>
<td>-0.13</td>
<td>0.512</td>
<td>-0.10</td>
</tr>
<tr>
<td>Correlation between Resting HR and SD2</td>
<td>-0.03</td>
<td>0.971</td>
<td>-0.05</td>
</tr>
</tbody>
</table>

All data (n=31 and n=31) are expressed as Pearson's correlation coefficient, as appropriate. *P≤0.05, **P≤0.01, ***P<0.001.

on these vital indices. This was in agreement with the earlier study done by Kwabeniewska M et al (17). However, this was not the case with the Systolic Blood Pressure (SBP, p=0.86), Diastolic Blood Pressure (DBP, p=0.64) and Mean Arterial Pressure (MAP, p=0.98).

DISCUSSION

In controlled deep breathing, an oscillation of the heart rate pattern around the basal mean value is well pronounced. The parasympathetic nervous system responds rapidly to rhythmic respiratory discharge. Thus, the HRV analysis especially at low respiratory frequencies provides information about cardiac vagal efferent activity of the autonomic nervous system (18, 19). HRV (HRV db) deep breathing is used as a major index of Heart Rate variation, which provides a simple measure of cardiac vagal effects (20). In our study, there is a significant change seen in the vagal efferent, which is observed in the POP.

From POP analysis, SD1 parameter is used as a marker of vagal influence, whereas SD2 parameter represents the more delayed R-R interval changes correlated to sympathetic activity with SD1 and SD2 being the two axes of the best-fit ellipse that contains the Poincaré points (21). SD1 (short term variability) is an indirect measure of parasympathetic activity. When the SD1 value decreases, correspondingly the parasympathetic activity also decreases. In the study, there was a significant positive correlation (p=0.047) observed between BMI and SD1 in obese individuals. This implies that the parasympathetic activity is decreased in obese individuals. SD2 (long term variability) is related more strongly to sympathetic activity than parasympathetic activity. When the SD2 value decreases, sympathetic activity is increased. In this
study, there was significant negative (p=0.039) correlation between BMI and SD2 in obese individuals. There was also significant difference in SD1 (p=0.042) and SD2 (p=0.039) values between both the normal and obese groups. In obese individuals, as both SD1 and SD2 decrease, there is decreased parasympathetic activity and increased sympathetic activity. Heart rate variability is caused by parasympathetic modulation of the sinoatrial (SA) node (8). Decrease in parasympathetic activity in obese individuals as in our study, causes decreased heart rate variations. Hence the scatter plot is more concentrated towards the line of identity, as compared to the normal subjects, where the points are more scattered from the line of identity. (Fig. I) SD values, which are expressions of standard deviation, are therefore decreased in obese individuals, explaining decreased heart rate variability. Decrease in heart rate variation in obese individuals increases the risk stratification for cardiovascular pathologies by decreasing the adaptability of the heart in stressed situations. This explains the association of obesity with cardiovascular accidents.

There is a positive correlation observed between BMI and pulse pressure in both groups significantly. This is probably due to the young age where the pulse pressure is not very much widened. This is in concurrence with earlier study done by Madanmohan et al (22). As Age and BMI increases there will be a wide pulse pressure. This increases the cardiovascular disease risk (20).

A positive correlation was observed between increase in BMI and resting heart rate (p=0.22) in the obese group. The probable explanation could be that an increase in BMI is expected to increase the heart rate due to increase in cardiac output required to compensate the extra tissue load.

Fig. 1 : Poincaré plots for A. A normal individual. B. An Obese individual.
The study had a few limitations. The foremost being that it was performed exclusively on male genders. Hence, the results cannot be generalized for the whole population. The gender-related differences in fat distribution, namely the presence of abundant subcutaneous fat in women and of visceral fat in men, are expected to have an impact on the cardiovascular risk profile (23) and may influence the mode of HRV modulation (1).

In a Poincaré plot due to complex dynamic behaviors, the SD1/SD2 statistics yield mixed results. This is because the technique relies on the existence of a single cluster or a defined pattern, which could not be achieved accurately in this study (18, 19). Poincaré plot in deep breathing has been shown to predict mortality following myocardial infarction (24) and also subsequent ventricular tachycardia (25). In other studies, it was found to be effective in the assessment of both arrhythmia and CHF against normal sinus rhythm (26). In future, it may be used as an efficient tool for cardiovascular risk detection (26).

**Conclusion**

The Poincaré plot is a promising technique of HRV analysis. It is so simple, that it can be used by anyone with a basic knowledge of computer. Reduced HRV seen in obese individuals clearly indicates that cardiac vagal effects (i.e. vagal modulation of RR intervals) are diminished and sympathetic activity seems to be increased. The current study is believed to serve a prelude to further studies involving easy evaluation of sympathovagal balance in order to answer the cardiovascular manifestations of various risk factors – obesity in this case.

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