Resting Rate Pressure Product In Type 2 Diabetics With And Without Cardiac Autonomic Neuropathy

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Abstract: Background: The mechanisms of silent death in diabetic cardiac autonomic neuropathy is unclear. Objective: An insight into resting rate pressure product, indirectly reflecting myocardial oxygen demand, in such patients in attempt to explain sudden adverse cardiovascular events. Method: Resting rate pressure product was recorded in forty age matched type 2 diabetics, twenty with / twenty without cardiac autonomic neuropathy and twenty controls (n = 60) between the age group of 40 – 60 years. Results: The results using one way ANOVA demonstrated (‘SPSS’ software) that resting rate pressure product was significantly increased in cardiac autonomic neuropathy patients compared to non neuropathy patients and controls (p<0.01) (95%CI). Resting rate pressure product was highest in neuropathy patients. Conclusion: We conclude that diabetic cardiac autonomic neuropathy significantly increases resting rate pressure product and indirectly reflects increased resting myocardial oxygen demand in such patients.

Key Words – Diabetic cardiac autonomic neuropathy, Rate pressure product, Myocardial oxygen demand

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Introduction: Cardiac autonomic neuropathy is associated with a high risk of unexplained sudden death, possibly related to silent myocardial infarction, cardiorespiratory arrests, cardiac dysrhythmias and hypoxia1.

We tried to observe resting rate pressure product, an indirect indicator of myocardial oxygen demand, in diabetic cardiac autonomic neuropathy patients which could possibly help in understanding silent adverse cardiovascular events occurring in this subset of patients. Studies in English peer reviewed literature have not observed it in this subset of patients.

Method: This study was approved by the Institutional Ethical Committee of our Hospital in accordance with the Helsinki Declaration of 1975. Study was done on total 60 subjects of either sex between 40 – 60 years taken from a tertiary health care centre in North India - 40 patients with type 2 diabetes mellitus with a duration of diabetes of 8-12 years and 20 healthy controls.

Fasting / Random capillary glucose was measured using glucometer. Details of history and examination were recorded on a proforma. Informed consent was taken from all subjects before the recording the blood pressure and heart rate.

Resting blood pressure and heart rate were recorded in all the subjects supine, at complete physical and mental rest. Blood Pressure was recorded using sphygmomanometer and heart rate calculated from lead II on cardiofax ECG machine (Medicaid Systems).

\[
\text{Heart Rate} = \frac{60 \times 25}{\text{R} - \text{R interval}} \times 1500
\]

Autonomic neuropathy was clinically assessed as postural fall in systolic BP > 30 mm Hg, cardiac involvement assessed as impaired response to hand grip.2 The subject was asked to hold hand grip dynamometer in dominant hand at 30% of maximum grip strength for three minutes. A rise in diastolic blood pressure 10mm Hg or less is abnormal, an indicator of sympathetic insufficiency.3 Rate Pressure Product was calculated as a product of heart rate and systolic blood pressure.4

\[\text{RPP} = \text{Systolic Pressure in mm Hg} \times \text{Heart Rate in beats/min} \times 10^2\]

The results were analysed using one way ANOVA (‘SPSS’ software).

Subjects: They were divided into 3 groups of 20 subjects each.

Group A: Patients having type 2 diabetes with clinically evident diabetic cardiac autonomic neuropathy.

Group B: Patients having type 2 diabetes without diabetic cardiac autonomic neuropathy.

Exclusion Criteria for patients: Ischemic Heart Disease, Congestive heart failure and Cardiac arrhythmias. Patients on α blockers, β blockers,
calcium channel blockers, diuretics, antiarrhythmics, antipsychotics were excluded from study.

**Group C**: 20 healthy age-matched controls (free from any systemic illness) were taken.

**Results**: Resting rate pressure product was significantly increased in diabetic cardiac autonomic neuropathy patients compared to non-neuropathy patients and controls ($p<0.01$) (95%CI). Resting rate pressure product was highest in neuropathy patients.

**Table 1: Resting Rate Pressure Product in patients with/without diabetic cardiac autonomic neuropathy & controls**

<table>
<thead>
<tr>
<th>Rate Pressure Product (mmHg bpm)</th>
<th>Group A 95%CI</th>
<th>Group B 95%CI</th>
<th>Group C 95%CI</th>
<th>F-ratio</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean $\pm$ SD</td>
<td>129.9$\pm$13.1, 123.8 – 136.1</td>
<td>117.7$\pm$18.6, 108.9 – 126.4</td>
<td>99.1$\pm$15.1, 92.1 – 106.2</td>
<td>19.4</td>
<td>0*</td>
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</table>

*p<0.01

**Discussion**: The present study was carried out with the aim to determine the potential of diabetic cardiac autonomic neuropathy to influence resting rate pressure product. Resting heart rate and systolic blood pressure was recorded in type 2 diabetic patients with/without cardiac autonomic neuropathy patients and controls.

Diabetic cardiac autonomic neuropathy patients had a significantly higher Rate Pressure Product at rest compared to non-neuropathy counterparts as well as controls ($p<0.01$) (Table 1).

Resting tachycardia could potentially be contributing to significantly heightened resting rate pressure product in such patients. Resting heart rates of 90-100 beats per minute and occasional heart rate increments upto 130 beats per minute occur. The highest resting heart rates have been found in patients with parasympathetic damage, occurring earlier in the course of cardiovascular autonomic neuropathy than sympathetic nerve dysfunction, in those with evidence for combined vagal and sympathetic involvement, the rate returns toward normal but remains elevated$^5$.

Based on the guidelines of the European Society of Hypertension and the JNC-7, BP was considered controlled in diabetics at $<130$ mm Hg systolic and $<80$ mm Hg diastolic pressure$^6$. Thus the major contribution to heightened rate pressure product was given by heart rate.

Foo K, et al, confirmed independent association of diabetes with RPP which was estimated to be 9% higher than in patients without diabetes$^7$. Our study confirms the same in diabetic cardiac autonomic neuropathy patients also. Another observation in the study was that resting rate pressure product was highest in cardiac autonomic neuropathy patients.

**Conclusion**: We conclude that resting rate pressure product is significantly elevated in diabetics with cardiac autonomic neuropathy reflecting heightened myocardial oxygen demand at rest which may be the potential agent aggravating regional myocardial ischemia leading to unexplained sudden deaths in such patients. Limitations of the study include lesser number of patients taken. Studies on patients with selective sympathetic/parasympathetic impairment will give a better insight into the state of resting rate pressure product.

**References**


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