Comparative Study Para Sympathetic Nerve Function Test In Normal and Moderately Hypertensive Patients

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Abstract: Hypertension involves almost all the systems of body, predominantly affecting ANS which controls involuntary aspects of body functions. When CVS is affected, it may lead to myocardial infarction, and cardiac failure which may ultimately lead to death. The onset and severity of complication in a system is said to be dependent on the duration of hypertension and the degree of its control. Autonomic dysfunction is drawing more and more attention of the medical fraternity for its supposed role in sudden deaths observed in hypertensive and diabetics. Further objective evaluation of autonomic disorder is becoming imminent both for prevention of such catastrophes and also better control of diseases. Our aim is to perform the autonomic function tests namely Valsalva ratio, E/I ratio during deep breathing. The study was programmed to assess autonomic functions based on cardiovascular parameters like blood pressure and heart rate measurement. Among the autonomic function tests discussed in the review of literature, following four tests were selected to evaluate autonomic function after considering the availability of equipments, feasibility and cost effectiveness. There was significant reduction in valsalva ratio in hypertensive group in comparison to the normotensive group. Unlike borderline hypertension where there is an increase in sympathetic drive and decrease in parasympathetic tone, in our study sympathetic drive is definitely less. The changes we have found could be attributed to both sympathetic and parasympathetic dysfunction or autonomic imbalance or increase in vascular resistance.

Keywords: Autonomic imbalance, Hypertension, parasympathetic dysfunction, Valsalva manoeuvre

INTRODUCTION:
Hypertension is one of the leading disorders contributing to significant morbidity and mortality in the world today. Today’s stressful life and modern life styles including the food habits (Junk food) have increased the incidence, acquiring a status of modern day epidemic along with diabetes mellitus [1]. The onset and severity of complication in a system is said to be dependent on the duration of hypertension and the degree of its control. Based on the dissection of ancient Egyptian mummies, high blood pressure (hypertension) has been a health problem since the early Egyptian empires [2]. The blood pressure cuff was invented in 1896, but it wasn’t until the 1950s that we became aware of the importance of high blood pressure as a herald of the complications commonly attributed to “old age”. Recent research has shown a critical relationship between high blood pressure and strokes, heart attacks, congestive heart failure, and vascular disease, which together comprise the leading cause of death in the world for men and women [3]. Hypertension is defined as sustained elevation in the blood pressure over the upper limit of normal as 135 mm Hg (Systolic pressure) and over 85 mm Hg (diastolic). If either the systolic or the diastolic blood pressure is persistently greater than this, hypertension is said to be present [4].

It is an important public health challenge throughout the world because of high prevalence and concomitant increase in risk of cardiovascular, cerebrovascular and renal complications. Various studies have shown that it is the most important modifiable risk factor for coronary heart disease, stroke and congestive heart failure. Blood pressure changes throughout our lives, and on a minute-to-minute basis. It tends to get higher as we age, and is usually lowest when we sleep. As we go through a normal day, it fluctuates and becomes elevated in response to stress,
pain, hormonal levels, meals, and exercise. These are normal responses to our environment. In hypertension, the blood pressure becomes higher than necessary to maintain normal body functions [5]. This elevation begins to damage the circulatory system, increasing the “wear and tear” wherever there are blood vessels. The heart, which must work harder to push the blood through a higher pressure environment, begins to increase in size. The walls of the heart thicken and the heart muscle doesn’t relax normally [6]. The walls of the blood vessels also thicken and become stiffer. They can develop damage to the inner lining of the blood vessel, with resulting cholesterol deposits at the places of damage. This process, known as atherosclerosis, can lead to strokes, heart attacks and blood vessel enlargements known as aneurysms. About 43 million Americans have hypertension. In India the prevalence is about 17% in urban population and 12% in rural population [7]. The asymptomatic nature of the disease especially in the initial 15-20 years even as it progressively damages the cardiovascular system, makes its control mandatory. A major cause of death in systemic hypertension is heart disease. De-Simone et al.; has shown that earliest changes in the heart’s function are either super normal systolic function or more commonly impaired diastolic function. The year 1992 was a major landmark in the history of hypertension as Rosenthal et al.; showed that impaired diastolic functions by itself may interfere with maximal exercise capacity and over a period of time is associated with heart failure and coronary disease? In the same year Brogen et al.; in 1992 proved that about 40% of episodes of congestive heart failure in systemic hypertensive patients is associated with normal left ventricular systolic function but with diastolic dysfunction induced by left ventricular hypertrophy, ischemia and after load [8].

Systemic Hypertension:

According to JNC-7, systemic hypertension is defined as systolic BP of 140 mmHg & above and a diastolic BP of 90 mmHg & above. It has got a very high prevalence rate and associated life threatening complications; so careful documentation of blood pressure is very important in diagnosis. The typical marker variability of blood pressure complicates the issue of defining the level at which it indicates hypertension. In the Multiple Risk Factor Intervention Trial (MRFIT) study, 3,50,000 men/women were followed up for a period of 12 years and it was found that the long term risk of cardiovascular mortality associated with various levels of blood pressure rise progressively over the entire range. So the best operational definition of systemic hypertension is the level of blood pressure at which the benefit of action exceeds the risks. “White Coat Hypertension” a term used to describe persistently high physician’s office or hospital readings, but persistently normal out of office reading is found in 20 to 30 percent of patients. This is suspected when there is discrepancy between level of hypertension and target organ damage [9, 10].

The JNC-VII’s guidelines for documentation of hypertension as follows:
1. Multiple readings should be obtained using appropriate techniques ideally ABPM reading over a period of 24 hrs.
2. Systolic elevation poses a risk equal to or greater than that posed by diastolic elevation. Isolated systolic hypertension (Systolic BP > 140 mm of Hg with Diastolic BB < 90 mm of HG), especially seen in elderly presents a risk for both stroke and myocardial infarction.
3. Pseudo hypertension is high cuff values of diastolic blood pressure because of sclerotic brachial arteries in elderly which cannot be occluded by the blood pressure cuff.
4. Elderly patients are more susceptible to orthostatic hypotension.

MATERIALS AND METHODS:
A comparative study of cardiovascular autonomic function tests in hypertensive and normotensive individuals was conducted in the Department of physiology, Department of Community Medicine, RMMCH-Urban Health Centre, Chidambaram on 25 hypertensive patients in the age group of 30-40 years and age matched control group of 25 normotensive individuals. The study was programmed to assess autonomic functions based on cardiovascular parameters like blood pressure and heart rate measurement. Before starting the study, approval was taken from the ethical committee. The Subjects were appropriately informed regarding the procedures involved in the study.

Inclusion criteria: a) Age group-30 to 40 years b)Sex-Both male and female c) Blood pressure-d)Newly diagnosed moderate hypertension e)Inclusion criteria for control group are same but they are normotensies.


Valsalva manoeuvre: subject is made to lie comfortably on a bed with ECG leads attached. Basal ECG is recorded and hr calculated. ECG is recorded continuously. Subject is asked to blow into the manometer with closed glottis maintaining a pressure of 40 mmg hg for 15 seconds duration. ECG is recorded for 1 minute before the manoeuvre, during the manoeuvre for 15 seconds and 45 seconds following the manoeuvre.

Valsalva ratio: the valsalva ratio is a measure of
parasympathetic and sympathetic function. For the response to occur in valsalva manoeuvre, parasympathetic acts as afferent and efferent and sympathetic as part of the efferent pathway. Therefore, the valsalva ratio assesses more of parasympathetic (cardio vagal) functions.

**Deep breathing test:** Subject is asked to lie down quietly with ECG leads connected. Basal ECG is recorded and heart rate calculated. Subject is asked to breathe deeply and slowly at a rate of 6 breaths per minute with equal time for inspiration and expiration. The maximum hr (shortest r-r interval) and minimum hr (longest r-r interval) is noted in a respiratory cycle.

**Heart Rate Response to Breathing:** The variation of heart rate with respiration is known as sinus arrhythmia. Inspiration increases and expiration decreases heart rate. This is primarily mediated by parasympathetic innervations of heart. Pulmonary stretch receptors, cardiac mechanoreceptors and bar receptors contribute to sinus arrhythmia. The difference between the maximum and minimum heart rate during a deep breathing is called Heart rate variation (HRV). HRV is more than 15 beats per minute in normal individual. It assesses the parasympathetic activity. HRV decreases with age.

**RESULTS**

**Table 1:** Changes in R-R interval during Valsalva Manoeuvre

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Group</th>
<th>Mean-R interval during Phase II (mm)</th>
<th>Mean R- R Interval during phase IV (mm)</th>
<th>Valsalva ratio</th>
<th>‘t’ value</th>
<th>‘p’ value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Valsalva</td>
<td>Normotensive (n=15)</td>
<td>14.267</td>
<td>20.000</td>
<td>1.405</td>
<td>-5.668</td>
<td>0.000*</td>
</tr>
<tr>
<td></td>
<td>Hypertensive (n=25)</td>
<td>15.760</td>
<td>18.480</td>
<td>1.182</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

In the normotensive group the mean R-R interval during phase II was 14.267 mm and during phase IV was 20.000 mm. Valsalva ratios was 1.405. In the hypertensive group the mean R-R interval during phase II was 15.760 mm and during phase IV was 18.480 mm. Valsalva ratios was 1.182. * Statistically significant.

**Table 2: I/E RATIO during deep breathing**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Group</th>
<th>Mean Heart rate during inspiration (per min)</th>
<th>Mean Heart rate during expiration (per min)</th>
<th>I/E Ratio</th>
<th>‘t’ value</th>
<th>‘p’ value</th>
</tr>
</thead>
<tbody>
<tr>
<td>E/E ratio</td>
<td>Normotensive (n=15)</td>
<td>85.267</td>
<td>79.267</td>
<td>1.077</td>
<td>1.795</td>
<td>0.083</td>
</tr>
<tr>
<td></td>
<td>Hypertensive (n=25)</td>
<td>86.960</td>
<td>78.480</td>
<td>1.112</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

In the normotensive group the mean heart rate during deep inspiration was 85.267 per minute and during deep expiration was 79.267 per min with I/E ratio of 1.007. In the hypertensive group the mean heart rate during deep inspiration was 86.960 per minute and during deep expiration was 78.480 per min with I/E ratio of 1.112. Statistically significant.

**Table 3: Heart rate variation during deep breathing**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Group</th>
<th>Mean Heart rate during inspiration (per min)</th>
<th>Mean Heart rate during expiration (per min)</th>
<th>HRV (per min)</th>
<th>‘t’ value</th>
<th>‘p’ value</th>
</tr>
</thead>
<tbody>
<tr>
<td>HRV</td>
<td>Normotensive (n=15)</td>
<td>85.267</td>
<td>79.267</td>
<td>6.000</td>
<td>1.711</td>
<td>0.097*</td>
</tr>
<tr>
<td></td>
<td>Hypertensive (n=25)</td>
<td>86.960</td>
<td>78.480</td>
<td>8.480</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

In the normotensive group the mean heart rate during deep inspiration was 85.267 per minute and during deep expiration were 79.267 per min with HRV of 6.000 per minute. In hypertensive group the mean heart rate during deep inspiration was 86.960 per minutes and during deep expiration were 78.480 per min with a HRV of 8.480. Statistically significant.
DISCUSSION:

Cardiac output is regulated by End-diastolic volume and myocardial contractility and peripheral resistance in influenced by viscosity of the blood and fourth power of the radius of the blood vessels. So, by altering the lumen of the blood pressure can be increased or decreased [11]. The autonomic nervous system as well as rennin – Angiotension, natriuretic peptide and kallikrein – kinin system plays a role in the physiological regulation of short – term changes in the blood pressure and has been implicated in the pathogenesis of essential hypertension [12]. In some young hypertensive patients there is an early increase in cardiac output in association with increased pulse rate and circulating catecholamine. This could result in changes in baroreceptor sensitivity which would then operate at a higher blood pressure. In chronic hypertension, the cardiac output in normal and maintains the increased blood pressure is the peripheral resistance [13]. The resistance vessels (the small arteries and arterioles) show structural changes in hypertension. These are an increase in wall thickness with a reduction in vessel lumen diameter. There is also some evidence for rarefaction (decreased density) of these vessels. These mechanisms would result in an increased overall peripheral vascular resistance. Autonomic function tests have been used extensively in patients with cardiac abnormalities [14]. In fact Valsalva ratio, Expiratory – Inspiratory (E/I) ratio in deep breathing, Hand grip test and cold pressure test provide a fast, non-invasive and inexpensive method of evaluation of cardiac functions. Baroreceptor sensitivity as determined by pulse interval and blood pressure responses to the valsalva manoeuvre was significantly reduced with hypertension in elderly patients. Abnormal valsalva ratios were recorded and compared to normal subjects. Valsalva ratio was inversely related to the left ventricular end-diastolic pressure and mean pulmonary wedge pressure in patients with aortic valve disease and mitral insufficiency. Valsalva ratio in the control subjects of all age group and chronic obstructive lung disease without cardiac failure in young age group was above 1.5 and slightly decreased with increasing age. In young non-diabetic patients with cardio vascular disease, E/I ratio and valsalva ratio were significantly lower than those without cardiovascular disease [15]. In young diabetic patients with CVD, only E/I ratio was significantly reduced compared to those without CVD. Beat to beat variation expressed by E/I ratio may serve as a single complementary test of parasympathetic cardiac integrity and hand – grip test as a sympathetic screening procedure [16]. The mean valsalva ratio of systolic heart failure patients was significantly lower than that for diastolic heart failure which in turn was lower than healthy subjects [17]. In another study by Monica mourya et al.; autonomic function tests were done to show the effects of slow and fast breathing exercises in patients with essential hypertension. Slow breathing had a stronger effect than fast breathing. Blood pressure decreased over a 3- month period with both exercises. However, E/I ratio, BP response to the hand grip and cold pressure test showed significant change with patients in slow breathing exercise [18].

CONCLUSION:

We performed autonomic function tests: Valsalva ratio, E/I ratio during deep breathing, test in both the groups. The findings are as follows. There was significant reduction in valsalva ratio in hypertensive group in comparison to the normotensive group. The change in E/I ratio and HR variation was statistically not significant. Unlike borderline hypertension where there is an increase in sympathetic drive and decrease in parasympathetic tone, in our study sympathetic drive is definitely less. The changes we have found could be attributed to both sympathetic and parasympathetic dysfunction or autonomic imbalance or increase in vascular resistance [19, 20].

REFERENCES:


