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Association of Obesity with Impaired Cardiac Autonomic Modulation in Children

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ABSTRACT

Background: Positive family history of hypertension is one of the risk factors for being hypertensive in the later part of life. The incidence of childhood obesity is on the rising trend and obese children are more vulnerable to various health disorders such as heart disease, stroke, diabetes and hypertension. As obesity and family history of hypertension are known to influence the cardiovascular health, this study was designed to assess the alteration in the cardiovascular autonomic function in the obese children with and without family history of hypertension by comparing their HRV parameters with the HRV of non-obese counterparts.

Materials and Methods: This cross-sectional observational study was conducted in children of age group 9-19 years (160 subjects) in a private school in Ariyur, after obtaining institutional ethical committee clearance and permission from the head master of the school and parents. Children were divided into 4 groups with 40 subjects in each group. (Obese children with no family history of hypertension, obese children with family history of hypertension, non-obese children with no family history of hypertension and non-obese children with family history of hypertension). Height, Weight, Waist circumference, Waist Hip ratio, skin fold thickness, blood Pressure in supine and standing position, Resting Heart Rate, values were measured. ECG was recorded in Lead II configuration in supine posture for 5 minutes and then on standing for 5 min, using PHYSIOPAC PP4 MEDICAID system CHANDIGARH.

Results: Our findings showed that the heart rate, DBP and LF nu and LF/HF ratio were found to be significantly increased and total power and HF nu in obese children with family history of hypertension. There is significant decrease in total power in obese children without family history of hypertension. There is a significant increase in SBP, DBP, LF/HF ratio and LF nu and there was a significant decrease in total power and HF nu in non-obese children with family history of hypertension. This shows that there is altered sympathovagal balance in obese children with and without family history of hypertension and also in non-obese with family history of hypertension.

Conclusion: From our study, we observed that there is alteration in sympathovagal balance among the obese and non-obese children with positive family history of hypertension and also in obese without family history of hypertension. As a result, they may suffer from increased cardiovascular morbidity. Hence Periodic assessment of HRV in these vulnerable population, can help detect early changes in cardiovascular health whereby timely interventions could be implemented and vital advices on weight reduction and regular exercise could be provided to avert the risk of cardiovascular disease in them.

INTRODUCTION

Obesity is a complex multifactorial disease in which excess body fat has accumulated to the extent that it may have an adverse effect on health. Obesity is a global epidemic and a major contributor to some of the leading causes of death including heart disease, stroke, diabetes, hypertension and some types of cancer. (1)

Obesity can result from excess calorie intake, sedentary lifestyle, environmental factors, genetic factors, endocrine factors, depression, drugs such as corticosteroids, antidepressants, antiepileptics, emotional eating, lack of sleep or by a combination of above mentioned factors. The morbidity and mortality rates are higher in obese individual when compared to the non-obese.(2) When co-existence of diabetes and hypertension occurs, it alarms the need to intervene promptly to avert their impact on global health. The normotensive children of hypertensive parents are reported to have altered cardiovascular functions signifying their transition from health to hypertensive state. Therefore prevention of non-communicable diseases namely diabetes, hypertension, obesity, stroke and cardiovascular diseases in adults should begin from childhood. If early intervention for childhood hypertension is not instituted, it may result in target organ damages, such as thickening of the carotid vessel wall, left ventricular hypertrophy, retinal changes, indefinite changes in cognition, and even premature development of atherosclerosis.(3,4) There is normally a balance between sympathetic and parasympathetic nervous system in our body called as sympathovagal balance. This balance is lost in various diseases includinghypertension, diabetes and obesity. Disorders of

the ANS cause autonomic insufficiency or failure and can affect any system of the body.(5) Heart rate variability measures the changes in the time interval between individual heart beats. It is done by using a specialized heart rate monitor that measures the time between each heartbeat interval, also known as R-R intervals, on the ECG trace. A low HRV is even associated with an increased risk of death and cardiovascular disease. (6,7)
Some studies have shown that obesity and family history of hypertension are intertwined variables that can influence negatively the cardiovascular health.(8) Therefore, this study has been designed to assess the effect of obesity on heart rate variability on the background of family history of hypertension as the positive family history itself is a predisposing factor for inheritance of essential hypertension which can alter sympathovagal balance.

REVIEW OF LITERATURE

In a study conducted by Amar Taksand et al. among Indian population age, height, weight, and BMI were positively correlated with both SBP and DBP and this study established the strong relation between hypertension and overweight and obesity.(9) Similar effort by Kiran Kumar Chintala et al. showed that the baseline heart rate, blood pressure, total body fat, subcutaneous fat and visceral fat were higher in the overweight group and among frequency domain parameter of HRV, LFnu and LF: HF were more and HFnu was less in the overweight group and also inferred that Overweight individuals may suffer from an increased mortality risk due to CVD related to either continuously lowered parasympathetic or altered sympathetic activation.(10)
An Indian study by Rajalakshmi et al. reported that the obese children had a significantly raised blood pressure and a significantly reduced total HRV when compared to normal weight boys while the obese girls had a significantly raised total HRV when compared to normal weight girls making the inference that hormonal changes that occur during puberty may have a differential impact on the cardiac autonomic status of male and female children.(11)
The observation of altered sympathovagal activity was also observed by the assessment of classic autonomic function test by Preeti Rathi et al. who reported that the resting heart rate, systolic and diastolic blood pressures were not significantly raised in subjects of study group. The sympathetic function tests as assessed by rise in diastolic blood pressure following cold pressor test and hand grip test were highly significant in study group as compared with control one. However, the vagally mediated tests, for heart rate responses, denoting parasympathetic functions were similar in both groups. It seems that there is increased sympathetic activity in children of hypertensive parents without parasympathetic modulation in early life.(12)
G. K. Pal et al. reported the Sympathovagal imbalance (SVI) in prehypertensive offspring was observed due to increased sympathetic and decreased vagal activity.(13)
From the scientific evidences discussed above it could be inferred that obesity and parental history of HTN are predisposing factors for cardiovascular derangement and when confluence of both these conditions prevail in an individual as in case of obese children with positive parental history of hypertension the cardiovascular consequences could be expected to be modulated towards early onset of HTN in these vulnerable population.

AIM

To assess the heart rate variability in the healthy obese and non-obese school children with and without family history of hypertension

OBJECTIVES

- To assess the heart rate variability in the healthy obese and non-obese school children with family history of hypertension.
- To assess the heart rate variability in the healthy obese and non-obese school children without family history of hypertension.
- To compare the degree of sympathovagal imbalance between the groups.

MATERIALS AND METHODS

A cross sectional, observational study was done in children of age group 9-19 years (160 subjects) in a private school in Ariyur. Children were divided into 4 groups with 40 subjects in each group.

- Group 1: healthy obese children with no family history of hypertension (n=40).
- Group 2: healthy obese children with family history of hypertension (n=40).
- Group 3: healthy non-obese children with no family history of hypertension (n=40).
- Group 4: healthy non-obese children with family history of hypertension (n=40)

Inclusion criteria:

- Children of age group 9-19 years (both the gender) with and without the family history of hypertension
- Children with systolic BP = 90-119mmHg and Diastolic BP =50-79mmHg were included in the study.
- In the Obese group, children with BMI >30 were included and in the non-obese group, children with normal BMI (18.5-24.9) based on BMI (WHO guidelines) were included.

Exclusion criteria:

- Any acute or recent illness during the past three weeks.
- Any signs and symptoms suggestive of renal and other endocrinal diseases.
- History of congenital heart disease and asthma
- Athletic activity or yoga.
- Any medication that affects ANS.

Methods:

- The study was conducted after obtaining the Institutional Ethical Committee Clearance, the experimental procedures were performed after receiving the informed written consent from the study population (parents of the students and the head master of the institution)
- Parents meeting was conducted after getting permission from the head master of the school and the purpose of study and procedure was explained to them. The subject's parents were enquired about the family history of hypertension. BP was recorded for them and was advised for treatment if they were found to be hypertensive.
- For female subjects, HRV was recorded during early follicular phase of menstrual cycle during which the female hormones have low influence on HRV.
- His /Her Blood Pressure, Resting Heart Rate, Height, Weight, Waist circumference, Waist Hip ratio, skin fold thickness values were measured.

Measurement of obesity indices:

- Height was measured using a stadiometer in centimeters
- Weight was measured using weighing machine (Omron HN-286) in kilograms
- Body Mass Index: BMI was calculated by the formula weight divided by square of height in meters.
- Waist circumference was measured as the midpoint between lowest rib and iliac crest in the standing position at the end of a gentle expiration.
- Hip circumference was measured around the point with maximum circumference over the buttocks.

- Waist hip ratio was calculated by formula (Waist circumference/ hip circumference)
- Skin fold thickness was recorded using skin fold caliper (Magideal RC)
- Triceps skin fold thickness- measuring a vertical fold on the posterior midline of the upper arm, halfway between the acromion and olecranon process, with the arm held freely to the side of the body.

Blood pressure, both systolic (SBP) and Diastolic(DBP) pressure were recorded using Sphygmomanometer (Diamond) in right arm in sitting position and standing position after 5 minutes of rest.

After 15 minutes of rest, 5 minutes of ECG recording was recorded in Lead II configuration in supine posture and then on standing posture for 5 min, in the morning time between 9 AM-12 noon in a quiet and temperature-controlled room, in the school premises, using PHYSIOPAC PP4 MEDICAID system CHANDIGARH. The subjects were advised to avoid caffeine at least 2 hours before the test and recording was done at least 2 hours after the meal. Instructions were given to all the subjects before each test and their doubts were clarified.

Measurement of HRV:

The analysis can be performed on short electrocardiogram (ECG) segments lasting from 0.5 to 5 minutes or on 24-hour ECG recordings.(14) Heart rate variability can be assessed in two ways:

1. Time domain analysis of RR interval
2. Frequency domain analysis of RR interval

Frequency domain parameters:

Frequency domain (power spectral density) analysis describes the periodic oscillations of the heart rate signal decomposed at different frequencies and amplitudes and provides information of their relative intensity (termed variance or power) in the heart's sinus rhythm. Power spectral analysis can be performed in two ways:

1) by nonparametric method, the fast Fourier transformation (FFT), which is characterized by discrete peaks for the several frequency components.
2) by parametric method, the autoregressive model estimation resulting in a continuous smooth spectrum of activity.

While the FFT is a simple and rapid method, the parametric method is more complex and needs verification of the suitability of the chosen model (85) Variables of Frequency domain of HRV: (a) Low frequency (LF) power – obtained by integrating the power spectral density between 0.04–0.15 Hz indicates sympathetic and parasympathetic activity (b) High frequency (HF) power – obtained by integrating the power spectral density between 0.15–0.4 Hz- indicates parasympathetic activity(c) LF normalized units (LF nu) obtained by dividing LF power by the sum of LF and HF power (Total power-VLF= LF+ HF)- indicates sympathetic activity (d) HF normalized units (HF nu) obtained by dividing HF power by the sum of LF and HF power (Total power- VLF = LF+ HF) - indicates parasympathetic activity (e) LF+HF - sum of LF power and HF power-indicates total heart rate variability (f) LF/HF obtained by dividing the LF power by the HF power-indicates sympatho-vagal balance.(15)

STATISTICAL ANALYSIS

The data analysis was done by using SPSS software 21st version. The variables which follow parametric distribution were expressed as Mean ± SD and independent t test was used to find out the statistical difference. The variables which follow non-parametric distribution and were expressed as median and Interquartile range and Mann Whitney U test was used to find out the statistical difference. P value of <0.05 was considered to be statistically significant.

RESULTS

Table 1: Comparison of anthropometric parameters between obese children with family history of hypertension and without family history of hypertension

Sl.no	Parameters	obese with family History of Hypertension (n=40)		obese without Family History of Hypertension (n=40)		P Value
		mean	SD	Mean	SD	
1	Weight (kg)	81.85	14.393	81.35	12.614	0.869
2	Height(cm)	158.73	12.277	158.65	10.531	0.977
3	BMI(Kg/m^2)	32.25	2.23	32.13	2.37	0.808
4	Waist Circumference (cm)	94.88	10.03	91.63	11.02	0.172
5	Hip Circumference(cm)	106.88	10.52	104.00	11.00	0.236
6	WHR	0.90	0.05	0.88	0.04	0.378
7	SFT- Triceps(mm)	24.05	3.97	22.28	5.33	0.058

BMI- Body Mass Index, WHR- Waist Hip Ratio, SFT- Skin Fold Thickness

The above table shows comparison of anthropometric parameters between obese children with family history of hypertension and without family history of hypertension. Independent t test was used to compare the groups. The values are calculated as Mean ± SD. P <0.05 was considered to be significant.

Table 2: Comparison of anthropometric parameters between non-obese children with family history of hypertension and without family history of hypertension

Sl.no	Parameters	non-obese with family History of Hypertension (n=40)		Non-obese without Family History of Hypertension (n=40)		P Value
		Mean	SD	Mean	SD	

1	Weight(kg)	45.25	10.14	57.20	6.23	0.000*
2	Height(cm)	148.43	12.92	164.08	7.53	0.000*
3	BMI(Kg/m^2)	20.25	2.22	21.18	1.13	0.020*
4	Waist Circumference (cm)	65.13	7.40	73.20	5.22	0.000*
5	Hip Circumference(cm)	80.85	8.70	91.58	4.61	0.000*
6	WHR	0.81	0.03	0.80	0.04	0.446
7	SFT- Triceps(mm)	15.03	3.76	16.10	5.29	0.298

BMI- Body Mass Index, WHR- Waist Hip Ratio, SFT- Skin Fold Thickness

The above table shows comparison of anthropometric parameters between non-obese children with family history of hypertension and without family history of hypertension. Independent t test was used to compare the groups. The values are calculated as Mean±SD. P <0.05 was considered to be significant

Table 3: Comparison of cardiovascular Parameters between obese children with family history of hypertension and without family history of hypertension

Sl.no	Parameters	Obese with Family History of HTN (n=40)		Obese without family History of HTN (n=40)		P Value
		Mean	SD	Mean	SD	
1	HR(supine)(bpm)	89	10.792	84.35	9.496	0.044*
2	HR(standing)(bpm)	90.77	24.74	85.03	17.67	0.2361
3	SBP (supine)(mmHg)	117.08	4.305	116.20	6.749	0.491
4	DBP (supine)(mmHg)	78.40	4.396	76.18	5.458	0.048*
5	SBP(standing)(mmHg)	118.45	9.190	117.08	2.820	0.3702
6	DBP(standing)(mmHg)	79.12	4.940	76.45	4.560	0.014*

HR- Heart Rate, SBP- Systolic Blood Pressure, DBP- Diastolic Blood Pressure.

The above table shows comparison of cardiovascular Parameters between obese children with family history of hypertension and without family history of hypertension. The values are calculated as Mean ± SD. Independent t test was used to compare the groups. P <0.05 was considered to be significant.

Table 4: Comparison of cardiovascular parameters between non- obese children with family history of hypertension and without family history of hypertension

Sl.no	Parameters	non-Obese with family History of HTN (n=40)		Non-Obese without family History of HTN (n=40)		P Value
		Mean	SD	Mean	SD	
1	HR(supine)(bpm)	82.90	7.23	86.33	8.70	0.059
2	HR (standing) (bpm)	88.37	9.53	84.9	7.07	0.0682
3	SBP(supine)(mmHg)	110.45	9.41	106.45	9.41	0.041*
4	DBP(supine)(mmHg)	74.23	7.14	69.53	7.43	0.005*
5	SBP (standing)(mmHg)	111.18	7.77	107.12	11.31	0.065
6	DBP(standing)(mmHg)	74.22	3.53	72.45	10.6	0.31

HR- Heart Rate, SBP- Systolic Blood Pressure, DBP- Diastolic Blood Pressure.

The above table shows comparison of cardiovascular parameters between non- obese children with family history of hypertension and without family history of hypertension. The values are calculated as Mean ± SD. Independent t test was used to compare the groups. P <0.05 was considered to be significant.

Table 5: Comparison of Frequency Domain HRV Parameters in Supine position between obese children with family history of hypertension and without family history of hypertension

S No		Obese with Family History of HTN (n=40) Obese without family History of HTN (n=40)			Obese with Family History of HTN (n=40) Obese without family History of HTN (n=40)			P value
	Parameters	Median	95% CI - Lower	95% CI - Upper	Median	95% CI - Lower	95% CI - Upper	

1	Total Power	1890.50	1821.190	2374.61	2862.5	2408.9	3536.03	0.05
2	LF/HF ratio	1.00	0.980	1.57	1	0.73	1.22	0.156
3	LF nu	44.50	39.650	49.90	39	33.78	43.47	0.165
4	HF nu	55.50	49.750	59.95	60	56.01	65.74	0.189
	LF/HF ratio: A ratio of Low Frequency to High Frequency; LF nu-Low Frequency in normalized units ; HF nu- High-Frequency power in normalized units							

The above table shows comparison of Frequency Domain HRV Parameters in Supine position between obese children with family history of hypertension and without family history of hypertension. The values are calculated as Median and IQR. Mann Whitney U test was used to compare the groups. P value<0.05 was considered to be significant.

Table 6: Comparison of Frequency Domain HRV Parameters in Supine position between non- obese children with family history of hypertension and without family history of hypertension

Sl.no		Non-Obese with Family History of HTN (n=40)			Non-Obese without family History of HTN (n=40)			P value
	Parameters	Median	95% CI - Lower	95% CI - Upper	Median	95% CI - Lower	95% CI - Upper	
1	Mean RR (milliseconds)	2674.00	2668.940	3995.61	3797	3904.1	5837.01	0.028*
2	SDNN (milliseconds)	1.000	0.82	1.28	1	0.64	1.06	0.191
3	RMSSD (milliseconds)	44.500	40.09	49.96	44	40.45	49.95	0.919
4	PNN50 (%)	55.000	49.66	59.39	56	49.71	59.09	0.885

SDNN- Standard Deviation of NN interval, RMSSD- Root Mean Square of Successive Differences, PNN50- PNN50- percent of difference between adjacent NN intervals that are greater than 50 ms

The above table shows comparison of Frequency Domain HRV Parameters in Supine position between non- obese children with family history of hypertension and without family history of hypertension. The values are calculated as Median and IQR. Mann Whitney U test was used to compare the groups. P value<0.05 was considered to be significant.

DISCUSSION

In our study, the obesity indices like the mean weight, height, BMI, waist circumference (WC), hip circumference and waist hip ratio were observed to be higher in the obese children with and without the family history of when compared to the non-obese counterparts. Mário Augusto et al. observed that the presence of abdominal fat, as assessed by the WC measurements, was found to be higher in obese children when compared to the non-obese children (1) and Mazharul Haque et al. observation of the linear relation that exists between age, weight, body mass index, waist circumference and waist-to- hip ratio with blood pressure, makes us to infer that the forewarning shadows of obesity's impact on health could be reflected by BP changes. Hence WHR is considered as a stronger obesity marker and in predicting the risk of cardiovascular diseases than BMI. (16)

Cardiovascular parameters:

In our study, heart rate (supine) (P=0.044), diastolic blood pressure in supine position (P=0.048) and standing position (P=0.014) were found to be significantly increased in obese children with family history of hypertension when compared to obese children without family history of hypertension. But, there was no significant difference in systolic blood pressure between the two groups. There is significant increase in SBP (supine) (P=0.041) and DBP (supine) (P=0.005) in non-obese children with family history of hypertension when compared to non-obese children without family history of hypertension. But, there was no significant difference in heart rate between the two groups.

Similar observations were also reported by Rajalakshmi, Veliath et al. who reported that the obese children had significantly increased systolic and diastolic blood pressure and mean arterial pressure when compared to children with normal BMI.(17) This may indicate that there exists an increased sympathetic vasomotor tone in the obese children. Renata Claudino Rossi et al. attributed the higher resting diastolic pressure to hyperactive sympathetic nervous system (SNS) and observations of tachycardia in obese people have been reported by several studies, which could be attributed to the altered autonomic modulation of the intrinsic heart rate. (18)

Lopes, in 2007 reported the existence of elevated sympathetic tone in the vascular beds of kidneys, skeletal muscles and peripheral vessels of the obese subjects and attributed the possible cause for this increased vascular reactivity to hyperinsulinemia, hyperleptinemia and increased fatty acid arising from obesity. (19)

As Elevated BP values in childhood can continue into adult life as hypertension and such higher pressure could predispose to premature atherosclerotic changes in the obese who are already in the high-risk range (values above 115/75 mm Hg), it is worth pondering at this moment the need to intervene and avert the transition of the apparently normal obese children to subjects with cardiovascular disease states. Hence it could be inferred that the alteration in the basal BP values reflect the silent transition of health to disease spectrum.

In contrary to our findings, Krishnan Muralikrishnan et al. observed that the basal systolic and diastolic pressure were not significantly higher between subjects with and without parental history of hypertension. Though the change was not appreciated with the values of BP, the HRV variables of these subjects were found to be altered, thus casting the shadow of cardiovascular derangement in their apparently normal normotensive frames. (20)

Basal HRV Parameters:

The observations of decrease in total power, HF nu and increase in LF/HF ratio and LF nu in obese children with family history of hypertension which were not

statistically significant could imply an alteration in the sympathovagal balance in its early stages.

The frequency domain parameters of Total power and HF nu are reduced and LF/HF ratio and LF nu were also found to be increased in non-obese children with family history of hypertension among which only total power (P=0.028) was found to be statistically significant. The reduction in Total power which represents the vagal activity of the heart, is found to be reduced in non-obese children with positive family history of hypertension thus once again inferring a derangement of sympathovagal balance by virtue of positive family history of HTN. Thus, it could be inferred from these observations that the children with family history of hypertension have altered sympathovagal balance.

As obesity is a state of reduced glucose threshold, insulin resistance and hyperinsulinemia and evidence exist for reduction in HF nu, an indicator of respiratory sinus arrhythmia with acute insulin administration, it could be inferred that the insulin resistant state or the hyperinsulinemia that occurs in obesity could be the cause for disruption of sympathovagal balance, thus, the low cardiac vagal activity. (17)

In contrary to these observations were the observations of where they reported a reduction in both sympathetic and parasympathetic activity in obese children and the reduction of global HRV was associated with the duration of obesity. (21) Therefore, it could be observed from the above findings that the alterations in basal HRV obtained in the supine rest relates to the cardiovascular impairment in the apparently normal obese children.

Hypertensive family history and HRV changes:

While an observation of decrease in both SDNN and RMSSD along with LF and HF values, among the normotensive offspring of hypertensive parents was also reported by Surekharani Chinagudi et al. RMSSD reflects vagal modulation of heart rate, and therefore RMSSD is considered as an important short term indicator of parasympathetic drive. These observations indicate that there is an altered sympathetic and parasympathetic activity in the study group when compared to controls.(22) Julius et al. had described the probable reason for such rise to be the hyperactive sympathetic nervous system (SNS) which is responsible for the higher basal values as increased SNS activity which leads to hypertension by alternating baro and chemo reflexes at both central and peripheral levels.(23)

In a study of classic autonomic function tests among the normotensive offspring of hypertensive by Preeti Rathi et al, it was observed that there was a significant increase in diastolic blood pressure ($p<0.001$) following both CPT and HGT (cold pressor test and hand grip test) in study group when compared to control group. (12) Unfortunately, in our study, we were not able to analyse the data according to which parent had hypertension owing to the inadequate number of participants with a positive history of maternal hypertension.(24)

Obesity and family history of HTN on cardiovascular health

Though sympathetic hyperactivity develops in children of hypertensive parents in spite of they being normotensives initially, there is a possibility of development of hypertension in future. On the basis of the observations of the present study and other inferences, the children of hypertensive parents may be advocated to change or bring modification in their lifestyles so as to prevent or delay the onset of hypertension at later stages of life as they obese population is predisposed to develop them early. Regular practices of slow breathing exercises have been reported to improve autonomic functions by improving sinus arrhythmia which strengthens the vagal tone and these Slow breathing exercises have been reported to improve vagal tone and decrease sympathetic tone, especially in pre hypertensive.(13)

From the above presented scientific evidences, it could be inferred that obese children and children with family history of hypertension may suffer from an increased mortality risk due to cardiovascular disorders related to either continuously lowered parasympathetic or altered sympathetic activation, if unchecked. Early detection and management by weight reduction and regular exercise can reduce the risk of cardiovascular diseases. HRV analysis can detect changes even before clinical signs appear. Thus, periodic assessment of HRV in vulnerable population viz obese and offspring of hypertensive parents help detect early changes in cardiovascular health and timely intervention help avert the progress of cardiovascular disease conditions in them.

CONCLUSION

Obesity and family history of hypertension are intertwined variables that can influence negatively the cardiovascular health. Therefore, this study has been designed to assess the effect of obesity on heart rate variability on the background of family history of hypertension as the positive family history itself is a predisposing factor for inheritance of essential hypertension which can alter sympathovagal balance.

In our study, heart rate (supine) and DBP (supine position and standing position) were found to be significantly increased and there is a significant decrease in Mean RR interval (supine position) and a significant increase in LF/HF ratio (standing position) in obese children with family history of hypertension when compared to obese children without family history of hypertension, which infers that there is an alteration in the sympathovagal balance in obese children with family history of hypertension in both supine and upright postures. There is significant increase in SBP (supine) and DBP (supine) in non-obese children with family history of hypertension.

There was a significant decrease in Total power and HF nu and LF/HF ratio and LF nu were increased in non-obese children with family history of hypertension when compared to non-obese children without family history of hypertension which infers that there is reduction in parasympathetic activity and increase in sympathetic activity in the non-obese children with family history of hypertension. This shows that there is an alteration in the sympathovagal balance in non-obese children with family history of hypertension in supine and upright postures. The alteration in the sympathovagal balance in upright posture signifies an enhanced response of cardiovascular adaptation in these study population which might add to the other precipitating factors for the development of hypertension as a response to persistent enhanced sympathetic activation beyond normal.

When frequency domain HRV Parameters were compared between obese children with family history of hypertension and non-obese with family history of hypertension, there was a significant decrease in total power (supine) and there was a significant decrease in total power (standing)and HF nu (standing) and there was a significant increase in LF nu (standing) in obese children with family history of hypertension. When frequency domain HRV Parameters in supine position and standing position were compared between obese children without family history of hypertension and non-obese without family history of hypertension, there was a significant decrease in total power in obese children without family history of hypertension.

These findings strongly indicate the need for the early care of these children to allow them to avoid the onset of future complications. These children are more prone for the development of cardiovascular diseases in later life. Early detection and management by weight

reduction and regular exercise/yoga may reduce these risks. Thus, regular assessment of HRV measures can serve an effective tool for early detection and subsequent management of cardiovascular diseases in these vulnerable population.

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