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RESEARCH ARTICLE

A COMPARATIVE STUDY OF THE EFFECT OF BODY MASS INDEX ON SYMPATHOVAGAL ACTIVITIES IN HYPERTENSIVE INDIVIDUALS

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Abstract

The rising rates of obesity and hypertension can be traced to a combination of factors including decreased physical activity, increased psychological stress, and increased work-related stress. Sympathetic overactivity is the primary pathophysiologic process in the development of obesity and hypertension, regardless of origin. Many clinical diseases can be traced back to a sympathovagal imbalance, or disruption of autonomic functioning due to sympathetic excess activity and vagal withdrawal. Autonomic dysfunction has been linked to obesity, hypertension, and diabetes mellitus separately. The measurement of heart rate variability (HRV) has become a popular, non-invasive method for studying cardiac autonomic dysfunction quantitatively. The current research sought to answer the question of whether obesity contributes to the worsening of autonomic dysfunction, especially in hypertensive individuals. Adult males and females in their forties and fifties who were patients at the OPD in UPUMS, Saifai, Etawah, India, were enlisted for the study.HRV was measured using the one-minute method while the subjects were in a relaxed state and deep breathing was encouraged. A total of 105 participants were divided into Groups I (obese hypertensive), II (non-obesity hypertension), and III (non-obese normotensive, control). The mean heart rate variability (HRV) of obese hypertension patients was considerably lower than that of non-obese hypertensive patients ($p<0.005$ at α 0.05 HRV, data provided as Mean± SD; inferential statistics by One Way ANOVA and Tukey's Post Hoc test). Our findings suggest that obesity and hypertension may have an additive effect in inducing autonomic dysfunction.

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Introduction:-

Obesity and hypertension (HTN) have become more common due to modernization, urbanization, and industry, which have lowered physical exercise and increased job and psychosocial stress[1]. Most of the body's visceral activities are regulated by the autonomic nervous system (ANS), which plays a crucial role in homeostasis maintenance via its two major branches, the sympathetic nervous system, and the parasympathetic nervous system [2]. The primary pathophysiologic mechanism in the development of obesity and HTN is increased sympathetic activity. Many clinical illnesses, including obesity, hypertension, and diabetes mellitus, have been linked to a sympathovagal imbalance or dysregulation of autonomic systems due to sympathetic excess activity and vagal withdrawal [3-5].

The importance of the autonomic nervous system (ANS) to cardiovascular health, it is not unexpected that there has been considerable interest in the monitoring of human sympathetic and vagus nerve traffic [6].

Variations in both the R-R interval and the current heart rate are referred to as heart rate variability (HRV) [7]. Heart rate variability (HRV) is a non-invasive electrocardiographic marker reflecting the activity of the sympathetic and vagal components of the autonomic nervous system (ANS) on the sinus node of the heart, which accounts for the majority of the natural variations in heart rate [8]. It quantifies the overall spread in both heart rate and R-R intervals over a given time period[9]. As a result, HRV evaluates the tonic baseline of autonomic activities. Continuous physiological changes of sinus cycles reflecting a balanced sympathovagal state and proper HRV are indicative of a normal heart with an intact ANS. Necrotic cardiac injury causes a decrease in HRV due to a sympathovagal imbalance caused by altered activity of afferent and efferent fibres of the ANS and local neural control [10-11].

By calculating the difference in heart rates between the shortest and longest intervals in an electrocardiographic recording of six cycles of deep breathing, heart rate variability with deep breathing (HRVdb) provides a very sensitive indicator of cardiovagal or sympathetic cardiac function. As a result, HRVdb is a valid and sensitive clinical diagnostic for detecting cardiac vagal dysfunction in a variety of autonomic illnesses [12]. When six cycles of deep breathing are recorded on an electrocardiogram in one minute, this is the difference in beats per minute between the shortest and longest heart rate interval. The shortest time between breaths will occur during inspiration, the longest during exhalation. When a sympathetic tone dominates, HRV is low, and vice versa [13].

Autonomic dysfunction has been linked to obesity, hypertension, and diabetes mellitus separately. An inadequate sample size of the obese participants and the non-inclusion of the control group may explain why a recent study found no significant difference in autonomic function between the obese and non-obesity hypertension groups [14-15].

Thus, this study seeks to determine if obesity with hypertension disrupts autonomic processes further so that early intervention can reduce mortality, morbidity, and health hazards associated with hypertension and obesity.

Materials And Methods:-

Location of the study as well as ethical considerations

The study was conducted at the U.P. University of Medical Sciences, Saifai,which is located inEtawah, after receiving approval from the ethical committee. The participants in the study were asked for their informed and signed consent before they were allowed to continue.

Study design

There were total of 105 people that took part in this research, and they were split up into three groups, each with a size of 35 people. These groups were labelled Group I (obese hypertensive), Group II (non-obesity hypertensive), and Group III (normal weight hypertension) (non-obese normotensive, control). The size of the sample was determined based on an estimation provided by prior research of a comparable nature, which assumed a power of 80% and an alpha error of 5%.

Standards for admittance

According to the BMI cut-off for the Indian population, patients of both sexes aged 30 to 45 years old who visited the Medicine OPD, UPUMS, and had a BMI that was greater than 25 kg/m2 were classified as obese. Patients who

were diagnosed with hypertension and receiving therapy, smokers, drinkers, and those who had consumed coffee, tea, or cola during the previous 48 hours were not included in our study sample. After three readings taken on separate days, blood pressure that measures more than 140/90 mm Hg on average is regarded to be hypertensive, whereas blood pressure that measures less than 130/80 mm Hg on average is considered to be normotensive.

A measurement of the heart rate variability

The HRV was assessed using a straightforward bedside test that lasted for one minute and involved deep forced breathing. The CARDIART 6108-T electrocardiograph was utilised in the performance of the test. Every morning between 10 and 11 in the morning, an ECG was taken. The participants were instructed to maintain complete silence while lying in a supine position and a conventional twelve-lead electrocardiogram was connected. For the sake of our investigation, we decided to use rhythm strip with lead II moving at a speed of 25mm/sec. Following the completion of the baseline recording, the patients were given the directive to take slow, deep breaths at a rate of 6-8 per minute. We manually measured the HRV interval using a sliding calliper. The HRV interval is the R-R interval that is found between two consecutive QRS complexes. R-R intervals that surrounded premature ventricular contractions were disregarded as irrelevant. The difference in beats per minute between the lowest heart rate interval and the longest heart rate interval was used to compute HRV for one minute [16].

Statistical analysis of the data

The data are shown as the Mean Standard Deviation, and inferential statistics were determined using One Way ANOVA and Tukey's Post Hoc test; p-value< 0.005 was regarded as significant. The data were entered into Microsoft Excel, and SPSS® Version 28.0 was used to do the analysis.

Results:-

The median ages of the groups did not differ significantly. Allgroups had a mean age between 37-39 years. Obese people weighted on average 66.8±6.5 kg, while the non-obese weighted 55.8±6.5 kg and the control group weighted 58.4±4.5kg, the averages height for the obese 1.52±0.72 meter, Non-obese height 1.58±0.112 meter, and the height of Control groups 1.62±0.096 meter were found. The average body mass index (BMI) of the obese population was 28.91±2.28Kg/m^2, whereas that of the non-obese was 22.40±2.12Kg/$m^2$and that of the control group was 22.29±1.86Kg/m^2(Table 1).

Table1:- Patient characteristics and study variables.

Variables	Group	Mean	Std. Deviation	95% Confidence Interval for Mean	
				Lower Bound	**Upper Bound**
Maximum HR (beat/minute)	I	85.60	1.30	84.30	86.90
	II	87.80	1.02	86.78	88.82
	III	90.20	2.00	88.20	92.20
Minimum HR (beat/minute)	I	72.00	0.98	71.02	72.98
	II	68.60	1.72	66.88	70.32
	III	66.80	2.06	64.74	68.86
HRV	I	14.40	1.42	12.98	15.82
	II	19.20	1.62	17.58	20.82
	III	23.40	2.00	21.40	25.40
Age (years)	I	54.60	2.88	51.72	57.48
	II	54.20	3.46	50.74	57.66
	III	52.80	3.36	48.92	55.64
Weight (Kg)	I	66.80	7.60	59.20	68.40
	II	55.80	6.80	49.00	62.60
	III	58.40	4.80	53.60	63.20
Height (m)	I	1.52	0.072	1.448	1.592
	II	1.58	0.112	1.468	1.692
	III	1.62	0.096	1.524	1.716
BMI (Kg/m^2)	I	28.91	2.28	26.63	31.19
	II	22.40	2.12	20.28	24.52
	III	22.29	1.86	20.43	24.15

Through the use of One-Way ANOVA, we discovered that there was a statistically significant distinction between the three groups with regards to the average maximum heart rate (F) = 30.960 bpm, P= 0.003), the average minimum heart rate (F) = 99.987 bpm, P= 0.002, and the HRV (F) = 219.230 bpm, P= 0.004 (Table 2). Tukey's Post Hoc analysis showed that both obese and non-obese hypertensives recorded significantly lower Maximum Heart Rates than control subjects (90.20±2.00 bpm). The Max HR measured in hypertensives who are obese and those who are not obese differs significantly. With regards to Minimum HR, both Obese (72.00±0.98 bpm) and Non-obese hypertensive (68.60±1.72 bpm) individuals record significantly higher values than Controls (66.80±2.06 bpm) individuals. There is also a statistically significant difference between people with high blood pressure who are obese and those who are not obese. When compared to controls (23.40±2.0 bpm), both obese and non-obese hypertensives had significantly lower recordings of one-minute HRV. Additionally, the average HRV over the course of one minute is significantly different between obese (14.40±1.42 bpm) and non-obese (19.20±1.62 bpm) hypertensives.

Table 2:- Displays the mean differences in Maximum HR, Minimum HR, and HRV between Obese Hypertensives, Non-Obese Hypertensives, and the Control group.

		Sum of Squares	Degree of freedom	Mean Square	F-Ratio	P-Value
Max HR	Between Groups	158.840	2	87.860	30.960	0.003
	Within Groups	242.680	93	1.440		
	Total	401.520	95			
Min HR	Between Groups	554.000	2	269.820	99.987	0.002
	Within Groups	257.820	93	1.586		
	Total	811.820	95			
HRV	Between Groups	1308.420	2	649.650	219.230	0.004
	Within Groups	272.820	93	1.680		
	Total	1581.240	95			

Tukey's Post Hoc revealed a significantly lower recording of Maximum Heart Rate among the Obese Hypertensives (85.60±1.30 bpm) and Non–obese Hypertensives (87.80±1.02 bpm) in comparison to the Controls (Non-obese and Non-Hypertensive) (90.20±2.00 bpm). The maximum heart rate that was recorded in obese and non-obese hypertensive patients was shown to be significantly different from one another. In terms of the Minimum HR, there is a substantial difference between the Controls (66.80±2.06bpm) and the Obese (72.00±0.98bpm) and Non-obese hypertensive (68.60±1.72bpm). The Controls have a lower recording of the Minimum HR than the Obese have (72.00±0.98bpm). A statistically significant gap can also be seen in the mean of the minimum heart rate (Min HR) between obese and non-obese hypertensives. When compared to the control group, both obese and non-obese hypertensives had significantly lower recordings of their mean one-minute HRV than the controls (23.40±2.00bpm). There is also a substantial difference in the mean HRV for one minute in those who are obese (14.40±1.42bpm) and those who are not obese (19.20±1.62bpm) with regard to hypertension (Figure 1).

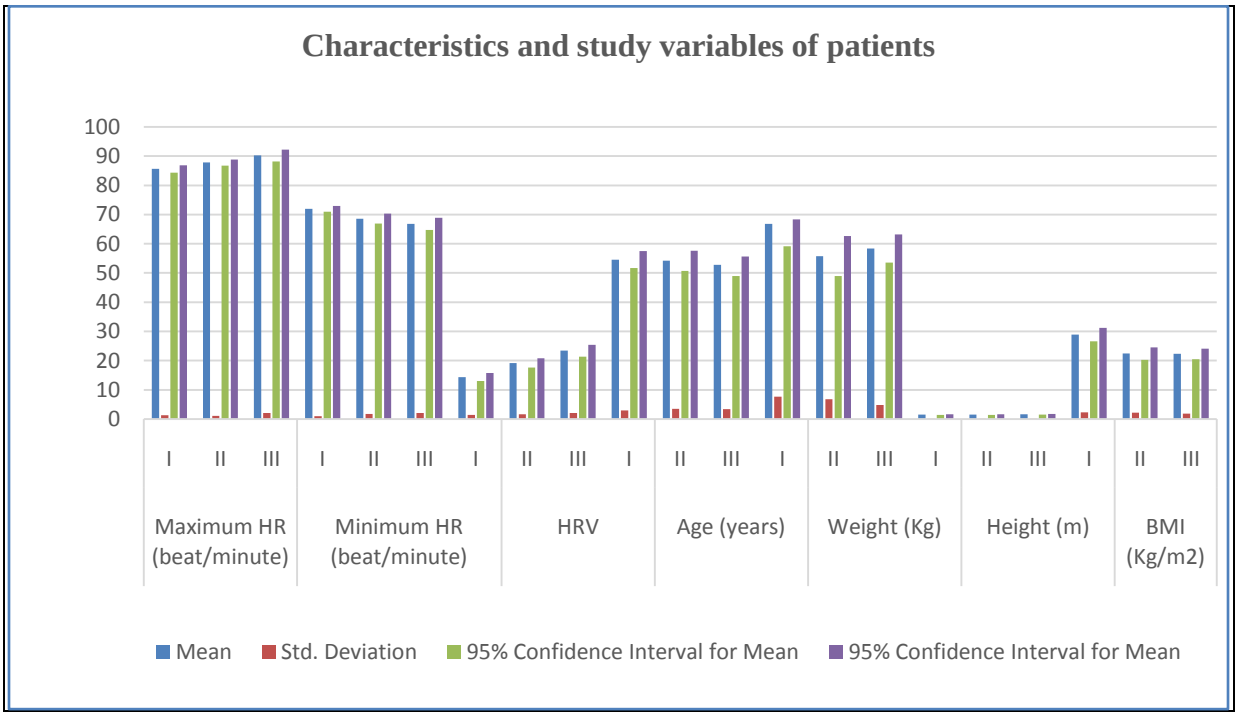

Figure 1:- Characteristics and study variables of patients.

Discussion:-

An altered baroreceptor reflex i.e. autonomic system, detectable by HRV, makes the arterial pressure of hypertensive and obese persons more sensitive to different types of physical stimuli. To determine whether HRV fluctuations had additive effects in obese hypertensive patients, this study compared the responses of newly diagnosed non-obese hypertensives, obese hypertensives, and controls. If obesity and hypertension both impair HRV responsiveness, then patients with both conditions may experience more severe HRV changes that pose a threat to their health. This hypothesis was tested by measuring HRV, which revealed a decrease in HRV in both obese and non-obesity hypertensives compared to normal healthy persons and a greater decrease in HRV in obese hypertensives compared to non-obese hypertensives.

In India, an increasing percentage of the population is displaying signs of being overweight or obese, and the prevalence of hypertension is also rising. Overeating processed foods and high-fat diets contributes to the epidemic of obesity. According to the World Health Organization, obesity rates are rising in every region of the world [17-18]. Being overweight or obese is a risk factor for several diseases on its own. There is a strong correlation between hypertension and both genetic and environmental factors. Increased sympathetic tone can be seen from increased catecholamine levels in both obesity and hypertension [19]. Increased sympathetic activity causes the kidneys to secrete more renin, which in turn raises blood levels of the renin antagonizer angiotensin. Reducing HRV, angiotensin II stimulates the sympathetic ganglia and adrenal medulla but inhibits the vagus nerve's output to the heart [20-24].

Current findings demonstrate that HRV is consistently decreased in patients with newly diagnosed hypertension. We find that the autonomic instability associated with lower HRV is reflected in the fact that there are only little differences in max HR and large fluctuations in min HR between obese and non-obese hypertensives [21]. We also found that the connection between HRV and blood pressure persisted across the entire range of blood pressure in patients with newly diagnosed HTN. Previous research has revealed that individuals with hypertension alone and people who are obese alone have lower HRV [3]. Our findings are the first to show that the HRV of obese hypertensives is significantly lower than that of non-obese hypertensives, with larger variations in max HR and min HR between the two groups. This suggests an alteration in parasympathetic and sympathetic outflow, which is supported by the stronger correlation of HRV with obese hypertensives than with non-obese hypertensives. The sympathetic and parasympathetic nervous systems are responsible for short-term regulation of heart rate, therefore changes in heart rate can indicate autonomic instability [3]. The results of our study reveal that the connection of

blood pressure with HRV was not reliant on HR, even though HR was higher in obese hypertensives compared to non-obese hypertensives and the control group.

Finally, in a population like India's, where the prevalence of diabetes has surpassed 50%, regular monitoring of HRV and diagnosis of autonomic instability and rehabilitative interventions can help lower the occurrence of not only hypertension and obesity but possibly diabetes as well. This study's flaw was that it did not attempt to calculate how long the participants had been overweight. To determine whether hypertensive people with lower heart rate variability have an increased risk of cardiac mortality, prospective trials are required. Traditional methods of measuring HRV, such as those utilised in the present investigation, will be superseded by the superiority of the updated version of HRV analyzer.

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