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COMPARATIVE STUDY OF RATE PRESSURE PRODUCT IN OBESE WOMEN WITH NON OBESE WOMEN

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Abstract
Background and Objective: Nowadays Obesity is considered as global epidemic. It exacerbates a large number of health problems. Obese people are at risk of impaired cardiac performance, commonly seen in women. So our study aimed to assess the Rate pressure product in obese women and to compare the same with non obese women aged 35-55years. As above mentioned parameters are associated with increased risk for cardiovascular diseases
Methods and Material: Study A comparative cross sectional study including 140 women (70- obese , 70- non obese) of age 35-55years. The study group was divided into four according to BMI Group I (BMI 25-29.9 kg/m2), Group II(BMI 30-34.9 kg/m2) , Group III(BMI 35 – 39.9 kg/m2) , Group IV (BMI > 40 kg/m2) including Control(BMI < 25 kg / m^2) . Anthropometric parameters like height (cm), weight (Kg), WHR, BMI were recorded. After 20 mins of rest in supine position ECG and brachial SBP, DBP were recorded. HR was calculated by using formula 1500/R-R. The rate pressure product was calculated as = SBP X HR. As RPP is an index of myocardial oxygen consumption.
Results: We found statistically significant higher values of RPP in group IV when compared to control group. Also found statistically insignificant increase in values of RPP in groups I, II, III.
Conclusion: obesity is a significant predictor of increased MVO_2 and decreased efficiency. This may cause increase in fatty acid metabolism & decrease in cardiac efficiency in obese women.
Keywords: Body Mass Index, Rate Pressure Product, Myocardial oxygen consumption, Heart rate.

1. Introduction:

There is an increasing concern worldwide regarding the fact of obesity is that, it is growing in a out of control manner. Nowadays obesity is considered as global epidemic[1].

Other than the genetic predisposition, adoption of sedentary life style, inappropriate intake of caloric rich easily available junk food and automated working profile has made the environment favorable to the development of obesity[2]. Obesity is among the most significant contributors to ill health. It aggravates a large number of health problems, either independently or in club with other diseases[3]. It is an independent risk factor for cardiovascular diseases. There is an increased prevalence of heart failure in obesity. "Obesity cardiomyopathy" is the important clinical entity. It is featured by left ventricular remodeling, reduced efficiency & left ventricular diastolic dysfunction[4].

However the mechanisms involving the relation between heart failure & obesity in humans aren't well understood. But the data from animal studies suggest that obesity increases myocardial fatty acid metabolism & oxygen consumption leading to increased oxidative stress, cardiac dysfunction & apoptosis[5].

We limited our study population to women because obesity is more common in women than in men and the relative risk of heart failure is greater in obese women than in obese men[5]. Women gain weights mostly between ages of 35 – 45 yrs of age.

The aim of present study was to assess the Rate-Pressure product in obese women and to compare the same with non obese women. As above mentioned parameters are associated with increased risk for cardiovascular diseases. [6]

Rate-Pressure Product is a product of heart rate & systolic blood pressure is a major determinant of myocardial oxygen consumption. It is an indirect & easy method of measuring MVO_2.

AS direct invasive technique of measurement of MVO_2 is difficult in routine clinical practice[7].
The study will be helpful to those persons who are overweight or obese, so that these subjects can be identified and proper instructions such as change in lifestyle, regular exercise, and proper diet management are advised. As weight loss in humans ameliorates obesity related cardiac hypertrophy & diastolic dysfunction. Weight loss also improves the excessive myocardial fatty acid uptake & myocardial oxygen consumption.

2. Material &Methods:

This is a comparative cross sectional study assessing the RPP in obese women to non obese women. The study was conducted on 140 (control= 70, obese= 70) volunteer women of age 40-55 years. After explaining details of the study, Informed consent was obtained from each of the subject. Ethical clearance was obtained from institution.

2.1 Inclusion criteria: Women with BMI between< 25 kg / m^2- > 40 kg/m^2 aged between 40-55 years, included in the study.

2.2 Exclusion criteria: subjects with Diabetes mellitus or any cardiovascular diseases, taking medications interfering with vascular reactivity and those are underweight, pregnant and lactating women.

All the parameters were recorded in the departmental laboratory between 8 to 10 am. Anthropometric parameters like height (cm), weight (Kg), WHR were recorded. BMI was calculated for each subject from her height (meter) and weight in kg. Depending upon body mass index, these subjects were divided into following groups.

(Control –BMI < 25 kg / m^2),(Group I - BMI 25-29.9 kg/m2) (Group I - BMI 25-29.9 kg/m2),(Group II- BMI 30-34.9 kg/m2) ,(Group III- BMI 35 – 39.9 kg/m2) ,(Group IV- BMI > 40 kg/m2) .The above groups were made on the basis of WHO classification of obesity. After 20 mins of rest in supine position ECG and brachial SBP, DBP were recorded. HR was calculated by using formula 1500/R-R. The rate pressure product was calculated by using the formula: Rate Pressure Product (mm Hg/min) = Systolic Blood Pressure X Heart Rate.

2.3. Statistical Methods: All values are presented as Mean ± Standard Deviation. Comparison of mean values of parameters between groups and controls was done by using student's 't' test. p Value <0.05 is considered as significant.

3. Results:

Table 1: comparison of parameters between control and group I

PARAMETERS	CONTROLS Mean ± SD	GRI Mean ± SD	't' TEST	'P' VALUE
HEART RATE	75.30±9.21	78.44±7.05	0.12	>0.1
SBP	120.80 ±6.24	121.33 ±15.11	0.14	> 0.1
DBP	79.70 ±4.87	80.33 ±10.48	0.40	> 0.4
BMI	21.68 ±2.35	30.92 ±2.65	0.002	<0.01**
W/H RATIO	0.78 ±0.03	0.84 ±0.06	0.000089	<0.001**
RPP mmHg/ min	9094 ± 1193.39	9308 ±1407.12	0.33	>0.1

Values are expressed as (mean ± SD). *p<0.05 significant, **p<0.01 Highly significant, ***p<0.001 very highly significant; SBP: systolic blood pressure; DBP: diastolic blood pressure; BMI: body mass index, W/ H ratio. Values of BMI and W/H ratio are significantly higher (0.0 1, 0.001 respectively) in group I compared to control. Statistically insignificant higher values of RPP in group I compared to control.

Table 2: comparison of parameters between controls and group II

PARAMETERS	CONTROLS Mean ± SD	GRII Mean ± SD	't' TEST	'P' VALUE
HEART RATE	75.33 ± 2.83	76.88 ± 5.11	0.14	> 0.1
SBP	127.22 ± 7.39	119.38 ± 13.60	0.02	< 0.05*
DBP	80.67 ± 5.13	74.50 ± 5.68	0.0012	<0.01**
BMI	21.92 ± 1.50	30.92 ± 2.72	$2.3X10^{-11}$	< 0.001***
W/H RATIO	0.77 ± 0.02	0.84 ± 0.08	$1.7X10^{-3}$	< 0.001***
RPP mmHg/min	9405.76±616.91	9208 ± 1126.54	0.36	> 0.1

Values are expressed as (mean ± SD). *p<0.05 significant, **p<0.01 Highly significant,

***p<0.001 very highly significant; SBP: systolic blood pressure; DBP: diastolic blood pressure; BMI: body mass index, W/ H ratio. Values of SBP and DBP are statistically significant in group II compared to control. BMI and W/H ratio are very significantly higher in group II compared to control.

Table 3: comparison of parameters between control and group III

PARAMETERS	CONTROLS Mean ± SD	GRIII Mean ± SD	't' TEST	'P' VALUE
HEART RATE	76.20 ± 3.99	76.50 ± 5.23	0.41	> 0.1
SBP	120.80 ± 8.95	123.70 ±10.43	0.17	> 0.1
DBP	79.40 ± 7.65	80 ± 7.68	0.40	> 0.1
BMI	22.50 ± 2.39	30.93 ± 4.39	$1.1X10^{-8}$	< 0.001***
W/H RATIO	0.78 ± 0.02	0.85 ± 0.05	$1.7X10^{-6}$	< 0.001***
RPP mm/Hg	8865.7 ± 1684.06	8896±2284.21	0.2	> 0.1

Values are expressed as (mean ± SD). *p<0.05 significant, **p<0.01 Highly significant,
***p<0.001 very highly significant; SBP: systolic blood pressure; DBP: diastolic blood pressure; BMI: body mass index, W/ H ratio. Values of BMI and W/H ratio are significantly very high in group III compared to control. Statistically insignificant higher values of RPP in group III compared to control.

Table 4: comparison of parameters between control and group IV

PARAMETERS	CONTROLS Mean ± SD	GRI Mean ± SD	't' TEST	'P' VALUE
HEART RATE	75.50 ± 3.21	76.75 ± 4.84	0.2	> 0.1
SBP	126.83 ± 5.62	133.63 ± 16.07	0.06	> 0.05
DBP	82 ± 10.13	80.13 ± 8.96	0.30	> 0.1
BMI	21.71 ± 2.54	31.94 ± 5.57	$7.4X10^{-7}$	<0.001***
W/H RATIO	0.79 ± 0.02	0.86 ± 0.05	$1.4X10^{-3}$	<0.001***
RPP mmHg/min	9971.28± 1048.43	10268.5±1232.85	0.04	<0.05*

Values are expressed as (mean ± SD). *p<0.05 significant, **p<0.01 Highly significant,
***p<0.001 very highly significant. SBP: systolic blood pressure, DBP : diastolic blood pressure, BMI : body mass index, W/ H ratio. Statistically significant increase in values of BMI, W/H ratio and RPP in group IV compared to control.

4. Discussion:

Results of our study demonstrate that, in middle aged women an insignificant increase in myocardial oxygen consumption with increasing BMI. This could be due to altered left ventricular (LV) remodeling, increased hemodynamic load and enhanced neurohormonal activation[3].

The increase in MVO_2 associated with an increase in BMI is likely related to the effect of obesity on cardiac remodeling and the fact that obesity increases sympathetic tone, preload and fatty acid metabolism. Increased fatty acid uptake and oxidation by the heart can also increase MVO_2, because more oxygen is required to generate ATP from fatty acid than by glucose metabolism[5]. In agreement with our study, Linda R. Peterson *et al* demonstrated in young women, obesity is a sign ificant predictor of increased MVO_2 and decreased cardiac efficiency[5]. Increased myocardial oxygen consumption leads to increased oxidative stress, apoptosis and cardiac dysfunction[4].

Y T Zhou showed from animal studies that there is an initial increase in myocardial fatty acid oxidation and myocardial O_2 consumption which can decrease cardiac efficiency further. This can leads to an accumulation of fatty acid intermediates and increased ceramide production which cause cardiomyocyte apoptosis and impairment of cardiac function[8].

Heinrich taegtmyer *et al* reported that both myocardial blood flow & MO_2 were directly related to body mass index in women but not in men. In spite of greater cardiac work the heart's efficiency was less in women than in men. It is due to impaired coupling of fatty acid oxidation to adenosine triphosphate production[9].

H K Vincent, *et al* reported in animal studies that RPP was significantly higher in both fatty (fa/fa) animals and the obese high fat fed animals. This elevation appeared to be predominantly due to elevated SBP. In obesity elevations in SBP increase after load, ventricular contractile work & ventricular O_2 consumption[10].

P Kopelman *et al* reported Changes in lifestyle are major contributors to the current epidemic of

overweight and obesity. The use of low-calorie diets with a treatment period beyond 6 months has been associated with a mean weight loss of 8%, although with longer use (3–4.5 years), this is reduced to 4%[3].

Hanna Laine *et al* reported in their study that myocardial oxygen consumption per gram of tissue was significantly higher in the left ventricular hypertrophy group than in other groups[11].

Our finding that increasing BMI is an independent predictor of increased MVO_2 extends the findings of previous ex vivo studies to obese humans. In this study we showed that, in middle aged women obesity is significant predictor of increased MVO_2 and this may lead to decreased cardiac performance.

However, recent evidence highlights a requirement for 45–60 min per day to maintain lowered weight and prevent weight regain. Reduction in the time spent in sedentary behaviours (such as television watching) is an important strategy for increasing physical activity and energy expenditure in children and young people[12].

Conclusion:

We found that an increase in BMI is associated with an increase in MVO_2. Obesity is an independent predictor of increased MVO_2. This may cause increase in fatty acid metabolism & decrease in cardiac efficiency. These metabolic changes may play a role in the pathogenesis of decreased cardiac performance in obese women.

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