

The Impact of Obesity on Physiologic Indicators

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Abstract—Obesity results in an alteration in the stress response that often results in adverse perinatal outcomes. This study investigated physiologic changes in 21 obese and 20 overweight women during pregnancy and the impact on vagal response (heart period and respiratory sinus arrhythmia), oxygenation, hemoglobin A1c (HbA1c) and systolic blood pressure at 20, 28 and 36 weeks of gestation. The impact of obesity on perinatal outcomes was investigated. Blood oxygen, systolic blood pressure, and HbA1c levels were significantly higher for the obese women as compared with overweight women. Monitoring physiologic mal-adaptation may permit early detection and intervention to improve perinatal outcomes.

Keywords— *vagal response; oxygenation; blood pressure; hemoglobin A1c; obesity; overweight; pregnancy*

I. INTRODUCTION

The staggering burden associated with obesity among women of childbearing age and the increased risk of adverse perinatal outcomes associated with it invites the need for prevention and early detection and treatment of complications during pregnancy. Increased knowledge of the autonomic nervous system response, Hemoglobin A_{1c} (HbA_{1c}) and oxygenation might facilitate the recognition of a maladaptive response pattern and promote an early intervention toward increasing positive maternal and neonatal outcomes. Before preventative strategies can be considered, unraveling the mechanistic steps wherein changes in physiology that increase the risk of adverse perinatal outcomes occur will be a critical necessity. Because nurses are the primary patient educators, nurses are in a unique position to monitor physiologic indicators to identify alterations in autonomic response and pregnancy maladaptation, impart diagnostic treatment, and initiate preventative education that may otherwise go unsaid. [1] In order to improve outcomes, nurses and patients should collaborate to formulate a plan that incorporates both evidence-based data and patient preferences that are individually tailored to meet the needs of the pregnant woman.

The purpose of the present study was to investigate if there are differences between obese and over-weight women in adaptation to pregnancy in physiologic indicators. This research was previously supported by Award F: 31 NR009611 by National Institute of Nursing, NIH. A

comparison was made between obese and over-weight women on physiologic indicators at 20, 28, and 36 weeks of pregnancy in women with varied body mass indices (obese BMI with ≥ 30 kg/m², and over-weight women with BMI 24.9-30 kg/m²). The dependent physiologic indicators included vagal response (heart period [HP] and respiratory sinus arrhythmia [RSA]); oxygenation (peripheral oxygenation [SPO₂] and oxy-hemoglobin [HbO₂]); average hemoglobin blood glucose level (HbA_{1c}); and systolic blood pressure (SBP). Previous researchers have examined one or two of the physiologic indicators in obese women with BMI ± 30 kg/m² and non-obese women with BMI 20-24.9 kg/m². However, little is known about physiologic changes in the over-weight women (BMI 24.9-30 kg/m²). This may be the first study to examine the association of obesity and over-weight status in several physiologic indicators in combination at 20, 28 and 36 ± 2 weeks gestation. The hypothesis of this study was that at 20, 28 and 36 weeks' gestation, obese women as compared to over-weight women would have lower vagal response and oxygen saturation and higher HbA_{1c} and SBP. An additional purpose was to describe the frequency of pregnancy complications experienced by the study groups.

II. REVIEW OF THE LITERATURE

Obesity during pregnancy is a common condition that has serious impact on the stress response to physiologic pregnancy changes. According to the National Health and Nutrition Examination Survey,[2] for women ages 20-39 years, the age-adjusted prevalence of obesity defined as having a body mass index ≥ 30 kg/m² was 31.8%. The range was from 11.4 % among non-Hispanic Asian, 32.8% among non-Hispanic white, and 44.4% among Hispanic women to 56.6% among non-Hispanic black women [2] Obesity results in an alteration in the stress response that may impair the cardiovascular system and result in adverse perinatal outcomes. For example, obesity during pregnancy affects cardiovascular function in many ways and is associated with fast heart rates,[3] elevated sympathetic activity,[4] hyperglycemia [4,5,6] and hypertension.[7,8] Further, obese women are more likely to have higher rates of induction, dysfunctional labor patterns, risk of cesarean births; [9,10] and risk of preterm birth.[11]

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The autonomic nervous system is a control system that acts largely unconsciously and regulates heart rate, digestion, respiratory rate, pupillary response, urination, and sexual arousal. This system is the primary mechanism in control of the fight-or-flight response, and its role is mediated by two different components. Stimulation of the sympathetic component of the ANS originates in the spinal column and mediates physiologic resources of fight or flight that are manifested as increases in heart rate and blood pressure, mobilization of needed energy stores, and heightened arousal.[12] In contrast, the parasympathetic branch originates in the spinal cord and medulla and is responsible to activate the "rest and digest" response that results in increased blood flow and tissue oxygenation and a return to homeostasis after the fight or flight response. When the vagal response is high, sense of control may be increased, and thus the body is more adaptive in response to environmental demands.[13] It is expected that in individuals with poor vagal response, sympathetic influences to the heart will remain unchecked, and the person will not have a calm physiologic state. Obesity and impaired insulin sensitivity are noted to drive the sympathetic activity and decrease vagal response.[13] With obesity, the expected physiologic response may be disturbed due to the strain of excessive sympathetic tone and reduced adaptive capacity of the parasympathetic nervous system.[4] The inadequate adaptive capacity may result in adverse outcomes, such as hypertension. Indicators of the vagal response included in this paper are heart period and respiratory sinus arrhythmia. Heart period (HP) is the time in milliseconds between the consecutive R-waves of the electrocardiogram and has an inverse relationship to heart rate. [13] Respiratory sinus arrhythmia (RSA) arises from the parasympathetic branch to the heart and acts as a brake to slow the heart rate and allow adaptation to environmental changes.[13] Interventions that have been shown to increase the vagal response include detection and treatment for gestational diabetes, dietary education to promote euglycemia, [4,14] and relaxation techniques, such as deep breathing exercises, to decrease stress. [15] Techniques that increased the vagal response in various non-pregnant populations have included self-hypnosis [16] and Yoga therapy. [17]

During pregnancy vagal response decreases with increasing gestation, and the body reacts quicker to sympathetic activation. However, this increased sympathetic response may occur earlier for obese women and result in attenuated vagal response and, subsequently, reduced adaptive capacity to adjust to stress caused by physiologic pregnancy changes. Increased sympathetic activity results in vasoconstriction, and this may lead to increased heart rate and decreased oxygen. With an intact physiologic oxygen transport process, oxygen is absorbed in the lungs by the deoxygenated red blood cells and carried to the peripheral tissues. Additionally, the oxygen carrying capacity of blood may be affected by hyperglycemia and elevated glycosylated hemoglobin levels (HbA_{1c}).[4,18] Hyperglycemia in early pregnancy may result in vasoconstriction that affects placental development, thus contributing to the susceptibility to hypoxia seen in these infants.[19] Hemoglobin A1c forms when excess glucose binds with hemoglobin and reflects the average blood glucose level over the previous 60 to 90 days.

Thus, HbA_{1c} reveals whether glucose levels have been controlled. Increased HbA_{1c} may result in increased sympathetic activity, decreased vagal response, and decreased oxygen. [20] Obese women tend to have a higher HbA_{1c} at the beginning of pregnancy, and it continues to increase during the pregnancy: [6,18] An elevated HbA_{1c} level decreases the cardiac brake and increases sympathetic activity that may lead to vasoconstriction, thus increasing the possibility of hypoxia.[6] SBP has been reported to decrease early in gestation, [21] reaching a nadir by 20 weeks, then the SBP starts a gradual increase, returning to or exceeding early pregnancy levels by 35 weeks. [22] From the 1st trimester onwards, SBPs were higher in each trimester for obese women as compared to non-obese pregnant women. [4,23] However, changes over time were not significant.[23] Less is known of the differences between obese and overweight pregnant women.

III. METHOD

This was a minimally-invasive, repeated-measures study of obese or overweight pregnant women who visited the university's low risk perinatal clinic. The study was approved by the research ethics committee, and participants provided informed consent before any data collection. Although consent forms were available in English and Spanish, participants' signed the English form. All investigations were performed in accordance with Declaration of Helsinki as revised in 2000. The SpO₂ was the determining factor in calculating the power for the study, as it indicated the need for the largest number of participants. The mean of SpO₂% for pregnant women with risk factors was 96% ± 2, as compared with the mean of 98% ± 2 for pregnant women free of risk factors, for a difference between the means of -2 and an effect size of 1. [25] For an effect size of 1, it was determined that 17 subjects would be needed to detect a difference with alpha at .05 and a power of 80%. [25] Considering a 20% attrition rate for a longitudinal design, a sample size of 20 per group with complete assessments at all three time points was proposed for this study. Eligible women were 19 years or older, pregnant at 20 +/- 2 weeks gestation with a singleton pregnancy, spoke English or Spanish, were able to attend the clinic for antenatal care, and were willing to commit to three assessments at 20, 28 and 36, +/- 2 weeks of gestation. Potential participants were excluded if they had known genetic hemoglobin diseases, severe health problems involving the brain, heart and lungs, or allergies to skin electrodes.

The participant's assessment for the study was conducted after a scheduled prenatal visit. Information collected in the first assessment was height, self-reported pre-pregnancy weight, current weight, and resting blood pressure. Participants were seated in a comfortable chair in a quiet room across the hall from the prenatal clinic. After familiarization with the laboratory and monitoring equipment, the ECG electrodes were attached to the chest wall and connected to the vagal tone monitor. The peripheral SpO₂ probes were applied to the client's second or the third finger. Participants were allowed another 3-5

minutes of rest and quiet sitting before the recording of vagal response was started. They were instructed to breathe naturally during data collection. The researcher assessed vagal response, SpO₂, and SBP; retrieved the blood for analysis of HbO₂ and HbA_{1c}; and clarified directions for the repeat assessments at 28 and 36 weeks gestation. The information gathering, assessments, and blood draw took approximately 45 minutes to complete. Participants were carefully observed for any sign or symptom of intolerance to positioning, such as restlessness, breathlessness, pallor, dizziness, or faintness. Although no intolerance occurred, the plan was to discontinue the ECG recording if adverse signs and symptoms were to occur. At the second and the third observations, the same procedure was carried out as on the first day. The weight measurements were updated.

IV. MEASUREMENTS

A. Data Collection

Demographic data was collected on maternal age, number of pregnancies, height, maternal pre and early pregnancy weight, race, ethnicity and country of origin, marital status, education, and if full time homemaker. A face to face interview was conducted to collect the information.

B. Independent Variable: Obesity

Participants were weighed to the nearest kilogram using a balance beam scale, and height was measured to the nearest meter on a wall-mounted rod ruler. The scale was balanced according to the manufacturer's recommendations. Shoes, sweaters, coats and other outer garments were removed prior to weighing. The formula used for BMI was weight (kg)/ height (m²). Women were grouped as obese or over-weight. Thus obesity was defined as a woman having a BMI of ≥ 30 kg/m² and over-weight as a BMI of 25-29.9 kg/m², which were determined by a measured weight at the first prenatal visit. It is recognized that excessive weight gain early in pregnancy could lead to a misclassification of obesity status based on weight objectively measured at the first prenatal visit, or at 20 +/- 2 weeks. To address this issue, women were asked to report their pre-pregnancy weight. Since there were no discrepancies between obesity status based on self-reported pre-pregnancy weight and obesity status based on measured weight evaluated at 20 +/- 2 weeks of pregnancy, the latter was used in the analysis.

C. Dependent physiologic indicators.

- Vagal response was measured by times series analyses of the HP, and RSA using a vagal tone monitor (Delta Biometrics, Bethesda, MD), and MXedit software. [13,26] Heart period (HP) is the time in milliseconds between the consecutive R-waves of the electrocardiogram and has an inverse relationship to heart rate. [27] Heart rate was measured by the number of R waves per epoch of 30 seconds recorded on the electrocardiogram recorder by placing three non-invasive leads on the chest and abdomen. Respiratory sinus arrhythmia (RSA) represents the amount of heart rate variability there is between typical breathing cycles between 0.1-0.40 Hertz, which is approximately

between 2.5 and 6.7 seconds between breathing cycles, for adults.[27, 28] During the process of inhalation, RSA temporarily suppresses vagal activity, causing an immediate increase in heart rate. Exhalation then decreases heart rate and causes vagal activity to resume. Thus, RSA acts as a vagal brake on the heart rate, allowing the heart to do less work while maintaining healthy levels of blood gases. [27] Fast heart rates are associated with increased RSA.

- Peripheral oxygenation (SpO₂), defined as the percentage of oxygenated hemoglobin, was measured as oxygen saturation by a Nellcor (NBP- 290, Pleasanton, CA) pulse oximeter with the protocol developed by the manufacturer. [29] The oximeter probe was applied to either the second or the third finger of the dominant hand. SpO₂ is accurate within $2\% \pm 1$ SD or higher for the ranges of 70-100% oxygen saturation.[30] Oxygenation was also assessed with blood analysis. Using adult blood, the difference for SpO₂ against HbO₂ was 1.9 ± 0.19 for 36 samples, and the correlation between SPO₂ and HbO₂ was 0.68.[30] Therefore, venous blood was drawn to test oxyhemoglobin (HbO₂), and was analyzed according to the manufacturer's protocol utilizing the OSM3 hemoximeter (Radiometer A/S Copenhagen NV, Denmark).[31]
- Blood samples were collected and analyzed to determine the average blood glucose level (HbA_{1c}) at 20, 28, and 36 weeks of gestation. HbA_{1c} forms when excess glucose binds with hemoglobin and reflects the average blood glucose level over the previous 60 to 90 days. The HbA_{1c} was measured by a high performance liquid chromatography (HPLC) procedure as per laboratory protocol and was then converted to the HbA_{1c} equivalent value. This method has the sensitivity to detect 0.1% (50 ng) of total hemoglobin in 0.5 ml of whole blood.[32]
- Systolic blood pressure (SBP) was measured using a digital oscillometric monitor with an appropriately-sized upper arm cuff (model: Omron 711c, HRM, USA Inc.) [33] in accordance with the American Heart Association's general guidelines. [34] At the 20, 28, and 36 +/- 2 week assessment times, the blood pressure measurements consisted of 3 measurements. Blood pressure measurements reported represent an average of the first reading at the initial point of the assessment, the second after being seated for 10 minutes in the assessment room, and the third after a 5 minute rest period at the end of the assessment. The researcher used the same equipment for all readings. The measurements were averaged and used in the analysis because this average was considered representative of the participant's blood pressure at that point in the pregnancy.

D. Data Analyses

All statistical analyses were performed using the SPSS® statistical package, version 19.0 (SPSS Inc. Chicago, IL., USA) for Windows®. A multivariate repeated measures general linear model (GLM) was used to examine the impact

of obesity on the physiologic indicators. The GLM model consisted of a doubly multivariate GLM computation to compare the change in physiologic variables over time (three levels: 20, 28 and 36 weeks) and the mean differences between the obesity groups (2 levels: obese and over-weight). The multivariate analysis of all six physiologic indicators was computed first and was followed by analysis of each indicator separately. The dependent variables included HP, RSA, SpO₂, and blood O₂, HbA_{1c} and SBP. A two-way contingency table was conducted to evaluate whether there was a tendency for increased frequency of pregnancy complications and perinatal events to occur for obese women as compared to over-weight status groups. The odds ratio and 95% confidence interval (CI) were calculated for each event in each time frame.

V. RESULTS

A. Sample Characteristics

Table 1 displays the sample characteristics according to obesity status. The sample consisted of 41 women completing all the assessments at 20, 28 and 36 weeks gestation that were from an economically homogeneous group of pregnant participants awaiting the birth of their neonates at a low risk obstetrics clinic. Prior to this study, 10 of the women had not been previously pregnant. In regard to ethnicity, 57% of the women in the obese group were African American as compared with 40% of the women in the over-weight group, and there were more Hispanic women (40%) in the over-weight group as compared with the women in the obese group (24%). Ninety-nine percent of the women were receiving governmental Medicaid funding for health care.

TABLE 1. Maternal characteristics by obesity status at 20 +/- 2 weeks

	AGE	Obese n = 21		Over-weight n = 20		Statistical significance
		n	%	n	%	
MATERNAL (YEARS)		25.22	4.86	25.73	5.78	NS
Gravida		2.76	2.3	2.95	1.61	NS
Height (Inches)		64.35	3.42	64.70	2.83	NS
kg/m ² at 20 wks.		38.21	5.42	25.23	2.78	<.001*
kg/m ² at 28 wks.		40.09	5.52	27.04	3.15	<.001*
kg/m ² at 36 wks.		41.43	5.52	28.49	3.07	<.001*
Lbs. GWG 20-28wks.		11.85	10.78	11.33	7.69	NS
Lbs. GWG 28-36 wks.		8.35	5.57	8.85	6.02	NS
Lbs. GWG 20-36wks		19.02	10.84	19.10	9.20	NS
First pregnancy		6	24	4	20	NS
Race / Ethnicity						
Non- Hispanic White		3	14	3	15	NS
African American		12	57	8	40	NS
Hispanic		5	24	8	40	NS
Country of origin						
Russia		1	4.76	0	0	NS
India		0	0	1	5	NS
Relationship						
Single		6	29	7	35	NS
Lives with FOB		9	42	9	45	NS
Married		6	29	4	20	NS
HS graduate or GED		16	76	18	90	NS
1 - 2 years of college		8	38	12	60	NS
Full time homemaker		15	71	10	50	NS

Data presented as mean ± SD; Abbreviations: kg/m², = BMI; Lbs. GWG , pounds of gestational weight gain between weeks of 20 – 28, 28 – 36 and 20

– 36; FOB = father of baby, * Significant difference P < 0.05 by obesity status or weight gain status

B. Multivariate Tests of Within and Between Subject Effects

The within-subject main effect of time (P <.001), the between-subject effect of obesity (P = 0.002) and obesity x time interaction effect (P = .027) were significant (Table 2).

Thus, the hypotheses was supported in that there were multivariate differences in the dependent variables between the obese and over-weight women at the various time periods of 20, 28 and 36 weeks gestation and significant changes in physiologic indicators over time.

TABLE 2. Multivariate tests of within & between subjects

Multivariate Tests					
Type of Effect	Effect	df (Hypothesis, Error)	F	P	Partial η ^{2(a)}
Within-Subject Effects	Time	12, 27	6.868	.000*	.753
	Time * Obesity	12, 27	2.433	.027*	.520
Between-Subject Effects	Obesity	6, 33	4.554	.002*	.453

*Significant at 95% confidence level, P < 0.05; (a)Partial η² is identical to Pillai's trace statisti; Outcomes tested: HP = heart period; RSA = respiratory sinus arrhythmia; SpO₂= peripheral oxygenation; HbO₂ = oxygen

C. Mean Differences Between Obese and Over-weight Status

The univariate effects of obesity on the physiologic indicators were computed to determine mean differences at 20, 28, and 36 weeks between the obese and over-weight groups and are displayed in Table 3. Significant differences were identified for the physiologic indicators of HbO₂, HbA_{1c}, and SBP. At 20 and 28 weeks, HbO₂ was significantly higher (94.94 and 94.56%) for the obese women, as compared to the over-weight women (93.90 and 93.93%, respectively). However at 36 weeks, HbO₂ levels between the groups were not significantly different.

TABLE 3. Main effect of obesity with mean differences between obese and over-weight at each time period¹

Variable	Mean differences between all means				
	df (Hypothesis, Error)	F	P	η ²	
HP	1, 38	3.997	NS	.095	
RSA	1, 38	2.388	NS	.325	
SpO ₂	1, 38	.183	NS	.070	
HbO ₂	1, 38	9.431	.004*	.560	
HbA _{1c}	1, 38	5.588	.023*	.635	
SBP	1, 38	11.988	.001*	.240	
Differences between obese & over-weight status					
	Obese	Over-weight	Significance		
20 weeks					
HP	695.58	15.11	725.47	15.48	NS
RSA	5.73	.26	6.23	.27	NS
HbO ₂	94.94	.11	93.90	.11	.000*
SpO ₂	99.42	.10	99.94	.11	NS
HbA _{1c} :	4.59	.12	4.22	.13	NS
SBP	121.62	2.41	108.65	.11	.001*
28Weeks					
HP	659.95	15.11	671.86	13.23	NS

RSA	5.32	.25	6.51	.25	NS
HbO2	94.56	.17	93.93	.18	.015*
SpO2	99.11	.12	99.11	.13	NS
HbA1c:	4.49	.08	4.37	.08	NS
SBP	120.71	2.13	112.05	2.18	.007*
36 Weeks					
HP	663.85	14.40	719.15	14.76	.001*
RSA	5.36	.24	6.01	.25	NS
HbO2	94.95	.20	95.20	.21	NS
SpO2	99.04	.22	99.00	.10	NS
HbA1c:	4.57	.08	4.42	.08	.014*
SBP	120.76	1.80	116.30	1.85	NS

Data presented as mean ± SD; *Significance = P < 0.05; HP = heart period; RSA = respiratory sinus arrhythmia; HbO₂ = oxygen with hemoglobin; SpO₂ = peripheral oxygenation; SBP = systolic blood pressure; HbA_{1c}: hemoglobin A1c; there were no significant findings for gestational weight; ¹There were no significant findings for gestational weight gain controlling for obesity

At 20 and 28 weeks, SBP was significantly higher for the obese women (121.62 and 120.71 mmHg), as compared with the over-weight women (108.65 and 112.05 mmHg, respectively). However at 36 weeks, a significant difference in SBP between the obese and over-weight groups no longer existed (P = .092). At 36 weeks, HP was significantly higher for the over-weight as compared to the obese women (719.15 compared with 663.50 milliseconds, P = .001). However, HbA_{1c} was significantly higher for the obese women than for the over-weight women (4.57 compared to 4.42 g/dl, P = 0.014).

D. Main Effects of Time and its Interaction with Obesity.

Changes over time were significant (Table 4) for the physiologic indicators of HP (P = .002), RSA (P = .006), HbO₂ = .000), and HbA_{1c}; (P = .031). Between 20 and 28 weeks, the marginal means decreased significantly over time for HP (P = .001) and RSA (P = .004). Between 28 and 36 weeks, the marginal mean increased significantly for HP (= .015) and HbO₂ (P = .000). Table 6 also displays differences over time with obesity. Between 28 and 36 weeks, there were significant group differences in the pattern of change over time for the over-weight as compared to the obese groups for the physiologic indicators of HP and HbO₂. Significant increases for the over-weight as compared with the obese women included HP (47.29 compared to 3.9 milliseconds, P = .048) and HbO₂ (1.27 compared to .39%, P =.003).

TABLE 4. Univariate effects of time and its interactions with obesity: Changes over time.

Variable	df (Hypothesis, Error)	F	P	Partial η ²
HP	2, 76	6.87	.002*	.153
RSA	2, 76	5.52	.006*	.127
SpO ₂	1.4, 54.1(b)	3.47	NS	.084
HbO ₂	2, 76	15.88	.000*	.295
HbA _{1c}	1.7, 63.8(b)	.526	.031*	.094
SBP	2, 76	1.544	NS	.039

Differences over time (shown for variables with significant effects)					
Variable	Mean change	SD	F	P	η ²
Weeks 20 – 28					
HP	710.525 – 665.905	14.73	11.98	.001*	.24
RSA	5.732 – 5.426	2.26	9.62	.004*	.20
HbO ₂	94.420 – 94.245	.25	1.29	NS	.03
HbA _{1c}	4.585 – 4.494	-.10	1.25	NS	.03

Weeks 28 – 36					
HP	665.905 – 691.500	13.38	6.47	.015*	.15
RSA	5.426 – 5.281	.25	3.03	NS	.07
HbO ₂	94.245 – 95.075	-.05	32.68	.000*	.46
HbA _{1c}	4.494 – 4.570	.00	3.99	NS	.10

Changes over time with obesity					
Obese		Over weight			
Variable	Mean difference	SD	Mean difference	SD	P
Weeks 20 – 28					
HP	-35.63	.00	-53.61	2.25	NS
RSA	-.41	.01	.28	-.02	NS
HbO ₂	-.38	.06	.03	.07	NS
HbA _{1c}	-.76	-.04	.15	-.05	NS
Weeks 28 – 36					
HP	3.9	-.71	47.29	1.53	.048*
RSA	.04	.01	-.50	.00	NS
HbO ₂	.39	.03	1.27	.03	.003*
HbA _{1c}	.50	.00	.05	.03	NS

*= significance P < 0.05; HP = heart period; RSA = respiratory sinus arrhythmia; SpO₂ = peripheral oxygenation; HbO₂ = oxygen with hemoglobin; HbA_{1c}= hemoglobin A1c; SBP = systolic blood pressure

E. Odds Ratios for Perinatal Events with BMI

Table 5 displays the prevalence, odds ratios, CI, Chi Square value, and significance of the association of pregnancy complications and perinatal events with BMI status. Significant findings for the obese women include a greater likelihood of positive vaginal cultures for group beta streptococcus (+GBS). Further, obese women frequently developed gestational hypertension and gave birth by cesarean section or were assisted by vacuum or forceps. Nuchal cords occurred more frequently for the over-weight women as compared to obese women.

TABLE 5. Statistical analysis of frequency of perinatal events for the obese and overweight women.

Complication	Obese	Over-weight	Adjusted RR (95% CI)	Statistical significance
<i>Pregnancy event</i>				
Group B streptococcus	13	6	1.03 – 13.91	.032*
Gestational Hypertension	13	5	1.27 – 18.65	.017*
Diabetes	4	2	.26 – 6.88	NS
<i>Labor & birth events</i>				
Cesarean or assisted	15	6	1.52 – 22.41	.007*
Nuchal cord	5	12	1.256 – 18.42	.018*
<i>Postnatal events</i>				
Anemia	11	7	.58 – 7.17	NS
<i>Neonatal Events</i>				
LGA or Macro	6	2	.006 – 2.661	NS
< 6 pounds	4	5	0.32 – 6.266	NS

*= significance P < 0.05; LGA = large for gestational age

VI. DISCUSSION

Results of the present study, show that expected changes of pregnancy impact the vagal response. Between 20 and 28 weeks, the decrease in HP (-24.62 milliseconds) and RSA (-0.306 hertz) for both obese and over-weight women is in line with observational studies reporting a decrease in heart period and increase in heart rate with the progression of pregnancy.[3,14,35,36] However, between 28

and 36 weeks, HP and HbO₂ increased significantly (+ 47.29 milliseconds. and 1.29%) for the over-weight women. The differences between our results and those of previous studies may be explained by findings that report some restoration to the vagal response occurs in the third trimester to women with an intact adaptive response. [36] The changes accompanying the restored vagal response may lead to a relaxed vascular tone.[37,38] Relaxed vascular tone results in improved blood flow and increased oxygen availability to meet the body's needs.[39,40,41] Although the pattern of change in SBP was not significant for the over-weight women, the pattern of a gradual rise, returning to or exceeding early pregnancy levels at 36 weeks, mirrors the reported findings of Moertl and colleagues. [22] The changes seen for over-weight women may be explained as the ability to flexibly respond to additional demands of pregnancy. [22, 37]

In comparison, a significant pattern of change in the physiologic indicators was not identified for the obese women. Although HbA_{1c} levels were higher at 36 weeks for the obese women, there was little change over time in vagal response, oxygenation, or systolic blood pressure. That is, the effect of obesity seemed to repress changes that are expected in pregnancy. In obese women the expected early events of vasodilation and increased plasma volume may fail to occur, resulting in failure of the blood pressure to decrease.[42] Researchers have reported that adipose tissue with constricted vasculature leads to decreased peripheral oxygenation, hypoxia, increased blood pressure, and inflammation.[24] One explanation of the repressed changes during pregnancy may be that obese women may have decreased vagal regulation of the heart with a subsequent increase in sympathetic mobilization behaviors. [4] Thus, obese women may have a decreased capacity to respond to challenging stimuli, including the physiologic changes of pregnancy and may lack self-regulatory capacity to adjust to stressful stimuli.[13,37] In contrast, HbO₂ was higher for the obese women as compared to the over-weight women at 20 and 28 weeks. We do not have a ready answer for why HbO₂ was higher for the obese group. One thought is that the obese women were compensating for physiologic changes, and these compensatory mechanisms were enough to maintain oxygen levels and the physiologic state. However, with time, a decline in the ability to flexibly respond to additional pregnancy demands may have resulted in a diminished adaptive response. An inadequate adaptive capacity may result in pregnancy complications such as hypertension or adverse birth outcomes. Further research needs to be conducted to more precisely understand how the change in one physiologic indicator impacts another indicator for women with various BMI indices.

The results of previous research reports have shown significant independent associations of higher maternal glucose concentrations, [43, 44] and obesity [7,8,9, 10, 11, 45] with adverse perinatal outcomes. An explanation of why obese women are more likely than over-weight women to have gestational hypertension is that an increase in peripheral vascular resistance resulting from increased sympathetic activity would lead to a decreased vagal response, faster heart rate, and increased blood pressure. [24]

Further investigation is needed to determine if this is true with over-weight pregnant women as well. This study adds to previous reports by examining the impact of obesity and gestational weight gain on adverse perinatal outcomes. At 36 weeks, obese women had significantly higher HbA_{1c} and increased frequency of positive cultures for Group B streptococcus, hypertension, and cesarean or assisted birth by vacuum or forceps as compared with the over-weight women.

A. *Clinical Implications*

Increased knowledge of the autonomic nervous system response, HbA_{1c}, and oxygenation might facilitate the recognition of a maladaptive response pattern and promote an early intervention toward increasing positive maternal and neonatal outcomes. Interventions that have demonstrated an increased heart rate variability in pregnant populations have included early recognition and dietary intervention to promote euglycemia.[4,46] In addition, for non-pregnant individuals, interventions that have increased HRV include relaxation techniques such as deep breathing exercises,[47,48] safe leisure time physical activities with others, harmonic vibrations of crystal singing bowls,[49] listening to the Native American flute, [50] tai chi exercise, [51] or grounding or earthing.[52] Further, simple back or foot massage promotes relaxation and decreases stress. [53] As families seek things to do for their pregnant family member, performing back or foot massages may result in a positive experience. Further study is needed to evaluate interventions that increase HRV in pregnant women.

To address problems of obesity, a sustained approach across the lifespan may be required, rather than a reliance upon targeted interventions during pregnancy. The amount of weight gained during pregnancy can affect the immediate and future health of a woman and her infant. It is important to discuss appropriate weight gain, diet, and exercise at the initial visit and plan with the patient the amount of weight she should gain. Individualized care and clinical judgment are necessary in the management of the overweight or obese woman who is gaining (or wishes to gain) less weight than recommended but has an appropriately growing fetus.⁵⁷ Weight gain guidelines need to balance the benefits of improving fetal nutrition with the risk of harm to the mother and infant, while identifying those at increased risk for adverse outcomes. New approaches to the education of pregnant women and care providers are needed to implement guidelines for gestational weight gain. Practices addressing obesity prevention, treatment and optimal weight loss and maintenance must be identified to provide nurses with an array of strategies to help curb the rising epidemic of obesity among women of child bearing age.

B. *Strengths and Weaknesses*

A main strength was the study provided data that enriched the knowledge of the association of obesity and excess gestational weight gain (GWG) on vagal response, oxygenation, HbA_{1c} and SBP simultaneously over time. A main limitation to the study was that the sample size was relatively small (n=41), However, the groups were balanced in confounding variables of age, family structure, level of education, being a full time homemaker, and first pregnancy.

Further, the sample was homogeneous in socioeconomic status: 99% of the study participants were medicaid recipients. The potential participation pool was limited as many women were not regular in attendance at the clinic where subjects were recruited. Of the 61 women recruited, only 41 were present at the 20, 28, and 36 weeks' assessment times. In addition, the women in the study were not representative of the population at large as the participants were predominately of African-American and Hispanic heritage, and the sample excluded women who had complications leading to attrition before 36 weeks. Although those that remained in the study were similar to those that dropped out, selection bias is a possible threat to internal validity. The small sample size limits the ability to generalize beyond the groups studied.

C. Conclusion

In conclusion, this present study demonstrated that there are differences between obese and over-weight women in adaptation to pregnancy that impact physiologic indicators and perinatal outcomes. The effects of maternal stress on pregnancy outcomes may be mediated through biological and behavioral mechanisms. The sympathetic nervous system may cause exaggerated heart rate and blood pressure responses when encountering behavioral stimuli experienced as engaging, challenging, or aversive. By identify alterations in autonomic response using minimally invasive measures, nurses can intervene well before problems manifest themselves to make a difference in perinatal outcomes. Nursing practice can be enhanced when the nurse collaborates with the patient to formulate a plan that incorporates both evidence-based data and patient preferences that are individually tailored to meet the needs of each pregnant woman. Nurses can make a difference!

REFERENCES

- [1] R. Amend, R., & A. Golden, "Evidence-based practice at the point Of care." JNP, Vol. 7(4), 2011, pp.303-308.
- [2] C.L.Ogden, M.D. Carroll, B.K. Kit, & K.M. Flegal; "Prevalence of obesity in the United States,"NCHS data brief, no 82. Hyattsville, MD: National Center for Health Statistics. 201, 2009-2010.
- [3] M.H.Davenport, C.D. Steinback, & M.F. Mottola, M.F. "respiratory responses during weight-bearing exercise." Respiratory Physiology & Neurobiology, vol. 16, 2009, pp 341-34.
- [4] N. Amador-Licona, J.M. Guizar-Mendoza, M. Juarez, B. Linares-Segovia. Sympathetic activity and pulmonary function in obese pregnant women. Acta Obstet Gynecol Scand, vol. 88 (3), 2009, pp.314-9.
- [5] Y. Bacci, I. Ustuner, H.L. Eskin, R. Ersoy, & A.F. Avsar, "Effect of maternal obesity and weight gain on gestational diabetes mellitus. Gynecol Endocrinol, vol. 29(2), 2013, pp.133-6. doi: 10.3109/09513590.2012.730571.
- [6] S. Riskin-Mashiah, A. Damti, G. Younes, & R. Auslander,"Pregestational body mass index, weight gain during pregnancy and maternal hyperglycemia." Gynecological Endocrinology vol. 27(7), 2011, pp.464-467
- [7] S. Bhattacharya, D.M. Campbell, W.A. Liston & S. Bhattacharya S. "Effect of Body Mass Index on pregnancy outcomes in nulliparous women delivering singleton babies." BMC Public Health, vol. 7:168, 2007. <http://www.biomedcentral.com/1471-2458/7/168>
- [8] J.M. Crane, J. White, P. Murphy, L. Burrage, D. Hutchens, "The effect of gestational weight gain by Body mass index on maternal and neonatal outcomes." J Obstet Gynaecol Can, vol. 31(1), 2009, pp.28-35.
- [9] M.A. Kominiarek, P. VanVeldhusien, J. Hibbard, J., et al., "The maternal body mass index: a strong association with delivery route." American Journal of Obstetrics & Gynecology, vol.203(3), 2010, pp.264.e1-7. doi:10.1016/j.ajog.2010.06.024.
- [10] D.B.Ehrenthal, X. Jiang, & D.M.Strobino, D.M. "Labor induction and the risk of a cesarean delivery among nulliparous women at term." Obstetrics & Gynecology, vol. 116, 2010, pp. 35-42
- [11] S.D. McDonald, Z. Han, S. Mulla, & J. Beyene, "Overweight and obesity in mothers and risk of preterm birth and low birth weight infants: systematic review and meta-analyses." BMJ, Jul 20; vol. 341, 2010, ppc3428. doi: 136/bmj.c3428
- [12] Vanek, A.I. The conductor of the autonomic orchestra. Front Endocrinol (Lausanne). 2012; 3:71: 71-84.
- [13] S. W. Porges, "The polyvagal perspective. Biological Psychology, vol. 74, 2007, pp.116-143.
- [14] Kuo, C.D., Chen, G.Y., Yang, M.J., Lo, H.M., & Tsai, Y.S. Biphasic changes in autonomic nervous activity during pregnancy. British Journal of Anesthesia, 2000; 84(3), 323-329.
- [15] M. Berkelaar, E.M. Eekoff, A.M. Simonis-Bik, D.J. D.I. Boomsma, M. Diamant, "Effects of induced hyperinsulinaemia with and without hyperglycaemia on measures of cardiac vagal control." Diabetologia, vol. 56, 2013, pp. 1436-1444.
- [16] VandeVusse, L., Hanson, L., Berner, M.A., & White Winters, J.M.Impact of self-hypnosis in women on select physiologic and psychological parameters. J Obstet Gynecol Neonatal Nurs. 2010; 39(2):159-68.
- [17] Telles, S., Raghavendra, B.R., Manjunath, N.K., Kumar, S., Subramanya, P. Changes in autonomic variables following two meditative states described in yoga texts. J Altern Complement Med. 2013; 19(1):35-42.
- [18] Catalano, P.M., McIntyre, D., Cruickshank, J.K., McCance, D.R.,Dyer, D.R., Metzger, B.E., Lowe, L.P. et al. The hyperglycemia and adverse pregnancy outcomes study: association of GDM and obesity with pregnancy outcomes. Diabetes Care. 2012; 35(4):780-787.
- [19] J.L. Mills, L. Jovanovic, R. Knopp, J. Aarons, M. Conley, E. Park, et al. "Physiological reduction in fasting plasma glucose concentration in the first trimester of expected pregnancy: The diabetes in early pregnancy study." Metabolism, vol. 47(9), 1998, pp.1140- 1144.
- [20] R.J. Kaaja, & M.K. Pöyhönen-Alho, M.K. "Insulin resistance and sympathetic over-activity in women." Journal of Hypertension, vol.24(1), 2006, pp.131-141. J.S.
- [21] S.S. Jarvis, S. Shibata, T.B. Bivens, Y. Okada, B.M. Casey, et al. "Sympathetic activation during early pregnancy in humans." The Journal of Physiology, vol. 590(15):2012, pp.3533-3543
- [22] M.G. Moertl, H.K. Lackner, I. Papousek, A. Roessler, H. Hinghofer- Szalksy, et al. "Phase synchronization of hemodynamic variables at rest and after deep breathing measured during pregnancy." PLoS One 2013; vol. 8(4): e60675
- [23] J.E. Ramsey, W.R. Ferrell, L. Crawford, A.M. Wallace, A. M., Greer, I.A., Sattar, N. Maternal obesity is associated with dysregulation ometabolic, vascular, and inflammatory pathways. Journal of ClinicalEndocrinology & Metabolism, vol. 87(9): 2002, pp.4231-4237
- [24] R. Gaillard, E.A.P. Steegers, A. Hofman, V.W.V. Jaddoe, "Association of maternal obesity with blood pressure and the risks of gestational hypertensive disorders. The Generation R Study," Journal of Hypertension, vol. 29(5): 2011, 937-44.
- [25] Cohen, J. (1988). Statistical power analysis for the behavioral sciences (2nd ed.). Hillsdale, NJ: Lawrence Earlbaum Associates.

- [26] S.W. Porges, "Vagal tone: a physiologic marker of stress vulnerability," *Pediatrics*, vol. 90 (3Pt 2), 1992, pp.498-504.
- [27] Ben-Tal, A., Shamailov, S.S., & Paton, J.F.R. (2012). Evaluating the physiological significance of respiratory sinus arrhythmia: looking beyond ventilation-perfusion efficiency. *The Journal of Physiology*. 2012; 590:1989-2008.
- [28] E.A. Byrne, & S.W. Porges, "Data-dependent filter characteristics of peak-valley respiratory sinus arrhythmia estimation: A cautionary note." *Psychophysiology*; vol. 30: 1993, pp.397-404.
- [29] L.A. Jensen, J.E. Onyskiw, & N.G. Prasad, (1998). "Meta-analysis of zounarterial oxygen saturation monitoring by pulse oximetry in adults." *Heart & Lung*, vol. 27(6), 1998, pp. 387-408.
- [30] S-Y.P.K. Shiao, "Desaturation events in neonates during mechanical ventilation." *Critical Care Nursing Quarterly*, vol. 24(4), 2002, pp.14-29.
- [31] Radiometer Medical Ap S., Denmark. "Quality Control Service Manual for OSM 3 hemoximeter", 2006. Retrieved Oct.31, 2014 www.radiometeramerica.com/.../manuals/.../quality-control-system-manu
- [32] C.N. Ou, & C.L. Rognerud, "Rapid analysis of hemoglobin variants by cation-exchange HPLC." *Clinical Chemistry*, vol. 39(5), 1993, pp.820-824.
- [33] Omron Health Care. Service manual for Omron products: (model: Omron 711c, HRM, USA Inc.), 2001. Retrieved Oct.31, 2014 www.heartratemonitorsusa.com/omron-healthcare.html
- [34] American Heart Association. Understanding blood pressure readings.2011. <http://www.heart.org>, Retrieved Oct.31, 2014
- [35] J.A. DiPietro, K.A., Costigan, & E.D. Gurewitsch, "Maternal psychophysiological change during the second half of gestation." *Biopsychol* 2005; vol 69(1), 205, pp. 23-28.
- [36] J.C. Ouzounian, & U. Elkavam, "Physiologic changes during during normal pregnancy and delivery." *Cardiology Clinics*, IV. Vol. 30(3): 2012, pp.317-29.
- [37] R.J. Helmreich, V. Hundley, & P.Varvel, "The effects of obesity on heart rate (heart period) and physiologic parameters during pregnancy." *Biological Research for Nursing*, vol. 10(1),2008, pp.63-78.
- [38] W. Gazenvoort, G.J. Bonsel, J.I. deVries, & H. Wolf, "Plasma volume and blood pressure regulation in hypertensive pregnancy" *Journal of Hypertension*, vol. 22(7), 2004, pp.12235-1242.
- [39] M.T. Gladwin, "Role of red blood cell in nitric oxide homeostasis and hypoxic vasodilatation," *Advances in Experimental Medicine and Biology*, vol. 588, 2006, pp.189-205.
- [40] T.S. Isbell, C.W. Sun, L.C. Wu, X. Teng, D.A. Vitturi, et al. "SNO-hemoglobin is not essential for red blood cell-dependent hypoxic vasodilatation." *Nature Medicine*, vol. 14(7), 2008, pp:773-777.
- [41] Singel, D.J., & Staminer, J.S. Chemical physiology of blood flow regulation by red blood cells: the role of nitric oxide and S-nitrohemoglobin. *Annual Review of Physiology* 2005; vol. 67, 99-145
- [42] St Louis, J., & Brochu, M., (2008). The cardiovascular paradox of pregnancy. *Medical Science*, 2008; ol.23(11): pages 944-949.
- [43] Metzger, B.E., Lowe, L.P., Dyer, A.R., et al.: HAPO Study Cooperative Research Group. Hyperglycemia and adverse pregnancy outcomes. *N England J of Medicine* 2008; vol. 358:1991-2002.
- [44] HAPO Study Cooperative Research Group. (2010A). Hyperglycemia and adverse pregnancy outcomes (HAPO) study: preeclampsia. *American Journal of Obstetrics & Gynecology*, 2010A; vol. 202: 225, e1-e7.
- [45] HAPO Study Cooperative Research Group. Hyperglycemia and adverse pregnancy outcomes (HAPO) study: associations with maternal body mass index. *BJOG* 2010B, vol. 117: 575-584.
- [46] Kahn, S.E., Hull, R.L., & Utzschneider, K.M. Review Article Mechanisms linking obesity to insulin resistance and type 2 diabetes. *Nature* 2006, vol. 444: 840-846.
- [47] Klinkenberg, A.V., Nater, U.M., Nierop, A., Bratsikas, A., Zimmermann, R. and Ehlert, U. Heart rate variability changes in pregnant and non-pregnant women during standardized psychosocial stress. *Acta Obstetrica et Gynecologica Scandinavica*, 2009, vol. 88(1), 77-82.
- [48] Sutarto, A.P., Wahab, M.N., & Zin, N.M. Resonant breathing biofeedback training for stress reduction among manufacturing operators". *Int J Occup Saf Ergon*, 2012, vol. 18 (4): 549-61.
- [49] Wepner F, Hahne J, Teichmann A, Berka-Schmid G, Hordinger A, et al. Treatment with crystal singing bowls for chronic, spinal pain and chronobiologic activities-A randomized controlled trial. *FORSCHENDE KOMPLEMENTARMEDIZIN*. 2008; vol. 15(3):130-137
- [50] Miller, E.B., & Goss, C.F. "An Exploration of Physiological Responses to the Native American Flute" January 2014, arXiv: vol. 1401.6004. Retrieved 25 Jan 2014.
- [51] Chang, R-Y., Koo, M., Yu, Z-R., Kan, C-B, Chu, I-T., Hsu, C-T., and Chen, C-Y. Effect of *T'ai Chi* Exercise on Autonomic Nervous Function of Patients with Coronary Artery Disease. *The Journal of Alternative and Complementary Medicine*, November 2008, vol. 14(9): 1107-1113. doi:10.1089/acm.2008.0166.
- [52] Chevalier, G., & Sinatra, S.T. Emotional Stress, Heart Rate Variability, Grounding, and Improved Autonomic Tone: Clinical Applications Integrative Medicine • Jun/Jul 2011, vol. 10(3): 102-110.
- [53] Field, T., Figueiredo, B., Hernandez-Reif, M., Diego, M., Deeds, O., Angela Ascencio, A. Massage therapy reduces pain in pregnant women, alleviates prenatal depression in both parents and improves their relationships. *Journal of Body Works and Movement Therapy*; vol. 12(2): 146-150.

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