Mid Trimester Fall in Blood Pressure: Fact or Myth

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Abstract

It has been described in the late 1960s that there is a drop in blood pressure, especially Diastolic blood pressure in the second trimester at around 18 weeks. The blood pressure then subsequently rises to the pre pregnancy levels during the third trimester. The so called secondary wave of trophoblastic invasion, which produces deep modifications of the spiral arteries, and makes them insensitive to vasoconstrictors. However, recent studies by various authors show that there is no mid trimester drop; but rather, an increase as compared to the first trimester blood pressure. Materials and Methods: We conducted a prospective study in the antenatal department of JSS Medical College & Hospital, Mysuru; over a period of one year, from July 2016 to June 2017. The blood pressures of 120 women were recorded at four different of gestation compared. Results: The systolic pressures showed significant but steady increase up Corresponding Author: to 32 weeks and then a significant rise when compared to the first trimester. The diastolic blood pressure and mean arterial pressure showed a non significant decrease from baseline at 16 - 20 weeks. This was followed by a rise in both diastolic pressures and mean arterial pressure as gestation advanced. Conclusion: Our study disproves the age old concept that

pressure. It is the first of its kind in the Indian population.

Keywords: Systolic Blood Pressure; Diastolic Blood Pressure; Mean Arterial Pressure; Pregnancy.

there is a mid trimester fall in blood

Introduction

Normal pregnancy is associated with various physiologic and hemodynamic changes like increased blood volume, heart rate, cardiac output and decreased peripheral vascular resistance, which are secondary to peripheral vasodilatation [1]. conventional teaching is that systemic arterial blood pressure falls gradually in the second trimester of pregnancy [2]. The maximum fall occurs around 18-22 weeks, and then it rises again from 24-28 weeks. The blood pressure reaches pre pregnancy levels by 36 weeksof gestation. The hypothesis proposed to explain this mid trimester fall of blood pressure is that the trophoblast migrates deep into the median layer of the spiral arteries, making them insensitive to vasoconstrictors [3].

However, various studies have shown that mean arterial pressure, peak-systolic and minimum diastolic aortic pressures remain unchanged throughout pregnancy. We conducted a prospective study in the antenatal out patient department of JSS Hospital, Mysuru, which is a tertiary care teaching hospital. The aims of this study were: (i) To document whether a fall in blood pressure occurs in the second trimester; and (ii) To determine whether the absence of mid trimester fall in BP is associated with a higher risk of pre eclampsia.

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Received on 10.03.2018, **Accepted on** 02.04.2018

Materials and Methods

A prospective study was conducted in Department of Obstetrics and Gynaecology at JSS Hospital, Mysuru. The patients were recruited over a period of one year from July 2016 to June 2017. 129 women who were between the age of 18 – 35 years, and came to the antenatal clinic from the first trimester onwards were included in the study. Women withage <18 or >35 years, anaemia (Hb<10gm%), obesity (BMI >30), multiple pregnancy, molar pregnancy, spontaneous abortion, chronic hypertension; primary or secondary, renal, cardiac and connective tissue disorders, pre existing diabetes mellitus and hyperthyroidism were excluded from the study.

Method of Data Collection

A team comprising of a consultant obstetrician and a resident in Obstetrics & Gynaecology were trained to record the blood pressure in the correct way as described below. The BP was measured according to a strict protocol using the Diamond Mercury sphygmomanometer. The woman was asked to be seated in a quiet room with her right arm supported and the correct-sized cuff sited at the level of the heart. The first and fifth Korotkoff sounds were taken for systolic and diastolic BP respectively and a total of three readingswere recorded at intervals of five to ten minutes. The recorded blood pressure was the average of the three readings. The frequency of monitoring was as follows:

i: At 8 -14 weeks

ii: At 16 - 20 weeks

Table 1: Mean Blood Pressure Recordings

	Visit 1 (8-14 weeks)	Visit 2 (16- 20 weeks)	Visit 3 (22 – 28 weeks)	Visit 4 (32 - 36 weeks)	95% confidence interval for change from baseline	Trend in blood pressuresand trend in changes from baseline (P-value)
Mean gestational	11.5 weeks	18 weeks	25 weeks	34 weeks	-	-
age MEAN SBP	111.14	111.03			.109	1.000
WILAN SDI	-	-	113.49	-	-2.347	.099
-	-	_	-	115.19	-4.050*	.000
MEAN DBP	70.89	70.35	-	-	.535	1.000
-	-	-	71.75	-	868	1.000
-	-	_	-	71.81	923	1.000
MAP	84.305	83.911	-	-	.393	1.000
-	-	_	88.326	-	-4.022*	.012
-	-	_	-	89.588	-5.283*	.003

iii: At 22 - 26 weeks

iv: At 28 -36 weeks

These measurements were part of the regular antenatal monitoring of the patient.

Results

Out of the 129 women who were recruited, 9 were excluded from the final data analysis due to various reasons like miscarriage, fetal anomalies, or they were lost to follow up.

Statistical Methods

The summary statistics was done using mean, standard deviation and 95% CI of mean. The inferential statistics is done by using repeated measure ANOVA with bonfferoni post hoc test. All the measurements were done using SPSS version 21.0.

The average age of the women was 26.5 years (range 18 - 35 years). 50% of women were primigavida. 27% were second gravidas, and the remaining had higher order pregnancy. Tables 1 to 3 show the changes in systolic, diastolic and mean arterial pressures from baseline (8–14 weeks). In paired comparisons of SBP, there was a non significant increase from baseline to 16-20 weeks gestation (mean change 0.109 mmHg; 95% CI -2.5 to 2.7) p value 1.00, 22–28 weeks (mean change 2.34mmHg; 95% CI -4.9 to 0.24) p value 0.099. There was a significant rise in systolic BP at the fourth visit; ie at 32-36 weeks, when compared to the baseline (mean change 4.05 mmHg; 95% CI -6.5 to -1.5), with p value of 0.00 (Table 1).

Table 2: Change in Systolic blood pressure

SBP	Mean	SE	95% Confidence Interval	
			Lower Bound	Upper Bound
8-14 WKS	111.14	.859	109.441	112.844
16-20 WKS	111.03	.686	109.675	112.391
22-26 WKS	113.49	.667	112.169	114.810
28-36 WKS	115.19	.628	113.949	116.436

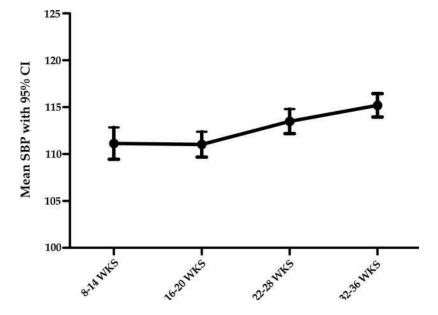


Fig. 1: Change in Systolic Blood Pressure

On comparing the Diastolic Blood Pressure, there was a non significant decrease from baseline to 16-20 weeks (mean change 0.53 mmHg; 95% CI - 1.3 to 2.45), p value 1.00. A nonsignificant increase was seen at 22 – 28 weeks (mean change 0.86 mmHg; 95% CI - 2.8 to 1.16), p value 1.00; and at 32-36 weeks (mean change 0.78 mmHg; 95% CI - 3.04 to 1.19), p value 1.00. Thus, it was demonstrated that the characteristic fall in diastolic blood pressure in the second trimester; was not seen in our patients. None of these women developed pre eclampsia, which has been linked to the absence of mid trimester fall in blood pressure (Table 2 and Figure 1).

The mean arterial pressure showed a non significant decrease from 84.3 mmHg at baseline to 83.9 mm Hg at 16–20 weeks (p value 1.00). However, there was a non significant increase seen at 22 – 28 weeks (mean change 4.02 mm Hg; p value

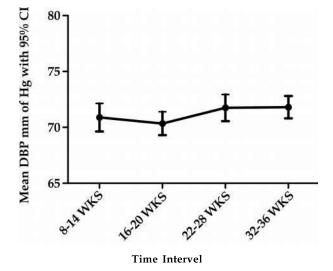


Fig. 2: Change in Diastolic Blood Pressure:

Table 3: Change in Diastolic blood pressure

DBP	Mean	Std. Error	95% confidence interval	
			Lower Bound	Upper Bound
8-14 WKS	70.886	.634	69.631	72.141
16-20 WKS	70.351	.525	69.312	71.389
22-28 WKS	71.754	.605	70.557	72.952
32-36 WKS	71.809	.505	70.809	72.809

0.012); and a significant rise in MAP seen at 32–36 weeks (mean change 5.2 mm Hg, p value 0.003). Thus, there was no significant mid-trimester drop observed in mean arterial pressure. (Table 3 & 4, Figure 2 & 3).

Table 4: Change in Mean Arterial Pressure

MAP	Mean	Std. Error	95% Confidence Interval Lower Bound Upper Bound	
1	84.305	.635	83.047	85.562
2	83.911	.511	82.899	84.924
3	88.326	1.221	85.910	90.743
4	89.588	1.395	86.826	92.350

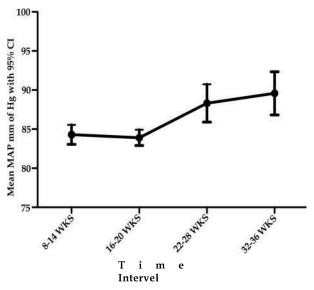


Fig. 3: Change in Mean Arterial Pressure

Discussion

The principal finding of our study is that there is no statistically significant fall in either systolic or diastolic blood pressure in pregnancy. There is general agreement that there is a fall in diastolic blood pressure during the middle trimester. Grindheim et al prospectively studied the blood pressure patterns in 63 Norwegian women [4]. They concluded that there was a statistically significant drop in SBP and DBP up to mid- pregnancy (22-24 weeks), followed by a progressive increase until delivery,

When the average of MAP readings taken during the fifth and sixth months of pregnancy is 90 mm. Hg or more, there is a significant increase in the stillbirth rate, in the frequency of intrauterine growth restriction, and in the frequency of proteinuria, hypertension, and diagnosed pre-eclampsia in the third trimester. This concept has been reinforced during the years by other studies. First, the blood

supply to the uterus is enhanced and a maternal circulation to the placenta is established, effectively diverting blood away from the lower limbs. Second, several haemodynamic adjustments in the mother's circulation occur; her blood volume and cardiac output increase quickly by over one third, yet her blood pressure falls [5]. This apparent paradox is reconciled by a profound reduction in systemic vascular resistance until mid-gestation, along with a reduction in blood viscositydue largely to haemodilution. These adjustments are synergistic in promoting an effective uteroplacental blood supply. This formed the explanation for the apparent mid trimester blood pressure drop that is observed in normal pregnancy. This drop in DBP in the second half of pregnancy became the basis for mid-trimester drop and many thought it could form the basis for screening tests for preeclampsia [6]. However, not all studies have confirmed a mid-trimester drop in SBP and/or DBP. A longitudinal study of 189 women by Rebelo et al. [7] showed that there was a non significant fall in blood pressure during the second trimester followed by a rise again in the third trimester and in the post partum period.

Volman et al. [8] using the thoracic impedance technique and Korotkoff V sounds to denote DBP, showed an increase in heart rate, a decrease in stroke volume, a stable cardiac output and an increase in SBP and DBP in the second trimester.

Silva et al. [9] demonstrated the absence of a midpregnancy fall in DBP in women with a low educational level and an increase in SBP in women of different educational subgroups. This observation of lack of BP fall was attributed to few measurement points being obtained during pregnancy or to some other confounding factors such as circadian and daily variations in BP [10].

Nama et al. [11] studied 255 primigravid, white, normotensive women and found that SBP progressively and significantly increased throughout pregnancy from 11 weeks gestation. After a very slight and nonsignificant dip at 22 weeks gestation, the DBP significantly increased throughout pregnancy. These findings are similar to our study, which also shows that there is no fall in either systolic or diastolic pressures during the second trimester. However, these studies, including our study have their limitations. The question which remains to be answered is that whether more frequent BP recordings could have had a different outcome on the results?

Thus the old school of thought of a significant drop in blood pressure during the second trimester and the absence of this fall being co related to the development of pre eclampsia no longer holds good. Although ours is a small study, it has set the base for further research into the conventional concepts of hemodynamic changes in pregnancy, and their effect on the outcome of pregnancy.

Some issues will have to be addressed once again. How do we explain and interpret the trophoblastic invasion of the spiral arteries, and what are its effects on systemic circulation? Is an increased mid-trimester BP or rather the absence of fall in BP, still to be considered ominous and the patient considered as having high risk for developing a poor obstetric outcome? Or, should new trends be described to define maternal and fetal risks?

Acknowledgement

This study is part of a project funded by the Cardiology Society of India, Karnataka Chapter.

Conflict of Interest

The authors declare that there is no conflict of interest.

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