



## A STUDY OF CARDIAC STATUS BY 2D-ECHOCARDIOGRAPHY IN THE PERIPARTUM PERIOD IN A RURAL SETUP.

### General Medicine

**Dr Amandeep Singh Kaloti\***

Professor and Head, Dept of Medicine, KCGMC, Karnal. \*Corresponding Author

**Dr Sunil Kumar**

Professor and Head, Dept of Medicine, JNMC, Wardha.

**Dr A H Inamdar**

Professor, Dept of Medicine, JNMC, Wardha.

**Dr Shourya Acharya**

Professor, Dept of Medicine, JNMC, Wardha.

### ABSTRACT

**Aim:-** To study the cardiac status in the peripartum period by 2D-echocardiography by studying the systolic and diastolic left ventricular function and also clinical examination.

**Materials and methods:-** 50 females in the peripartum period in the reproductive age group were selected randomly. A control group of 50 healthy non-pregnant females of similar age group were included in the study. Detailed history and clinical examination was done as per protocol. Routine investigations and echocardiography was done.

**Results:-** The two groups were comparable in respect to age and height. The comparison of weight and BMI was found to be statistically significant. The blood pressure in the two groups were found to have a mean blood pressure of  $114 \pm 2.58$  mm Hg and  $113.4 \pm 7.17$  mmHg ( $Z=1.80$ ,  $p>0.05$ ) respectively in the case and control groups. This was statistically insignificant. The mean arterial pressure showed a mean of  $86.7 \pm 3.56$  and  $85.5 \pm 6.06$  ( $Z=5.73$ ,  $p<0.05$ ) in the case and control groups, which was statistically significant. The systolic function as a whole did not show statistically significant difference between the case and control groups except a slight increase in LA size. The diastolic function parameters showed a statistically significant difference in the case and control groups.

**Conclusion:-** The study showed that body weight and BMI increase in the peripartum period. There is no systolic dysfunction in the peripartum period, though LA size increases. There is no diastolic dysfunction. E and A wave velocities increases but the ratio E/A is normal. IVRT and DT decreases in the peripartum period.

### KEYWORDS

#### INTRODUCTION

Peripartum period is a period extending from one month prior to delivery to five months post partum.

Pregnancy causes dramatic usually reversible changes in a woman's cardiovascular system. Maternal heart disease (present in 2% of pregnancies) (Mc Anutly, J.H. et al, 1990) is a most important nonobstetric cause of deaths in pregnant women. With noninvasive techniques, the patterns of maternal cardiovascular changes can be determined throughout pregnancy. The literature includes studies in which echocardiography was used to evaluate LV systolic function and cardiac haemodynamics in pregnant females (Duvekott, J.J. et al, 1994; Katz, R. et al. 1978; Mabie, W.C. et al, 1994; Robson, S.C. et al, 1987). Little information is available about the physiological changes in LV diastolic function that occur during pregnancy (Mabie, W.C. et al., 1994). Only within the last decade have clinicians and researchers discovered that abnormalities of LV diastolic function are important contributors to the symptoms of a variety of cardiac disorders, including those involving normal or near normal systolic function (Labovitz, A.J. et al, 1987). Therefore, a through study of LV systolic and diastolic function in normal pregnancy would be useful, not only to assess maternal health in pregnant women with cardiovascular disease but also to predict and possibly treat complicated pregnancies.

LV diastolic function can be assessed by recording the velocity of flow through the mitral valve. The mitral inflow profiles are affected by a complex interaction of many factors, including myocardial relaxation, ventricular compliance, pericardial restraint, preload and afterload, and myocardial contractility (Nishimura, R.A. et al, 1978). Pregnancy causes an increase in the preload (Katz, R. et al, 1978) and an increase in the LV wall thickness and mass (Hunter, S. et al, 1992).

One of the disease affecting the women in peripartum period is peripartum cardiomyopathy. It is characterized on ECHO by LV systolic dysfunction demonstrated by depressed fractional shortening or ejection fraction. It develops in last period of pregnancy or in puerperium.

Major adaptations occur in the maternal cardiovascular system during the normal pregnancy and knowledge of these changes is essential for the management of women with cardiovascular disease.

Therefore, we studied the changes in LV function that occurs in

peripartum period. To test this hypothesis we used diastolic mitral inflow profile to prospectively evaluate LV diastolic function and also evaluate the LV systolic function in healthy pregnant women by echocardiography.

#### Review of literature:-

The utopian objective of obstetrics is that, every pregnancy should culminate in a healthy mother in possession of a healthy baby. Quality of life for both mother and her new born rightfully has become the most important concern, because every child has a birthright to be born undamaged mentally and physically.

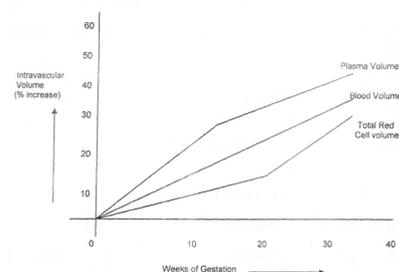
Pregnancy is a normal physiological event which is sometimes complicated by pathological processes, dangerous to the health of mother and foetus. There are some significant alterations in circulatory physiology during pregnancy. The knowledge of these is a prerequisite in understanding the diagnosis and management of pregnancy related complications.

The main homodynamic changes are seen in relation to:

1. Blood volume
2. Cardiac output, and
3. Peripheral resistance

These are probably mediated by oestrogens, prostaglandins and prostacyclins.

#### 1. Blood volume



**Figure 1 : Schematic representations of the percent increase in the intravascular volumes that occur during pregnancy.**

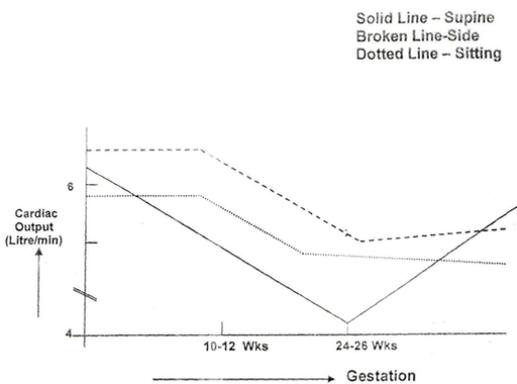
Plasma volume – increases by approximately 50%.  
 Total red cell volume – increases by approximately 25%.  
 Thus causing a blood volume increase by 40%.

It is well known that there is an increase in blood volume during pregnancy. The maternal blood volume starts increasing during first trimester and expands more rapidly during the second trimester. Then it rises at a much slower rate during the third trimester, when it is about 35% greater than the nonpregnant volume. Both the plasma volume and red cell mass are raised, the plasma volume being increased by about 50% and the increase in the total red cell volume by approximately 25% causing a 40% increase in total blood volume.

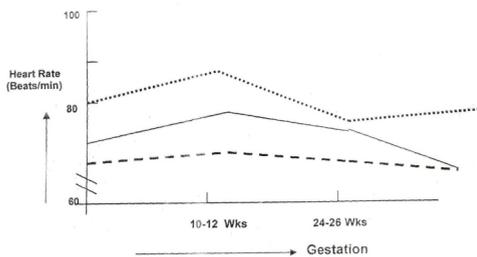
Pritchard, J.A. in 1965 and Ueland, K. in 1976 found that blood increased by about 40 to 50% during pregnancy. This is similar to that described by Dahlstrom, H. et al in 1988.

**2. CARDIAC OUTPUT**

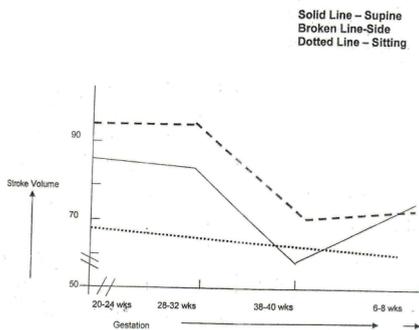
Walters, W.A.W. et al in 1966 demonstrated that there was a marked increase in cardiac output by the 12<sup>th</sup> week of pregnancy. Lees, M.M. et al in 1967 and Metcalfe, J. et al in 1974 in different studies made similar claims.



**Figure 2: Graph showing changes in Cardiac Output in relation to duration of pregnancy.**



**Figure 3: Graph showing changes in the Heart Rate in normal pregnancy in relation to duration of pregnancy.**



**Figure 4: Effect of time in gestation and of the maternal position on cardiovascular system of the mother. Data were obtained at three periods during gestation and once post-partum.**

It was mentioned that increase in cardiac output which is achieved mainly by increase in stroke volume rather than heart rate, reaches its

maximum of 30 to 40% above normal by mid term. Then it declines during the third trimester because in the supine position, venous return to the heart is obstructed by the gravid uterus which actually reduces the cardiac output by 20% or more (Kerr, M.G. et al, 1965).

Ikard R.W. et al (1971) found that cardiac output in late pregnancy is appreciably higher when the women is in the lateral recumbent position than when she is supine. As in the supine position the large uterus and its contents often impede the venous return to the heart. Ueland, K. (1976) found an increase in cardiac output (C.O.) by 1,100 ml (i.e. 22%) when the pregnant women was moved from her back onto her side. Easterling T.R. et al (1988) found that when pregnant women assumes the standing position after sitting, C.O. in her fell to the same degree as in non pregnant women.

**3. PERIPHERAL RESISTANCE**

Peripheral resistance decrease by 20 to 30% at 21 to 24 weeks which gradually returns to normal at term. This dramatic decrease is explained by trophoblastic erosion of maternal endometrial vessels causing an effect similar to AV fistula, increased production of placental and ovarian steroid hormones that decrease vascular resistance and the effect of prolactin (Rovinsky, J.J. et al, 1966), (Metcalfe, J. et al, 1974), (Szekely, P. et al, 1974) (Ueland, K. 1978). Bader, R.A. et al in 1955 mentioned that the profound fall in systolic arterial resistance which occurs in the first trimester because of the utero placental circulation, is not large enough at this time to affect total peripheral resistance.

Kerr, M.G. et al (1965) showed that compression of the inferior venacava by the gravid uterus in late pregnancy when the patient is supine, may cause complete occlusion, with a collateral circulation by means of the paravertebral veins being opened up. If these collateral vessels do not respond immediately, the venous return to the heart is reduced and the patient develops "Supine hypotension syndrome". This is of importance clinically in estimating the blood pressure and with regard to the position of the patient during labour and delivery.

As peripheral resistance decreases, there occurs a fall in diastolic blood pressure. It decreases near the end of the first trimester, and continues to decrease throughout the second trimester and thereafter, begins to rise towards nonpregnant levels. The fall in diastolic pressure exceeds that of the systolic, resulting in a widened pulse pressure.

**MEAN ARTERIAL PRESSURE**

Fallis, N. et al (1963) reported that primigravid women developing 'toxaemia' in the third trimester had high blood pressure earlier in pregnancy when compared with women who remained normotensive. Page, E.W. et al (1976) employed the concept of Mean Arterial Pressure (MAP) since he considered it to be the most physiological determinant. It can be obtained by the most commonly used formula advocated by Burton, A.C. et al (1965),

$$MAP = \frac{\text{Systolic BP} + 2(\text{Diastolic BP})}{3}$$

Page, E. W. et al (1976) studied the impact of mean arterial pressure in the mid trimester upon the outcome of pregnancy. They concluded that when MAP is 90 mmHg or more, there is a significant increase in the still birth rate and the increased frequency of intrauterine foetal growth retardation. Thus women having an average of 90 mmHg or more during the fifth and sixth month can be considered in high risk category and requires close periodic evaluation of fetoplacental function.

Arterial blood pressure is nearly always measured in clinical practice by sphygmomanometer and the technique and conditions of measurement must be standardized, if consistent results are to be obtained.

In nonpregnant females, point of disappearance of Korotzkoff's sound (Phase V) corresponds most closely to the true diastolic blood pressure as measured by intrarterial catheter. In pregnancy, however, the auscultated Korotzkoff's sound may persist to zero due to marked peripheral vasodilatation and the point of muffling (i.e. phase IV) provides a better measure of true diastolic BP.

Hence, in pregnancy WHO recommends that blood pressure should always be measured with the patient lying down and resting comfortably on her right side at 30 degree to the horizontal with the sphygmomanometer cuff at the level of heart with phase IV as the marker of diastolic BP. For the diagnosis of hypertension two consecutive abnormally higher measurements should be made atleast 4 to 6 hours apart.

Blood pressure normally falls at the beginning of pregnancy and

reaches its lowest level in the second trimester when "diastolic blood pressure is on an average of 15 mmHg low and systolic blood pressure 5 mmHg low in the lying down position than the pregnancy levels (McGillivray et al,1969; Friedman, E.A. et al, 1977). Friedman, E.A. et al in 1977 noted a significant and progressive increase in the perinatal mortality with increase in diastolic blood pressure above 86 mmHg.

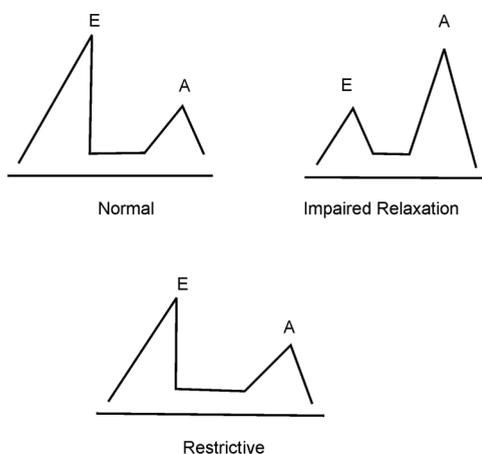
### ECHOCARDIOGRAPHY

Echocardiography involves the application: of reflected ultrasound i.e. sound waves of frequency far above the normal audible range, for the evaluation of the heart. Pulsed ultrasound waves in the frequency range of 1.60 - 3.50 MHz are emitted from a transducer probe that also act as a receiver for reflected waves. Since ultrasound is reflected at interfaces having different acoustic impedances, internal structures of the heart that possess different impedances e.g. myocardium and atrial and ventricular chambers and valves can be readily imaged. Thus, it provides both qualitative and quantitative data in variety of cardiac disorders.

### ECHOCARDIOGRAPHIC EVALUATION OF DIASTOLIC FUNCTION

Normal diastolic function allows adequate ventricular filling during rest and exercise without abnormal increase in diastolic pressure. Adequate diastolic filling ensures normal stroke volume according to Frank-Starling mechanism. The initial diastolic event is myocardial relaxation that cause pressure to decrease rapidly in the LV after the end of contraction and during early diastole. When LV pressure falls below LA pressure, the mitral valve opens and rapid early diastolic filling begins. Normally, the predominant determinant of early diastolic filling driving force is LV elastic recoil and the rate of LV relaxation, whereas LA pressure plays a lesser role as driving force. Approximately 80% of LV filling normally occurs during this phase. As a result of rapid filling, LV pressure increases and exceeds LA pressure, and this loss of positive driving force results in deceleration of mitral flow velocity. A positive transmitral pressure gradient and flow are again created by atrial contraction during late diastole, accounting for 15—20% of LV filling in normal individuals (Nishimura, RA. et al, 1989).

Mitral flow velocities are obtained by pulsed wave Doppler echocardiography with the sample volume located between the tips of mitral leaflets during diastole. Initial classification of diastolic filling is made from peak velocity of early rapid filling wave (E), peak velocity of the late filling wave caused by atrial contraction(A) and E/A ratio (Oh, J.K. et al. 1997).



**Figure 5: Graphic presentation of pulse wave Doppler with normal and impaired diastolic function.**

Diastolic filling pattern is characterized further by measuring the DT (Deceleration Time) which in the interval from the peak of E velocity to its extrapolation at the baseline. DT is characteristically prolonged in patients with relaxation abnormality as the predominant diastolic dysfunction, because it takes longer for LA and LV pressure to be equilibrated with a slower and continued fall in LV pressure until mid to late diastolic and reduced rate of filling during early diastole. DT is shortened when there is a rapid filling as a result of vigorous LV relaxation and elastic recoil in healthy normal subjects, or conversely if there is a decrease in LV compliance resulting in a more marked

increase in LV pressure in early diastole. The degree of shortening appears to be related to LV operating chamber stiffness. The IVRT is the time from aortic valve closure to mitral valve opening. It generally parallels DT becoming prolonged with abnormal filling pressure (Oh, J.K. et al, 1997).

The duration of mitral flow with atrial contraction compared with duration of reversal of atrial flow in the pulmonary vein has been a helpful means of estimating LV diastolic pressure in patients with heart disease.

### ECHOCARDIOGRAPHIC EVALUATION OF DIASTOLIC FUNCTION

LV systolic function can be assessed by M-Mode, 2D and Doppler technique. M-Mode gives excellent resolution and allows measurements of LV dimension and wall thickness. M-Mode can be used to measure LV cavity dimensions, wall thickness and motion. Poor LV systolic function is associated with increased LV dimensions. LVESd and LVEDd are made at the level of mitral valve leaflet tips in the parasternal long-axis view. Measurements are taken from endocardium of the left surface of the IVS to the endocardium of the LVPW. The ultrasound beam should be as perpendicular as possible to the IVS. Care must be taken to distinguish between the endocardial surface and chordae tendinae on the M-Mode tracings.

LVEDd	Normal	3.5-5.6 cm
LVESd	Normal	2.0-4.0 cm

Fractional shortening (FS) is a commonly used measure in the percentage change in LV internal dimensions (not volumes) between: systole and diastole.

### Aims and objectives:-

- To evaluate the cardiac status in peripartum period by studying systolic and diastolic left ventricular function by 2D-ECHO and clinical examination.

### MATERIAL AND METHODS

The study was conducted in the Department of Medicine, Jawaharlal Nehru Medical College and associated Acharya Vinoba Bhave Rural Hospital, Sawangi (Meghe), Wardha over a period of 18 months i.e. from 1<sup>st</sup> July 2004 to 31<sup>st</sup> December 2005.

### INCLUSION CRITERIA

- All pregnant women in the peripartum period attending the OPD for follow-up or admitted in Obstetrics and Gynaecology ward in AVBRH attached to JNMC, Sawangi (Meghe), Wardha were included in the study.
- Healthy nonpregnant females (control group) of similar age attending the OPD or accompanying other patients to hospital.

### EXCLUSION CRITERIA

- Females with previous heart diseases.
- Females with major diseases like liver failure, thyrotoxicosis, severe anemia, diabetes mellitus, etc.
- Females with hemoglobin less than 10 gm%.

### STUDY DESIGN

The clinical material comprised of 50 females in the peripartum period in the reproductive age group to be selected randomly. A control group consisting of 50 healthy non-pregnant females of similar age group were included in the study.

### STUDY PROTOCOL

Randomly selected pregnant females attending the OPD or admitted in Obstetrics and Gynaecology ward were explained about the study. With a voluntary consent 50 females in peripartum were selected as per

### inclusion criteria.

Detailed history and clinical examination was done as per protocol. Routine Investigations including Hb%, counts, peripheral smear, electrolytes, ECG and 2D-ECHO were done.

### INFORMED CONSENT

A written consent was taken from subjects after explaining the nature of the test to them.

### PREPARATION FOR THE TEST

The subjects were asked to relax and not to be apprehensive.

**CLOTHING**

Subjects were asked to wear hospital scrub shirts.

**SKIN PREPARATION**

Electrical jelly was used for probe contact. The probe position were as per standard recommendation.

**INSTRUMENTATION**

All subjects were subjected to ECHO in the last month of pregnancy.

**INVESTIGATIONS**

Baseline Investigation

1. Hb%
2. TLC
3. DLC
4. PS
5. Sr. Na+, Sr, K+
6. ECG

**ECHO :**

2D-ECHO was done on Phillips machine model number 888-358 Core Vision ECHO and USG machine.

Different views used were

- Parasternal long axis view
- Apical long axis view
- Apical 2 chamber view
- Apical 4 chamber view

Different modalities used were

- 2D
- M-Mode
- Pulse wave doppler

Following parameters were noted

- a) Systolic function on ECHO by following parameters:
  - Left ventricular internal diameter — Systole and Diastole
  - Left atrial size (LA)
  - E point septal separation (EPSS)
  - Left ventricular ejection fraction (LVEF)
  - Fractional shortening (FS)

b) Diastolic function on ECHO by following parameters (pulse wave Doppler):

- E-wave
- A-wave
- E/A ratio
- Deceleration time (DT)
- Iso volumetric relaxation time (IVRT)

**OBSERVATIONS**

Our study was a case control study.

Morphometric characteristics were noted and echocardiography was done in all randomly selected subjects consisting of study group (pregnant females in peripartum period, n=50) and control group (non—pregnant females of similar age group, n=50). Systolic and diastolic functions were evaluated.

The following variables—were compared and tested for statistical significance in both the groups :-

1. Age in years
2. Height in cms
3. Weight in kgs
4. Body Mass Index in (kg/m2)
5. Resting B.P.( Systolic and Diastolic)
6. Heart Rate
7. Edema Feet
8. Left ventricular systolic function
9. Left ventricular diastolic function

Haemodynamic changes were compared with the study and control groups.

LV size was compared with case and control groups.

LA size and EPSS was compared with the case and control groups.

Fractional shortening and LV ejection fraction was compared with the case and control groups.

E and A wave velocities were compared with the case and control groups.

IVRT and DT were compared with the case and control groups.

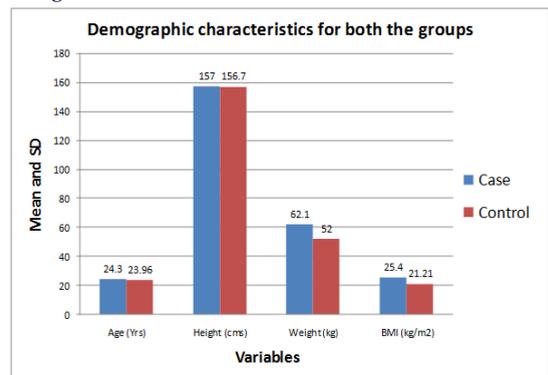
**TABLE 1**

Table showing demographic characteristic both the groups.

Variables	Cases n = 50	Control n = 50	Z – Value	P – Value
	Mean + SD	Mean + SD		
Age (Yrs)	24.3 + 2.99	23.96 + 2.91	1.90	NS p>0.05
Height (cms)	157 + 4.42	156.7 + 4.74	0.33	NS p>0.05
Weight (kg)	62.10 + 2.22	52.00 + 4.15	50.50	S p<0.05
BMI (kg/m2)	25.40 + 1.77	21.21 + 1.78	20.7	S p<0.05

The observations in table no. 1 is depicted in Bar Diagram No. 1

**Bar diagram No.1**



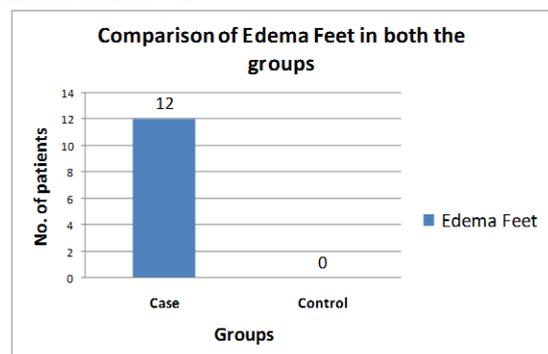
**TABLE 2**

Table showing comparison of edema feet in both the groups.

Variables	Cases n = 50	Control n = 50	X2 – Value	P – Value
Edema Feet	12	00	13.64	0.0002 S p<0.05

The observations in table no. 2 is depicted in Bar Diagram No. 2

**BAR DIAGRAM NO.2**



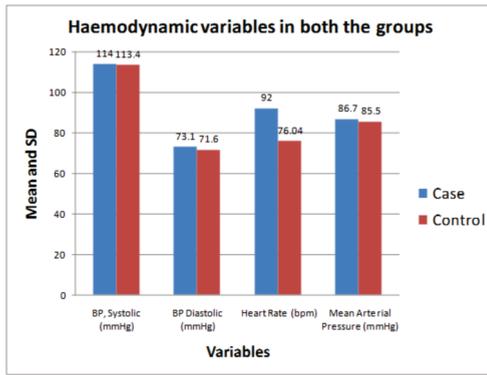
**TABLE 3**

Table showing haemodynamic variables in both the groups.

Variables	Cases n = 50	Control n = 50	Z – Value	P – Value
	Mean + SD	Mean + SD		
BP, Systolic (mmHg)	114 + 2.58	113.4 + 7.17	1.80	NS p>0.05
BP, Diastolic (mmHg)	73.1 + 4.91	71.6 + 6.17	1.35	NS p>0.05
Heart Rate (bpm)	92.0 + 3.10	76.04 + 4.49	20.7	S p<0.05
Mean Arterial Pressure (mmHg)	86.7 + 3.56	85.50 + 6.06	5.73	S p<0.05

The observations in table no. 3 is depicted in Bar Diagram No. 3

**BAR DIAGRAM NO.3**



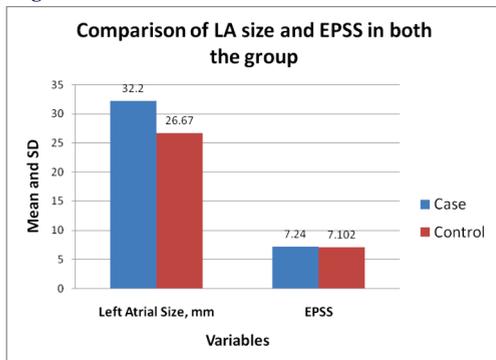
**TABLE 4**

Table showing comparison of LV size in both the groups.

Variables	Cases n = 50	Control n = 50	Z – Value	p – Value
	Mean + SD	Mean + SD		
LV End Diastolic diameter (mm)	39.3 + 4.19	39.12 + 4.91	1.00	NS p>0.05
LV End Systolic diameter (mm)	24.4 + 2.00	24.28 + 4.27	0.78	NS p>0.05

The observations in table no. 4 is depicted in Bar Diagram No. 4

**Bar Diagram No.4**



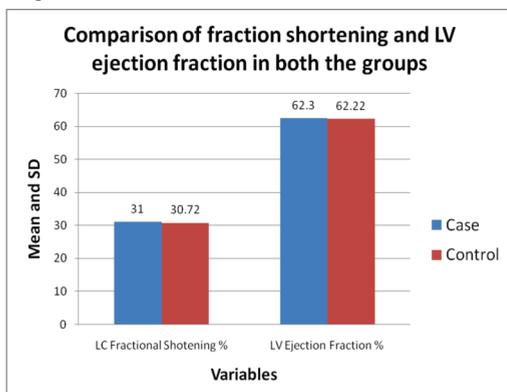
**TABLE 5**

Table showing comparison of fractional shortening and LV ejection fraction in both the groups.

Variables	Cases n = 50	Control n = 50	Z – Value	P – Value
	Mean + SD	Mean + SD		
LV Fractional Shortening %	31.0 + 3.8	30.72 + 0.86	1.40	NS p>0.05
LV Ejection Fraction %	62.3 + 9.71	62.22 + 1.23	0.23	NS p>0.05

The observations in table no. 5 is depicted in Bar Diagram No. 5.

**Bar Diagram No 5**



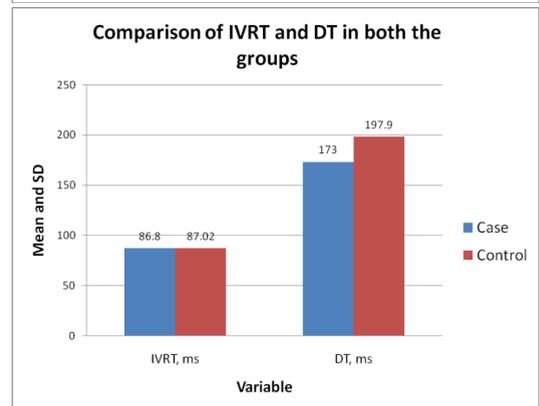
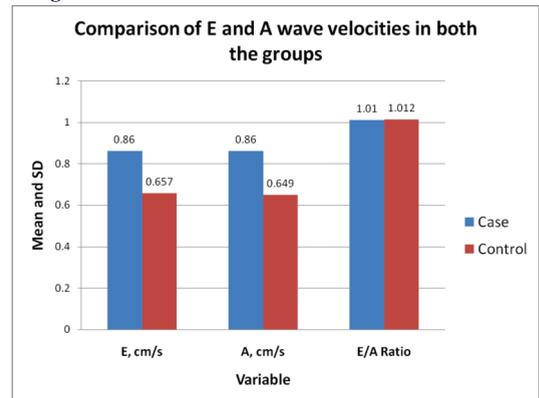
**TABLE 6**

Table showing comparison of E and A wave velocities in both the groups.

Variables	Cases n = 50	Control n = 50	Z – Value	P – Value
	Mean + SD	Mean + SD		
E (cm/s)	0.86 + 0.03	0.657 + 0.04	28.3	S P<0.05
A (cm/s)	0.86 + 0.03	0.649 + 0.03	33.8	S P<0.05
E/A Ratio	1.01 + 0.02	1.012 + 0.04	0.29	NS p>0.05

The observations in table no. 6 is depicted in Bar Diagram No. 6

**Bar Diagram No.6**



**DISCUSSION**

The present study was conducted at Acharya Vinoba Bhave Rural Hospital, Sawangi (Meghe), Wardha during the period from 1<sup>st</sup> July 2004 to 31<sup>st</sup> December 2005.

A total of 100 subjects, apparently healthy, were studied and were divided into study (n=50) and control group (n=50). All subjects in the study group were pregnant females.

The mean age in study and control group was 24.3 ± 2.99 years and 23.96 ± 2.91 years (Z=1.90, p>0.05) respectively. This was statistically insignificant. The subjects examined clinically and their heights and weights were recorded. The mean height (in cms) in the study and control group was 157 ± 4.42 and 156.7 ± 4.74 (Z=0.33, p>0.05). This was statistically insignificant. The mean weight (in kgs) in the study and control group was 62.10±2.22 and 52.00 ± 4.15 (Z=50.5, p<0.05) respectively. This was statistically significant. The BMI was calculated in the two groups. It was found to be mean value of 25.40 ± 1.77 and 21.21 ± 1.78(Z=20.7, p<0.05) respectively in case and control groups. This was statistically significant.

Thus, the two groups were comparable in respect to age and height. The comparison of weight and BMI was found to be statistically significant. This is because the weight increases during pregnancy due to the increased blood volume. These findings are consistent with the earlier study of James Ford Clap III et al(1997) who had shown in his study that the body weight increases in pregnancy and hence the BMI.

Pedal edema was shown to be present during pregnancy. This was present in 12 females in the case group ( $\chi^2$  value=13.64,  $p<0.05$ ). This was statistically significant. This was consistent with earlier studies of Howard, B.K. et al (1953) and Oian, P. et al (1985). This may be due to the decrease in the colloid oncotic pressure induced by pregnancy.

Our Study	Variables	Mean + SD	Mean + SD	P – Value
	BP, Systolic (mmHg)	114 + 2.58	113.4 + 7.17	NS $p>0.05$
	BP Diastolic (mmHg)	73.1 + 4.91	71.6 + 6.17	NS $p>0.05$
	Heart Rate (bpm)	92.0 + 3.1	76.04 + 4.49	S $P<0.05$
	Mean Arterial Pressure (mmHg)	86.7 + 3.56	85.50 + 6.06	S $P<0.05$

Andreas Mesa et al Study	Variables	Mean + SD	Mean + SD	P – Value
	BP, Systolic (mmHg)	113.0 + 11.0	111.0 + 7.00	NS $p>0.05$
	BP Diastolic (mmHg)	66.0 + 12.0	70.0 + 7.00	NS $p>0.05$
	Heart Rate (bpm)	87.0 + 10.0	68.0 + 6.00	S $P<0.05$
	Mean Arterial Pressure (mmHg)	82.0 + 10.0	84.0 + 7.00	NS $p>0.05$

The blood pressure in the two groups were taken and found to have a mean systolic blood pressure of  $114 \pm 2.58$  mmHg and  $113.4 \pm 7.17$  mmHg ( $Z=1.80$ ,  $p>0.05$ ) respectively in the case and control groups. This was statistically insignificant. The diastolic B.P. was shown to have mean of  $73.1 \pm 4.91$  and  $71.6 \pm 6.17$  ( $Z = 1.35$ ,  $p>0.05$ ) in the case and control groups respectively. The diastolic blood pressure was found to be statistically insignificant between the two groups. These findings were consistent with the earlier studies such as Andreas Mesa et al (1999), Mc Gillivray et al (1969), Friedman, E.A. et al (1977).

The Mean Arterial Pressure (MAP) was calculated for both the groups. This showed a mean of  $86.7 \pm 3.56$  and  $85.5 \pm 6.06$  ( $Z=5.73$ ,  $p<0.05$ ) in the case and control groups respectively. Thus this was statistically significant. This was consistent with the earlier study of Andreas Mesa et al (1999). Even though significant, it was less than a MAP of 90 mmHg which carries bad prognosis for fetal outcome as shown by Page, E.W. et al (1976), but not in our case.

Heart rate was taken of the subjects in the study and control groups and was found to be  $92.0 \pm 3.1$  and  $76.04 \pm 4.49$  ( $Z=20.7$ ,  $p<0.05$ ) respectively. This was statistically significant. This is due to the haemodynamic changes in pregnancy and the hyperdynamic circulation. These findings were consistent with the earlier studies such as Andreas Mesa et al (1999), Laird- Meeter et al (1979), Robson S.C. et al (1989).

Our Study	Variables	Mean + SD	Mean + SD	P – Value
	LV End Diastolic diameter (mm)	39.3 + 4.19	39.12 + 4.91	NS $p>0.05$
	LV End Systolic diameter (mm)	24.4 + 2.00	24.28 + 4.27	NS $p>0.05$

Andreas Mesa et al	Variables	Mean + SD	Mean + SD	P – Value
	LV End Diastolic diameter (mm)	43.00 + 4.00	43.00 + 3.00	NS $p>0.05$
	LV End Systolic diameter (mm)	28.00 + 3.00	28.00 + 2.00	NS $p>0.05$

The LVEDd was studied in the subjects by echocardiography and was found to be  $39.0 \pm 4.19$  and  $39.12 \pm 4.91$  ( $Z=1.0$ ,  $p>0.05$ ) respectively in the case and control groups. The LVEsD was studied in the subjects by echocardiography and was found to be  $24.4 \pm 2.00$  and  $24.28 \pm 4.27$  ( $Z=0.78$ ,  $p>0.050$ ) respectively in the case and the control groups. Thus, these were found to be statistically insignificant. These findings were consistent with the earlier studies of Andreas Mesa et al (1999), Laird-Meeter, K. et al (1979), Hunter, S. et al (1992).

Our Study	Variables	Mean + SD	Mean + SD	P – Value
	Left Atrial Size (mm)	32.2 + 1.39	26.67 + 3.45	S $P<0.05$
	EPSS	7.24 + 0.64	7.102 + 0.93	NS $p>0.05$

Andreas Mesa et al Study	Variables	Mean + SD	Mean + SD	P – Value
	Left Atrial Size (mm)	33.0 + 4.0	31.0 + 4.0	NS $p>0.05$
	EPSS	9.0 + 1.0	9.0 + 1.0	NS $p>0.05$

The LA size was studied in the subjects by echocardiography and was found to be  $32.2 \pm 1.39$  and  $26.6 \pm 3.45$  ( $Z=10.5$ ,  $p<0.05$ ) respectively in the case and control groups. This was statistically significant. This was due to the increased work that LA has to perform due to the volume overload state of pregnancy and also a lower Hb% in Indian population as compared to the European population. This was not consistent with the study of Andreas Mesa et al (1999) but was consistent with the study of Robson, S.C. et al (1989). Andreas Mesa et al (1999) study probably had subjects who had a higher Hb% as compared to Indian population.

The EPSS in the two groups was  $7.24 \pm 0.64$  and  $7.102 \pm 0.93$  ( $Z=0.71$ ,  $p>0.05$ ) respectively in the case and the control groups. Thus, this was statistically not significant. These findings were consistent with earlier studies of Andreas Mesa et al (1999), Laird-Meeter K. et al (1979), Hunter S. et al (1992), Valensise H. al (2002).

Our Study	Variables	Mean + SD	Mean + SD	P – Value
	LV End Fractional Shortening %	31.0 + 3.8	30.72 + 0.86	NS $p>0.05$
	LV Ejection Fraction %	62.3 + 9.71	62.22 + 1.23	NS $p>0.05$

Andreas Mesa et al Study	Variables	Mean + SD	Mean + SD	P – Value
	LV Fractional Shortening %	34.5 + 3.0	35.0 + 3.0	NS $p>0.05$
	LV Ejection Fraction %	60.0 + 3.0	60.0 + 3.0	NS $p>0.05$

The LV fractional shortening and LV ejection fraction in percentage were calculated by echocardiography for the study and control groups. The LV fractional shortening was found to be  $31.0 \pm 3.8$  and  $30.72 \pm 0.86$  ( $Z=1.4$ ,  $p>0.05$ ). The LV ejection fraction was found to be  $62.3 \pm 9.71$  and  $62.22 \pm 1.23$  ( $Z=0.23$ ,  $p>0.05$ ). Thus these findings were found to be statistically insignificant and were consistent with other studies such as Andreas Mesa et al (1999), Laird-Meeter, K. et al (1979), Hunter, S. et al (1992).

Thus, the systolic function as a whole in our study which included the parameters of LVEDd, LVEsD, LA size, EPSS, Fractional LV shortening, LVEF were studied. These did not show statistically significant difference between case and control groups except that a slight increase in the size of the LA occurs in the term patients. These findings were consistent with earlier study of Andreas Mesa et al (1999) except the LA size.

Our Study	Variables	Mean + SD	Mean + SD	P – Value
	E (cm/s)	0.86 + 0.03	0.657 + 0.04	S $P<0.05$
	A (cm/s)	0.86 + 0.03	0.649 + 0.03	S $P<0.05$
	E/A Ratio	1.01 + 0.02	1.012 + 0.04	NS $p>0.05$

Andreas Mesa et al Study	Variables	Mean + SD	Mean + SD	P – Value
	E (cm/s)	0.84 + 0.19	0.78 + 0.13	NS $p>0.05$
	A (cm/s)	0.68 + 0.14	0.56 + 0.05	NS $p>0.05$
	E/A Ratio	1.30 + 0.20	1.4 + 0.2	NS $p>0.05$

The diastolic function included parameters E-Wave, A-Wave, E/A Ratio, IVRT and DT studied by echocardiography. The E wave in cms/sec were found to be mean  $0.86 \pm 0.03$  and  $0.657 \pm 0.04$  ( $Z=28.3$ ,  $p<0.05$ ) in case and control groups respectively. The A-wave was found to be mean  $0.86 \pm 0.03$  and  $0.649 \pm 0.03$  ( $Z=33.8$ ,  $p<0.05$ ) in the case and control groups respectively. Thus these findings were statistically significant. This was not consistent with study of Andreas Mesa et al (1999) but similar findings which showed an increase in E and A velocity in the term patients. E/A ratio was found to be mean  $1.01 \pm 0.02$  and  $1.012 \pm 0.04$  ( $Z=0.29$ ,  $p>0.05$ ) in the case and control groups respectively. This was statistically insignificant. This was

consistent with earlier studies such as Andreas Mesa et al (1999), Duvekott J.J. et al (1994). This was probably due to the fact that females in Andreas Mesa et al (1999) study were compared with the above parameters in the third trimester and postpartum rather than in the term pregnant females and nonpregnant controls as in our study.

Our Study	Variables	Mean + SD	Mean + SD	P – Value
	IVRT (ms)	86.8 + 3.71	87.02 + 3.61	NS p>0.05
DT (ms)	173 + 2.93	197.9 + 5.82	S P<0.05	

Andreas Mesa et al Study	Variables	Mean + SD	Mean + SD	P – Value
	IVRT (ms)	82.0 + 12.0	84.0 + 8.0	NS p>0.05
DT (ms)	180.0 + 13.0	206.0 + 59.0	S P<0.05	

IVRT and DT were also studied in both the groups. IVRT showed the values of mean of  $86.8 \pm 3.71$  and  $87.02 \pm 3.61$  ( $Z=1.2$ ,  $p>0.05$ ) in the case and control groups respectively. This was statistically insignificant. DT showed a mean of  $173 \pm 2.93$  and  $197.9 \pm 5.82$  ( $Z=2.47$ ,  $p<0.05$ ) in the case and control groups respectively. This was statistically significant. These findings were consistent with earlier studies such as Andreas Mesa et al (1999), Valensise et al (2002). This was due to the increased heart rate in the term pregnancy.

So, in our study we have tried to correlate the different clinical and echocardiographic parameters in subjects in peripartum period with the controls. With reference to the above studies we found changes in some clinical and echocardiographic parameters which highlight the volume overload state of the pregnancy. We tried to find out the dysfunction that might have occurred in the hearts of females in peripartum period and correlate to those of controls.

## SUMMARY AND CONCLUSIONS

Pregnancy is a volume overload state. This can affect the female haemodynamics and can alter the LV function. This can be shown on echocardiography by evaluation of systolic and diastolic function. There have been previous studies on the diastolic function of the heart in pregnant women but few on systolic function. In pregnant women we had in the above context, underwent attempt to correlate the above facts. The outcome of this study has enhanced our understanding of the changes that might occur in pregnant females at term. This helps us understand the normal changes that might occur at term as compared to non-pregnant females.

1. Pregnant females in postpartum period have rise in heart rate.
2. Due to the volume overload state, the BMI and weight increased.
3. Pedal edema occur in pregnant females at term.
4. The LA size increases slightly at term due to the increased work it has required to do at term in volume overload state.
5. EPSS is not affected at term.
6. The LV dimensions do not show any change in the term pregnancy.
7. LVEDd and LVESd are not affected.
8. The E and A wave velocities show change in peripartum period. E and A wave velocities increase during the term pregnancies but the ratio E/A remains as in the non-pregnant state.
9. IVRT and DT are less in the peripartum period.

Thus, it can be concluded that :-

1. Body weight and BMI increase in the peripartum period.
2. There is no systolic dysfunction in the peripartum period, though LA size increases.
3. There is no diastolic dysfunction. E and A wave velocities increases but ratio E/A is normal. IVRT and DT decreases in peripartum period.

The limitations of our study :-

- Observer bias, if any, could have influenced the result to some extent, as blinding was not done.
- Females, though in the peripartum period were all studied in the last month of pregnancy only and not after delivery on echo.
- The group size in our study was small and studies with larger size are recommended to validate the results.

## ABBREVIATIONS

LV - Left Ventricle

LA - Left Atrium  
 LVH - Left Ventricular Hypertrophy  
 LVM - Left Ventricular Mass  
 P - Penn Convention  
 IVST - Inter Ventricular Septal Thickness  
 PWT - Posterior Wall Thickness  
 LVIDd - Left Ventricular Internal Dimension at end diastole  
 RWT - Relative Wall Thickness  
 CSA - Cross Sectional Area  
 MMT - Mean Muscle Thickness  
 IVRT - Iso Volumic Relaxation Time  
 DT - Deceleration Time  
 IVS - Inter Ventricular Septum  
 LVPW - Left Ventricular Posterior Wall  
 RI - Resistance Index  
 LVESd - Left Ventricular End Systolic Diameter  
 LVEDd - Left Ventricular End Diastolic Diameter  
 Hb% - Haemoglobin Percentage  
 TLC - Total Leucocyte Count  
 DLC - Differential Leucocyte Count  
 PS - Peripheral Smear  
 Sr Na+ - Serum Sodium  
 Sr K+ - Serum Potassium  
 ECG - Electrocardiogram  
 EPSS - E Point Septal Separation

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