LEFT VENTRICULAR DYSFUNCTION IN PREECLAMPSIA
(AN ECHOCARDIOGRAPHIC STUDY)

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CERTIFICATE

This is to certify that the dissertation entitled **"LEFT VENTRICULAR DYSFUNCTION IN PREECLAMPSIA (AN ECHOCARDIOGRAPHIC STUDY)"** is the bonafide original work of **Dr. A.EZHILMATHI,** done by her under my guidance in partial fulfillment of the requirements for MD (Obstetrics and Gynaecology) branch II examination of The Tamilnadu Dr. M.G.R. Medical University to be held in March 2010. The period of post graduate study and training was from May 2007 to February 2010. I forward this to the Tamil Nadu Dr. M.G.R. Medical University, Chennai, Tamil Nadu, India.

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INTRODUCTION

Preeclampsia is a pregnancy specific disorder which constitutes hypertension after 20 weeks of gestation and proteinuria. It is a multisystem disorders of unknown etiology.

It is one of the commonest complications of pregnancy and is a major cause of maternal and foetal mortality and morbidity. In developing countries it ranks second to anaemia as a cause of maternal mortality and morbidity, complicating 7-10% of all pregnancies. Cardiac failure with pulmonary edema may occur in hypertensive patients with normal heart. It is emphasised that pregnant women particularly if pre-eclamptic develop pulmonary edema more often than non-pregnant women. Further preeclampsia is a recognised predisposing factor for peripartum cardiomyopathy and future cardiovascular disease.

Pregnancy is characterised by a number of important hemodynamic changes. Blood volume increases by about 50%. The red cell mass increases by about 40%. The resting pulse rate increases by about 10 to 15 beats per minute. Cardiac output increases beginning in early pregnancy around 5^{th} week and continues to increase and reaches its peak between the middle of 2^{nd} and 3^{rd} trimesters and remains elevated during the remainder of pregnancy. Systemic arterial pressure begins to fall during the first trimester, reaches a nadir in midpregnancy and returns to pre-gestational level before term. The pulse pressure widens as the fall of diastolic pressure is greater than the fall of systolic pressure. The reduction in blood pressure results from a decline in systemic vascular resistance that occurs during pregnancy. These changes are largely physiological in normal pregnancies. But these changes are critical in patients with preeclampsia. Many studies have addressed the issues in relation to cardiac output, systemic vascular resistance, left ventricular stroke volume and pulmonary capillary wedge pressure.

It is also been speculated that associated subclinical left ventricular dysfunction may contribute to cardiac mortality and morbidity. There are only few data available regarding left ventricular function in pregnancy. So this prospective study on left ventricular systolic and diastolic function by echocardiography was undertaken in normal and preeclamrptic pregnancies.

REVIEW OF LITERATURE

historical prospective

Preeclampsia and Eclampsia is still shrouded in a cloud of mystery. Zweifel called it a disease of theories. Bernard wrote that Eclampsia was mentioned in the ancient Egyptian, Chinese, Indian and Greek literature. Probably the oldest reference is in the Advaitha Veda 200 BC.

classification of hypertensive disorders of pregnancy

Hypertensive disorders of pregnancy are classified in to five types by the working group of National High Blood Pressure Education Program (NHBPEP) (2000).

gestational hypertension

Blood Pressure (B.P) ≥ 140/90 for first time during pregnancy without proteinuria.
B.P returns to normal < 12 weeks postpartum final diagnosis made only postpartum and may have other signs and symptoms of preeclampsia for example epigastric discomfort or thrombocytopenia.

PreEclampsia

B.P ≥ 140/90mm Hg after 20 weeks of gestation Pronteinuria > 300 mg/24 hours or > 1 + dipstick.
INCREASED CERTAINTY OF PREECLAMPSIA
B.P ≥ 160/110 mmHg
Proteinuria 2g/24 hrs or > 2 + Dipstick.
Serum creatinine ≥ 1.2mg/dl unless known to be previously elevated
Platelets < 1,00,000/mm^3
Microangiopathic hemolysis
Elevated ALT or AST.
Persistent epigastric pain.
Persistent headache or other cerebral or visual disturbances.

Eclampsia:

Seizures that cannot be attributed to other causes in a woman with preeclampsia.

SUPERIMPOSED PREECLAMPSIA (ON CHRONIC HYPERTENSION)

New onset proteinuria > 300 mg/24hours in hypertensive women but no proteinuria before 20 weeks gestation.

A sudden increase in proteinuria or blood pressure or platelet count < 1,00,000/mm^3 in women with hypertension and proteinuria before 20 weeks gestation.

chronic hypertension

B.P > 140/90mmHg before pregnancy or diagnosed before 20 weeks gestation and persistent after 12 weeks postpartum.
Preeclampsia is classified in to mild and severe.

Indicators of severity

1. Severe hypertension B.P ≥ 160/110mmHg on two occasions atleast 6hours apart.
2. Proteinuria > 5g/24hours or 3+ or more on two random samples collected atleast 4 hours apart.
3. Elevated serum creatinine level
4. Oliguria < 500ml /24 hours
5. Pulmonary edema
6. Microangiopathic hemolysis
7. Thrombocytopenia (Platelets <100,000/mm^3)
8. Elevated ALT and AST
9. Pain in the epigastrium or right upper quadrant.
10. Headache, visual disturbances.

11. Intrauterine growth restriction.

INCIDENCE:

It occurs in 8 to 10% of pregnancies (Mudaliar and Menon 1972 ; Uphadhyay 1975).

The incidence varies with geographic location. Wide variation in the incidence have been reported from different institutions in different countries with widely varying maternal and perinatal mortality. Our hospital incidence is 6.5 to 8.5%.

PREDISPOSING FACTORS:

AGE :

Young primigravidas under 20 years are commonly affected. Because of the increased incidence of chronic hypertension with advancing age, older women are at greater risk for superimposed preeclampsia.

PARITY:

Preeclampsia is primarily a disease of primigravida (70%) and occurs much less frequently in subsequent pregnancies (Sibai and colleagues 1995).

SOCIAL STATUS:

Some investigators concluded that socioeconomically advantaged women have lesser incidence of preeclampsia. Lawlor and collegues (2005) did not observe this.

RACE:

Davies (1970) found that the incidence of preeclampsia was significantly higher in Muslim Arabs and Jews born in Iran than in Jews born in North Africa or Isreal. Conde – Agudelo and Belizan (2000), Sibai and colleagues (1997), Walker (2000) found increased incidence of preeclampsia in African American ethnicity.

GENETIC PREDISPOSITION:

Chesley and Cooper (1986) evidenced that familial and genetic factors have a strong influence on the occurrence of hypertension and proteinuria in pregnancy and are most likely due to Mendelian recessive trait.

TWIN PREGNANCY:

Mothers with twin pregnancies have a much higher incidence of preeclampsia compared with singleton (Macgillivary 1958, Sibai 2000, Maxwell 2001).

HYDATIDIFORM MOLE:

Preeclampsia occurs in large rapidly growing mole (Page 1939).

VITAMINS, TRACE ELEMENTS:

Earlier studies showed that women with low dietary calcium were at significantly increased risk for preeclampsia. Further studies failed to support this. Chappell and associates (1999) showed that treatment with antioxidants vitamin C or E significantly reduced endothelial cell activation and preeclampsia

ENVIRONMENTAL FACTORS:

Palmar and associates (1999) reported that living at higher altitude at Colorado increased the incidence of preeclampsia. Arterial blood pressure is higher in cold weather probably due to vasoconstriction.

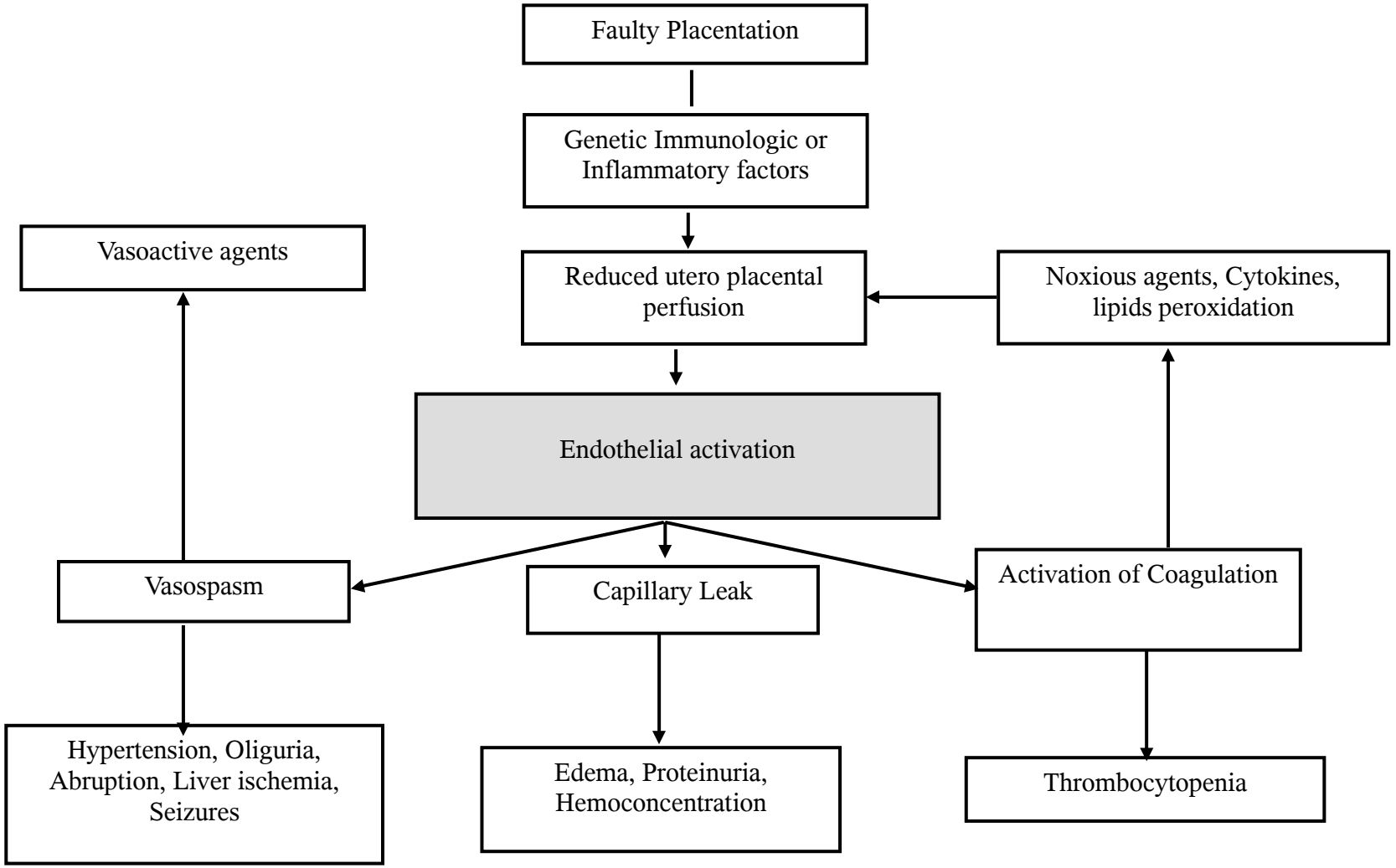
OBESITY:

The incidence of preeclampsia is significantly increased in obese women probably due to an underlying tendency to develop essential hypertension.

SMOKING:

Smoking is associated with reduced risk of hypertension during pregnancy. (Zang and colleagues 1999), (Bainbridge and associates 2005).

Pathogenesis of Pre-eclampsia



PATHOPHYSIOLOGY:

In normal pregnancies during implantation spiral arterioles undergo extensive remodelling as they are invaded by endovascular trophoblasts, replacing the endothelium and muscular wall of the vessel with subsequent enlargement of the blood vessel (Meekins and coworkers 1994). Madazali and colleagues (2000) showed that in preeclampsia there is incomplete trophoblast invasion. This leads to reduced uteroplacental perfusion and placental ischemia which causes endothelial dysfunction. Women with preeclampsia have elevated markers of inflammation. Increased soluble fms like tyrosine kinase (sflt-1), soluble endoglin (sEng) and cytokines predispose to endothelial dysfunction (Maynard and associates 2003). Sflt-1 antagonises vascular endothelial growth factor and placental growth factor by binding them and decreasing their unbound serum levels and impairs vascular endothelial growth factor and placental growth factor induced vascular relaxation. sEng is a component of TGF-β receptor complex and is a hypoxia inducible protein. It is antiangiogenic as it impairs TGF-β binding to cell surface receptors. An alteration in the balance of circulating factors leads to endothelial dysfunction (Luttum and Carmeliet 2003). Redman and colleagues 1999 have proposed that the endothelial cell dysfunction associated with preeclampsia can result from a generalized perturbation of the maternal intravascular inflammatory adaptation to pregnancy.

INCREASED PRESSOR RESPONSE:

Normally pregnant women develop refractoriness to infused vasopressors (Abdul karim and Assail 1961). Increased vascular reactivity to pressors in women with early preeclampsia has been identified by Raab and coworkers 1956 and Talledo and associates 1968 using either norepinephrine or angiotensin II and by Dieckmann and Michel1937 and Browne1946 using vasopressin. Gant and coworkers 1973 demonstrated that increased vascular reactivity to angiotensin II clearly preceded onset of preeclampsia.

MECHANISM:

It appears unlikely that the normally blunted pressor response to angiotensin II is due to downregulation or decreased affinity of angiotensin II vascular smooth muscle receptors (Mackangee and associates 1991). The metabolic clearance rate of angiotensin II is not altered in women with preeclampsia (Magneesand coworkers 1994). Based on the findings of number of studies it was concluded that the blunted pressor response was principally due to decreased vascular responsiveness mediated in part by vascular endothelial synthesis of prostaglandins or prostaglandins like substances (Cunningham and associates 1975, Gant and coworkers 1974).

VASOSPASM :

Atleast two vasoconstrictor mechanism may be operative in preeclamptic women. In these women arachidonic acid is converted to thromboxane A2, with an accompanying reduction of prostacyclin and prostaglandin E2 in the cyclooxygenase pathway (Cateila and associates 1990, Fitzgerald and colleagues 1990, Mitcheil and koeing 1991, Tannirandorn and coworkers 1991).

This pathway is responsive to low dose aspirin therapy. The second route is via the lipoxygenase pathway which results in an increased platelet production of 15-hydroxyecosatetraenoic acid. This inhibits prostacyclin production resulting in further vasoconstriction. Biag and coworkers 1990 also reported an increased lipooxygenase pathway activation in placentas from hypertensive women.

HEAMODYNAMICS:

The changes and deterioration of function in a number of organs and systems has been

identified in severe preeclampsia and eclampsia. Severe preeclampsia and eclampsia are accompanied by multiple derangements of cardiovascular system and fluid compartments.

During normal pregnancies the maternal blood volume increases markedly. At or near term it averages about 40 to 50% above the nonpregnant levels. It starts to increase during the first trimester, expands rapidly during the second trimester and then rises at a much slower rate during the third trimester to plateau during the last several weeks of pregnancy. Increased blood volume results from an increase in plasma and erythrocytes. Although more plasma than RBCs is usually added to the maternal circulation the increase in volume of circulating erythrocytes averages about 33%. Pregnancy induced hypervolemia serves to meet the demands of the enlarged uterus with its greatly hypertrophied vascular systems, to protect the mother and foetus against the deleterious effects of impaired venous return in the erect and supine position and very importantly to safeguard the mother against the adverse effects of blood loss associated with parturition.

Augmentation of blood volume alters the stroke volume and cardiac output.

Cardiac output during pregnancy is estimated to exceed the output to the nonpregnant state by 30 to 50%. It begins to rise around the fifth week and peaks between the middle of second and third trimesters. Cardiac output is maintained at the same level thereafter. Changes in body position can induce substantial changes in cardiac output, its level rising in the lateral position and declining in supine position. The early increase in cardiac output can be attributed to augmented stroke volume that results from decreased peripheral vascular resistance. Later in pregnancy resting pulse increases and stroke volume increases even more, presumably because of increased diastolic filling from the expanded blood volume.

Heart rate also increases peaking during the third trimester. The average is about 10 to 15 beats/minute (Stein and coworkers 1999). Systemic arterial pressure begins to fall during the first trimester reaches a nadir in midpregnancy and returns to prepregnancy level before term. As the fall in diastolic pressure is greater than the fall in systolic pressure, pulse pressure widens. During normal pregnancy the total peripheral resistance decreases by 25%. Blood pressure is a product of cardiac output and total peripheral resistance. Hence the reduction in blood pressure results from a decline in total peripheral resistance which decreases due to vasodilatation, probably mediated by gestational hormonal activity increased levels of circulating prostaglandins, increased heat production by the developing foetus and the creation of low resistance circulation in the pregnant uterus.

Duwekot and colleagues 1993 showed that the left ventricular size is increased in normal pregnancies as a result of eccentric enlargement that increases radius to wall thickness and thus end diastolic dimensions. Markedly attenuated afterload manifested by diminished vascular resistance and mean arterial pressure allows the appreciable increase in cardiac output. Because changes in stroke volume are directly proportional to end diastolic volume, the implication is that the inotropic state of the myocardium changes little during normal pregnancy (Katz andcoworkers 1978). The sustained cardiac changes during pregnancy are similar to acute changes reported for moderate to strenuous exercise.

An important study by Clark and colleagues 1989 contributed greatly to the understanding of cardiovascular physiology during pregnancy. Using invasive haemodynamic studies they showed that late pregnancy was associated with increases in heart rate, stroke volume and cardiac output. Systemic vascular and pulmonary vascular resistances both decrease significantly, as did colloid osmotic pressure. There

was no change in intrinsic left ventricular contractility. Pregnancy is characterized by normal left ventricular function and not hyperdynamic function as once thought. These investigators concluded that maintenance of normal left ventricular filling pressures comes about as the result of ventricular dilatation.

HAEMODYNAMIC ALTERATIONS IN PREECLAMPSIA;

In preeclampsia there are alterations in the physiological changes in the haemodynamics. It is known that increase in intravascular volume that occurs in normal pregnancy is diminished or completely absent in patients with preeclampsia. This may be due to generalised vasoconstriction made worse by increased vascular permeability. Haemodynamic values obtained in women with preeclampsia using invasive cardiovascular monitoring by various investigators showed variables that define cardiovascular status range from high cardiac output with low vascular resistance to low cardiac output with high vascular resistance. Similarly left ventricular filling pressures estimated by pulmonary capillary wedge pressure determination range from low to pathologically high. Atleast three factors may explain these differences. 1) Women with preeclampsia might present with spectrum of cardiovascular findings dependent of severity and duration. 2) chronic underlying disease may modify the clinical presentation or 3) therapeutic interventions may significantly alter these findings. It is likely that more than one of these is operative.

Haemodynamic status of women with preeclampsia prior to active treatment has also been studied by Cotton and associates 1964. They identified normal left ventricular filling pressures, high systemic vascular resistance and hyperdynamic ventricular function. Beneditti 1980, Hankins 1984 and their associates reported that women with severe preeclampsia who were being treated with magnesium sulphate and hydralazine plus aggressive intravenous fluid therapy for volume expansion had the lowest systemic vascular resistances and highest cardiac output. These women had filling pressures that exceeded normal but their ventricular function remained hyperdynamic because of increased cardiac output. Cotton and coworkers 1988 reported findings from 45 women with severe preeclampsia or eclampsia and described high systemic vascular résistance and hyperdynamic ventricular function in most of the patients.

LEFT VENTRICULAR (LV) MECHANICS IN PREECLAMPSIA:

Increased systemic vascular resistance and contracted blood volume are characteristic findings in preeclampsia. These alterations in cardiovascular hemodynamics can adversely affect ejection phase indices of left ventricular performance making it difficult to separate abnormalities resulting from changes in load from those caused by depressed myocardial contractility. To address this issue the contractility-sensitive, load-independent relationship between left ventricular end-systolic wall stress and rate-corrected velocity of fiber shortening was assessed in 10 nulliparous patients with preeclampsia. Comparisons were made with data obtained from 10 age-matched normotensive women with uncomplicated pregnancies (control subjects). Studies were performed by means of two-dimensionally targeted M-mode echocardiography and calibrated carotid pulse tracings during early labor, 1 day after delivery, and 4 weeks after delivery. During early labor and 1 day after delivery, patients with preeclampsia had elevated blood pressure and increased total systemic resistance. These parameters returned to normal by 4 weeks after delivery. Before delivery and 24 hours after delivery, the patients with preeclampsia had lower overall left ventricular performance (as measured by cardiac output and rate-corrected velocity of fiber shortening) and higher left ventricular afterload (as measured by left ventricular end-systolic wall stress) when compared with control subjects. These differences were no longer

> present 4 weeks after delivery. Despite the time-related intergroup differences in hemodynamics, left ventricular contractility was similar between normotensive and preeclamptic subjects at all stages of the study. Thus when load is eliminated as a confounding variable, the decrements in overall left ventricular performance measured in patients with preeclampsia reflect a mechanically appropriate response to increased afterload rather than an abnormality in the ventricular contractile state. (Rafik Hamad et al Am Heat J. 1991 Jun; 121)

In pregnancies complicated by preeclampsia, especially early-onset preeclampsia, the diastolic LV function is impaired and levels of biomarkers, NT-pro-BNP and cystatin C, are increased in comparison to normal pregnancy.

Cardiac natriuretic peptides include atrial natriuretic peptide (ANP) and B- natriuretic peptide (BNP) which are secreted by cardiomyocytes in response to stretch of atrial wall due to volume overload. They are vasoactive and promote sodium and water excretion. Lowe and coworkers (1992) showed that plasma levels of ANP during normal pregnancy are maintained in the nonpregnant range despite the increase in plasma volume. This physiological adaptation may be important in allowing the expansion of extracellular fluid volume in normal pregnancy. Borghi and colleagues (2000), Gallery and Lindheimer (1999) showed that secretion of ANP is increased in preeclampsia.

Recently BNP has been evaluated as a marker for left ventricular function (Heidenrich and associates 2004). In normal pregnancy median BNP values are less than 20 pg/ml and stable throughout gestation. In severe preeclampsia BNP levels elevated. This may reflect ventricular stress or subclinical cardiac dysfunction associated with preeclampsia.

Pulmonary edema occurs in 3% of preeclamptic patients and is a significance cause of maternal morbidity and mortality. Sibai et al 1987 found an incidence of 2.9% in preeclampsia. Pulmonary edema may occur due to left ventricular dysfunction secondary to high systemic vascular résistance , volume overload in the phase of contracted intravascular space, decreased plasma colloid oncotic pressure or pulmonary capillary membrane injury.

Diastolic dysfunction refers to an abnormality in the heart's filling during diastole. It is characterised by elevated diastolic pressure in the left ventricle despite normal or subnormal diastolic volume. Any condition or process that leads to impaired relaxation of left ventricle can lead to diastolic dysfunction. In preeclampsia due to increase in total peripheral resistance there is increase in afterload which causes the left ventricle to become stiff leading to diastolic dysfunction. When the left ventricle cannot be normally filled blood regurgitates into left atrium and in backward direction into the lungs leading to pulmonary edema.

There are four echocardiographic patterns of diastolic dysfunction grade 1 to 4. The mildest form is called an abnormal relaxation pattern or grade 1 diastolic dysfunction and patients will not have clinical signs and symptoms of heart failure whereas grade 2 to grade 4 patients are symptomatic. The subclinical diastolic dysfunction with normal systolic function of left ventricle in preeclamptic patients can be detected by echocardiography which cannot be detected by routine history, physical examination and investigations like chest X- ray and ECG.

ECHOCARDIOGRAPHY:

Using 2D and 2D derived M-mode echocardiography quantitative measurements of cardiac dimensions, area and volume are derived.

Doppler echocardiography measures blood flow velocities in the heart.

Echocardiography allows assessment of cardiac function by parameters like a) end

diastolic pressure and chamber volume.
b) end systolic pressure , ejection pressure and fractional shortening and
c) Cardiac output.
Echocardiography is the simple and accurate noninvasive method to assess cardiac function during pregnancy. It uses ultrasound to create real time images of the cardiovascular system in action.

2D, M- mode, Doppler echo:

Ejection fraction is a simple measure of how much of end-diastolic volume is ejected or pumped out of the LV with each contraction. Quantitatively left ventricular ejection fraction (LVEF) can be calculated from M-mode, 2D and 3D echocardiogram.
Fractional shortening (FS) is the percent change in LV cavity dimension with each LV contraction and is calculated from the following equation.

$$FS = \frac{LVED - LVES}{LVED} \times 100\%$$

Where LVED is the LV end diastolic dimension and LVES is the LV end systolic dimension. The thickness of the ventricular posterior wall and that of ventricular septum are measured from the same M- mode echocardiogram. These values are used to calculate LV mass thereby detecting LV hypertrophy.
The most important cardiac complications like pulmonary edema and congestive heart failure can occur in the presence of an entirely normal LV systolic function. So the noninvasive evaluation of left ventricular diastolic function has become increasingly important.
In contrary to the popular belief diastole is an active metabolic process during which myocardium relaxes. The process of myocardial relaxation is as important as myocardial contraction. There is phasic blood flow to the ventricle during diastole i.e. early rapid ventricular filling followed by late diastolic filling by atrial contraction. Alterations in this phasic blood flow pattern reflect diastolic dysfunction. These diastolic inflow patterns can be assessed accurately by pulse Doppler echocardiography and colour flow imaging. They are expressed as E- early rapid filling velocity , A – late filling velocity by atrial contraction in milliseconds. The normal E/A ratio being 0.8 to 2.

Diastole begins with myocardial relaxation at the end of systolic contraction. During diastole the left ventricle must receive an adequate volume while maintaining low intracavitary pressure throughout a wide range of heart rate and loading condition. To accomplish this the ventricle relaxes during early diastole and the walls distend readily allowing the chamber to receive a wide range of inflow volumes at low filling pressures. To ensure optimal diastolic filling at rest and during exercise the left atrium contracts before ventricular activation, providing an additional boost to ventricular filling. During sinus rhythm the mitral inflow velocity displays a biphasic morphology consisting of an early peak (E) followed by a deceleration phase and by a second peak during atrial contraction(A).

Another valuable index in assessment of left ventricular diastolic function is isovolumetric relaxation time (IVRT). IVRT is the short period of early diastole during which ventricular volume remains constant and the pressure falls. It is the time between the closure of aortic valve and opening of mitral valve. The normal value being 73 to 110 ms. The value of more than 110ms is considered as prolonged IVRT reflecting LV diastolic dysfunction.
Diastolic filling pattern is characterized further by measuring the deceleration time, (DT). DT is the interval from the peak of the E velocity to its extrapolation to baseline.

In patients with a relaxation abnormality as the predominant diastolic dysfunction , DT is prolonged because with a slower and continouos decrease in LV pressure until mid to late diastole, it takes longer for LA and LV pressures to equilibrate.

AIM

To determine the left ventricular dysfunction in preeclamptic women by measuring the left ventricular systolic and diastolic indices using echocardiography.

SUBJECTS AND METHODS

Patients with preeclampsia who had been admitted in the antenatal ward of Government RSRM lying in hospital for women over the period of one year from 2008 September to 2009 September were the subjects of the study.

Among them 54 patients who fulfill the study criteria were selected and they constituted group 1 of the study.

INCLUSION CRITERIA

1. Patients with mild Preeclampsia which was diagnosed when there is a sustained systolic blood pressure of 140 mm Hg or more, and or a diastolic blood pressure of 90mm Hg or more present on two or more occasions atleast six hours apart occurring after 20th week of gestation associated with a proteinuria of ≥ 0.3gm protein in a 24 hours urine sample or ≥ 30 mg/dl or ≥ 1+ on dipstick in a random urine sample.
2. Patients with severe Preeclampsia which was diagnosed when blood pressure was ≥ 160/110 mmHg or proteinuria of > 5gm/24 hours or 2+ or more.
3. Age 18 to 35 years
4. Gestational age 28 to 36 weeks.
5. The known period of hypertension averaged 20 days.

EXCLUSION CRITERIA:

1. Patients with preexisting hypertension or
2. Patients with known cardiorespiratory disease
3. Patients with anaemia
4. Patients with gestational diabetes mellitus
5. Patients in labour.

Out of 54 patients studied 24 patients fulfilled the criteria for severe preeclampsia, 30 patients fulfilled the criteria for mild preeclampsia.

All the patients were put on antihypertensive drugs. All the cases of mild Preeclampsia were put only on tablet alpha methyldopa 250mg 8th hourly. The severe cases of preeclampsia were put on tablet alpha methyldopa 250mg 8th hourly and tablet nifedipine 10mg 8th hourly.

None of the patients were studied during labour.

CONTROL GROUP 2

50 normal normotensive pregnant patients selected at random who come to the OPD of our institute at the same period constituted group 2. Their gestational age ranged from 28 to 36 weeks. Their age ranged from 18 to 35 years. Mean age 24 years.

CONTROL GROUP 3

50 normal nonpregnant patients selected at random who come to the OPD of our institute at the same period who were from 18 to 35 years of age constituted group 3. Mean age is 26 years.

Group 1	*Group 2*	*Group 3*
Pre Eclamptic Women	Normotensive Pregnant women	Normal non - pregnant women

Group 1

Age	*No of Patients*	*Percentage*
18-20	17	31.48
21-25	22	40.74
26-30	10	18.57
31-35	5	9.25

Mean Age - 23 years

Group 2

Age	*No of Patients*	*Percentage*
18-20	14	28
21-25	19	38
26-30	13	26
31-35	4	8

Mean age - 24 years

Group 3

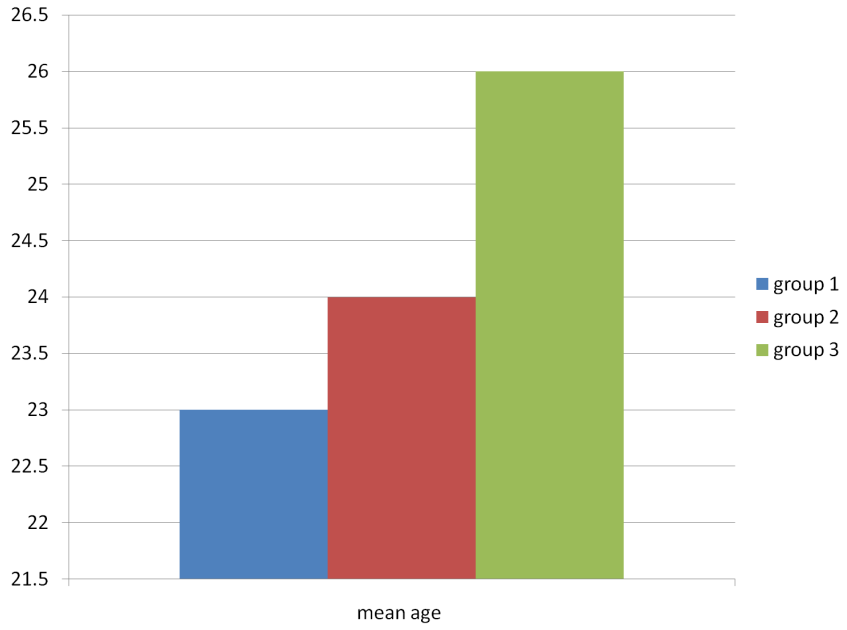
Age	*No of Patients*	*Percentage*
18-20	7	14

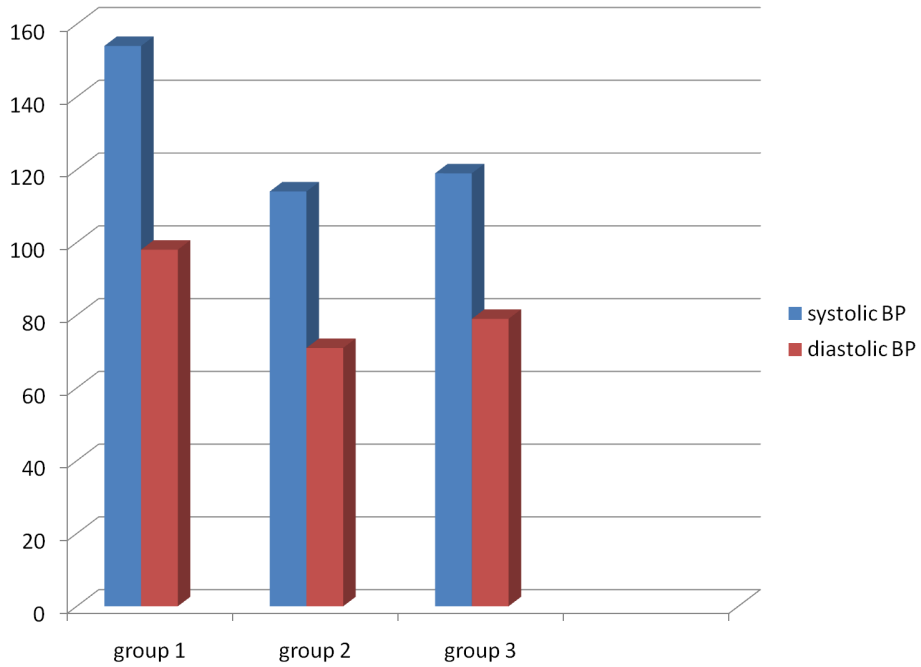
21-25	16	32
26-30	14	28
31-35	13	26

Mean age - 26 years

Parity

Group	*Nulliparous*	*Multiparous*
1	28	26
2	15	35
3	10	40





MEAN BLOOD PRESSURE OF THREE GROUPS

Group	*Systolic*	*Diastolic*
Group -1	154	98
Group - 2	114	71
Group - 3	119	79

ECHOCARDIOGRAPHIC STUDIES:

All patients were examined by a cardiologist using Aloka echo machine with 2.5mHz transducer at the Department of Cardiology Stanley Medical College Hospital. Echocardiography of left ventricle was obtained under standard conditions during quiet expiration with patients in the left lateral recumbent positions after the patients remained undisturbed in this position for 15 minutes.

MEASUREMENTS:

M-mode, 2D and Doppler echocardiographic evaluation were performed in all patients in the standard left parasternal axis view with continuous ECG gating according to the ASE guidelines. LV dimensions like EF, ESD and EDD are measured by 2D and M-mode. Interventricular septum and posterior wall diameter are also measured in M-mode. Using pulse doppler echocardiography mitral inflow velocities IVRT and DT are measured.

Parameters used to measure systolic functions are

$$EF = \frac{EDV - ESV}{EDV} \times 100$$

Normal 50 to 75%.

$$FS = \frac{LVED - LVES}{LVED} \times 100\%$$

Normal 25 to 46%.
EDV- end diastolic volume.
ESV- end systolic volume.
LVED – left ventricular end diastolic dimension.
LVES - left ventricular end systolic dimension.

Parameters used to measure diastolic function

E/A ratio: early filling / filling due to atrial contraction.
Normal 0.8 to 2.

Isovolumetric Relaxation Time (IVRT): Time interval between closure of aortic valve and opening of mitral valve. Normal 73 to 110ms. More than 110ms is considered as prolonged IVRT indicative of diastolic dysfunction.

Deceleration time: (DT)

Time taken for left atrial and left ventricular pressures to equilibrate.
Normal 160 to 230 ms. Prolonged in diastolic dysfunction.

RESULTS

The individual values of end diastolic dimensions, end systolic dimensions, EF, FS and mitral inflow velocities E,A are depicted in the graph.

The mean left ventricular end diastolic dimensions in group 1,2 and 3 were 4.81, 4.84 and 4.55 cms respectively. The mean end systolic dimensions of group 1,2 and 3 were 3.33, 3.30 and 3.06 respectively. There were no significant differences in the mean end diastolic and end systolic dimensions between preeclamptic and normotensive pregnant women, although the mean enddiastolic and end systolic dimensions in the two groups of pregnant women were significantly greater than in the nonpregnant group.

The mean EF in the three groups were 60%, 58.3% and 58.8% respectively. They were comparable and there is no statistical significance. 4 patients in group one showed EF < 50% that defines left ventricular systolic dysfunction.

Mean FS in the three groups were 30.3%, 31.6% and 32.6% respectively. Only 4 patients in group 1 showed FS < 25% that defines left ventricular systolic dysfunction.

MITRAL INFLOW VELOCITIES:

The mean E/A ratio in the three groups were 1.03, 1.23 and 1.24 respectively. 17 patients in group 1 had E/A ratio <1 indicative of diastolic dysfunction. Mean IVRT in the three groups were 108, 90 and 89 respectively. The mean DT in three groups were 209, 188 and 189 respectively . The same 17 patients who showed reversed E/A ratio also had prolonged IVRT and DT confirming the diagnosis of diastolic dysfunction.

STATISTICAL ANALAYSIS:

LV systolic and diastolic function indices were compared among the three groups using Tukey-HSD test with significance level .050. There was a statistically significant LV diastolic dysfnction in the preeclamptic group. P value <0.001.

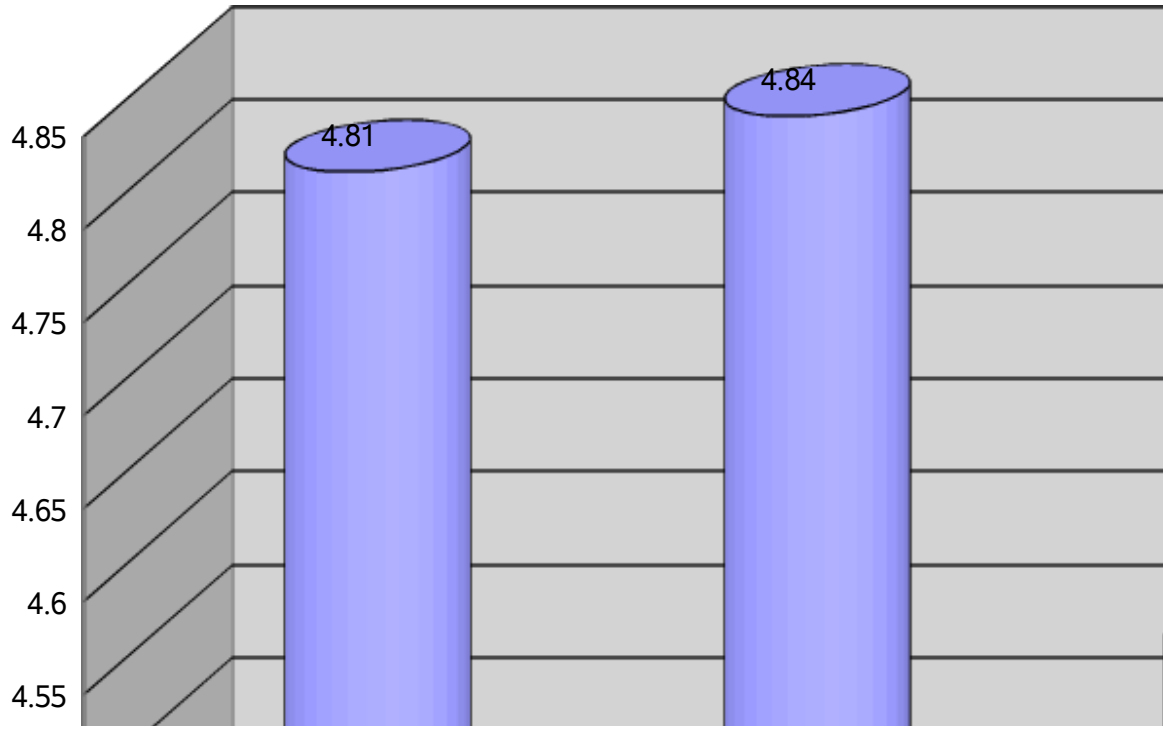
STATISTICAL ANALYSIS

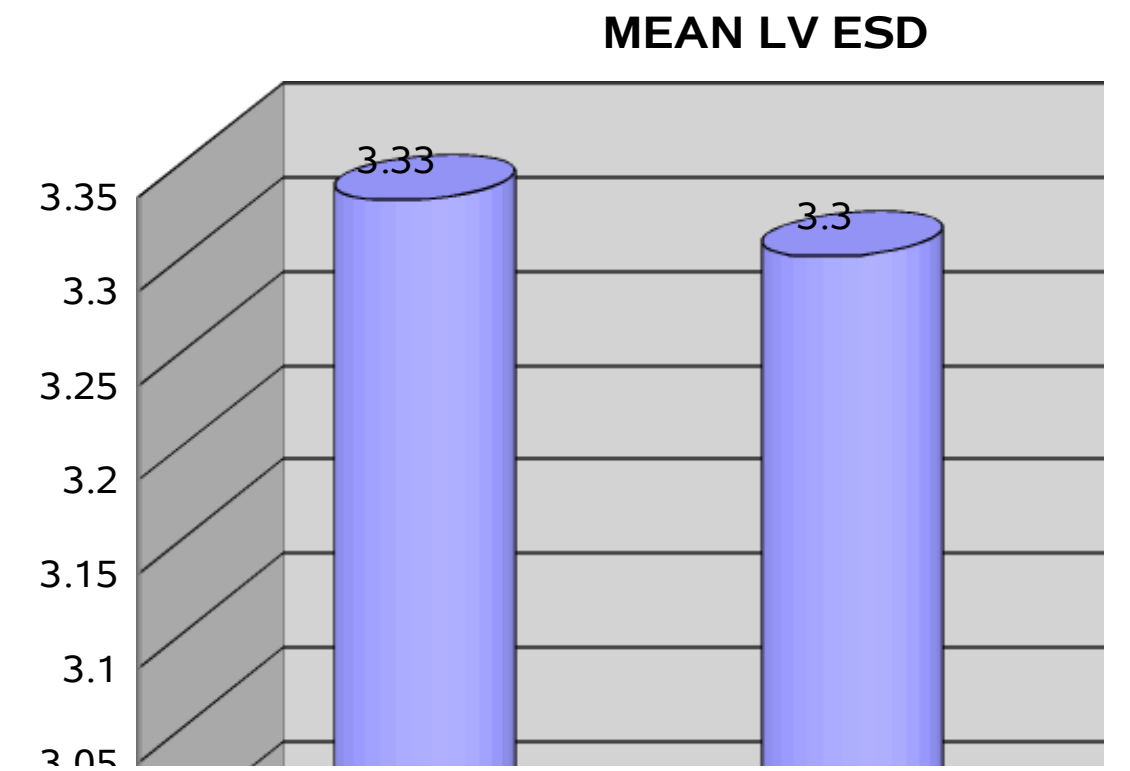
ıalysis

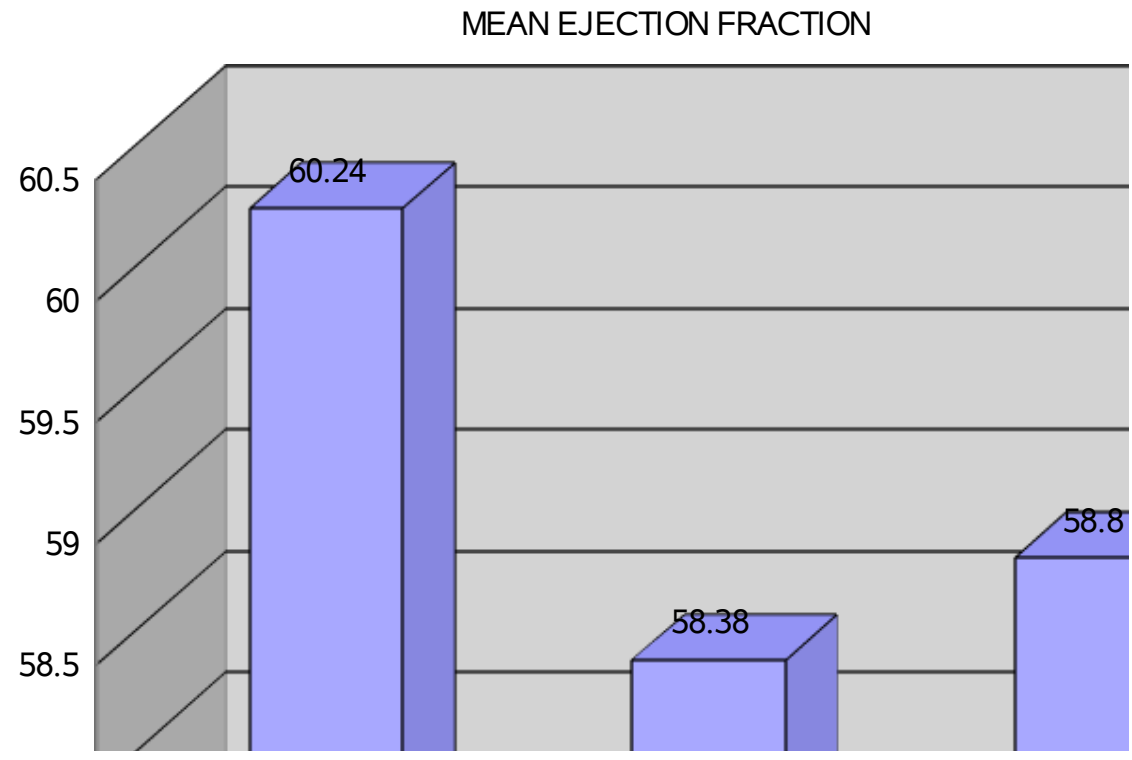
	Pre-eclamptic Pregnant Women		Normotensive pregnant women		Non Pregnant women		P Value		
	Mean	**SD**	**Mean**	**SD**	**Mean**	**SD**	**I Vs 2**	**1 Vs 3**	**2 Vs 3**
End disastolic dimension (cm)	4.81	0.5	4.84	0.44	4.55	0.28	NS	<0.001	<0.001
End systolic dimension (cm)	3.33	0.35	3.30	0.33	3.06	0.24	NS	<0.001	<0.001
FS	30.33	7.65	31.66	5.09	32.69	4.63	NS	NS	NS
EF	60.24	4.18	58.38	4.75	58.80	3.26	NS	NS	NS
Mitral inflowvelocity E/A	1.04	0.20	1.23	0.09	1.24	0.08	<0.001	<0.001	NS
IVRT (msec)	108.76	15.76	90	10.54	89	10.71	<0.001	<0.001	NS
DT (msec)	209.35	31.38	188.94	20.78	189.80	22.99	<0.001	<0.001	NS

Range OF LV SYSTOLIC AND DIASTOLIC INDICES

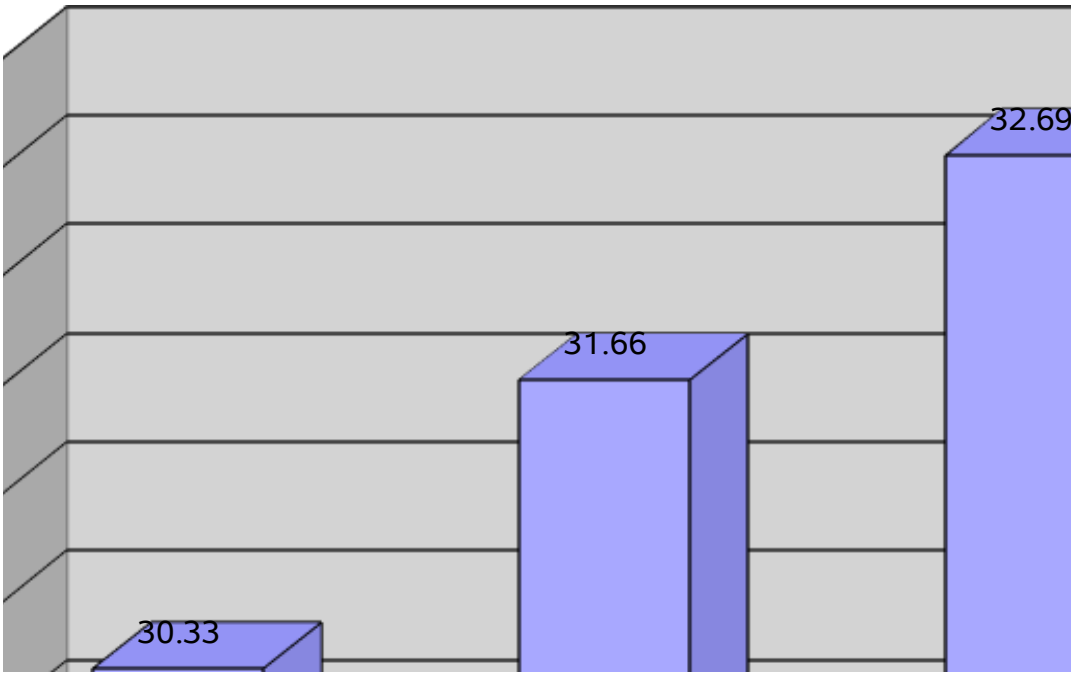
	Group - I	*Group - II*	*Group - III*
EDD	3.12 – 5.50	3.12 – 5.35	3.9 – 5
ESD	2.20 – 4.20	2.90 – 4	2.6 – 3.95
EF	51 – 67	50 – 65	50 – 65
FS	25 – 47.62	25 – 43.21	26.67 – 38.72
E/A	0.7 – 1.32	1.07 – 1.68	1.14 – 1.53
IVRT	86 – 136	73 – 106	76 – 110
DT	160 – 260	160 – 230	160 – 230



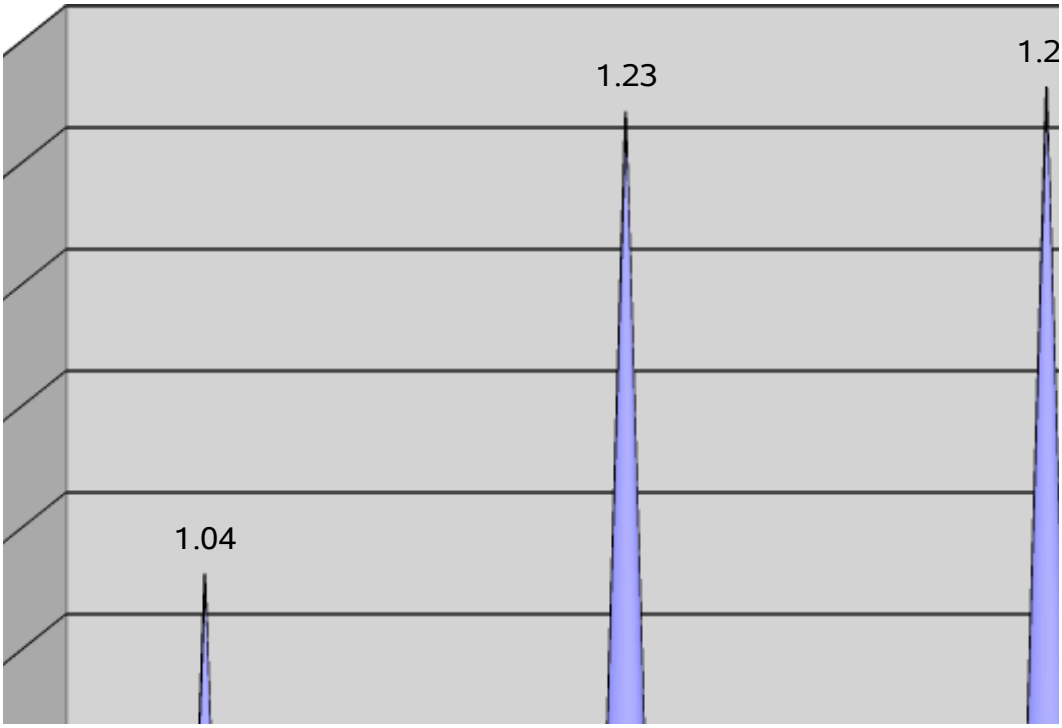

MEAN LV ESD
3.35
3.3
3.25
3.2
3.15
3.1
3.05
3.33
3.3

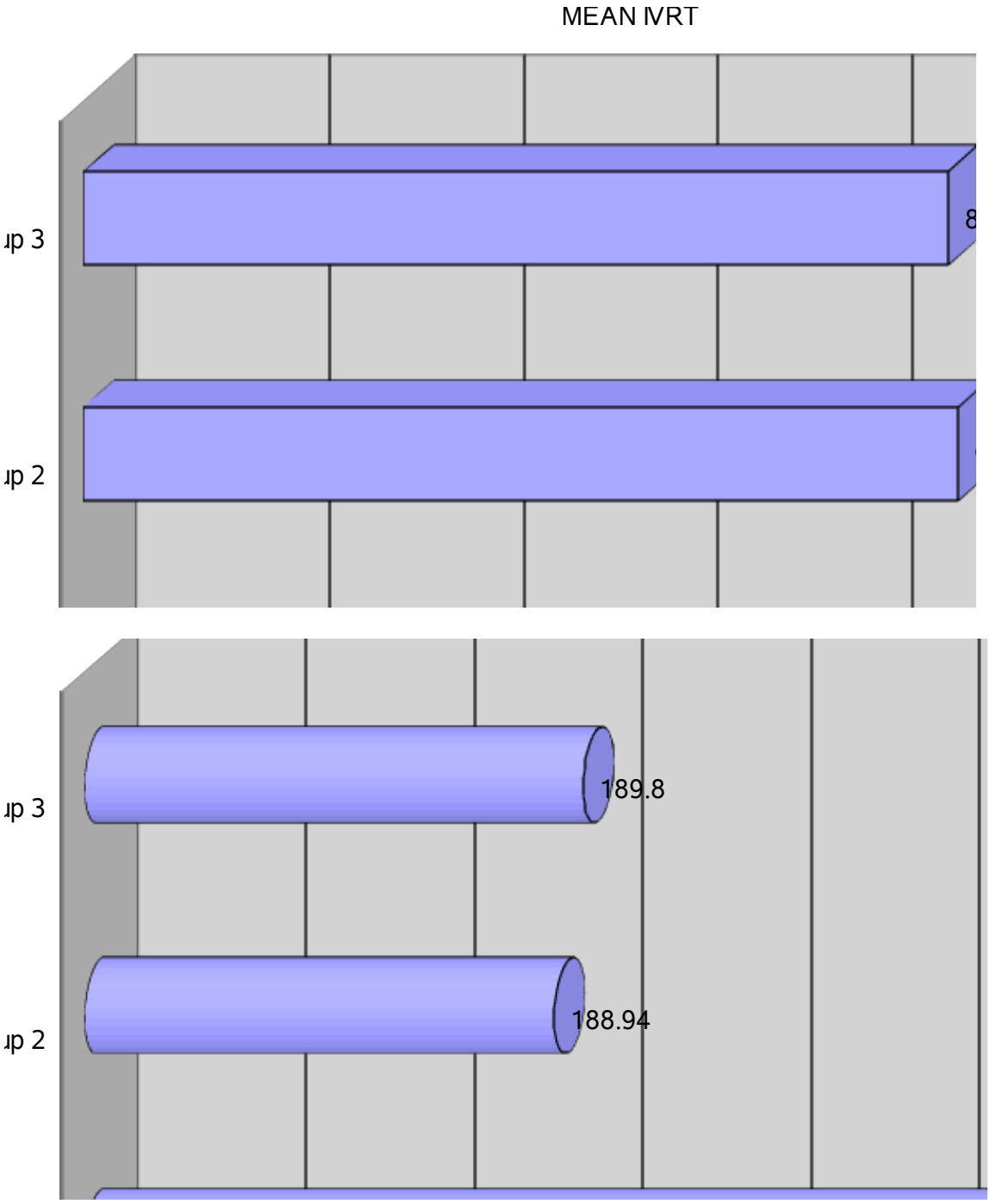

MEAN EJECTION FRACTION
60.5
60
59.5
59
58.5
60.24
58.38
58.8

MEAN FRACTIONAL SHORTENING



MEAN E/A RATIO




MEAN IVRT
ıp 3
ıp 2
ıp 3
189.8
ıp 2
188.94

DISCUSSION

The performance of left ventricle depends upon factors like

1. Preload (LV end diastolic volume or pressure)
2. Contractility (inotropic state)
3. Afterload (the résistance against which the LV has to contract).

In normal pregnancies there is marked increase in the blood volume that lead to significant volume overload of the heart. In preeclampsia there is an alteration in this haemodynamic pattern. Whether these haemodynamic changes influence the left ventricular function in preeclampsia remain unclear. In this study we attempted to address this question. J.Kuzniar et al 1983 in their study by echocardiography found that the mean end diastolic dimension and mean end systolic dimension were significantly elevated in pregnant patients above the mean of nonpregnant patients. In this respect, our data is comparable with the former study, though there is no significant difference of values between the preeclamptic and normotensive pregnant group. This may be due to very mild changes in the blood volume between the two groups (Silver and Seeback 1996).

The left ventricular performance during normal pregnancy was studied by Rubler and colleagues, Katz and associates using echocardiography. They did not find significant differences in percentage of FS between the subjects in the control group and those who are pregnant at term. J.kuzniar et al reported that left ventricular function as expressed by echocardiographic ejection phase indices did not differ substantially between the non pregnant patients and normal pregnant patients in the third trimester. In our study, the indices of left ventricular function were consistent with the above study showing no significant difference between the subjects in the control group and the normotensive pregnant group.

The findings of earlier studies on left ventricular contractile functions in hypertensive pregnant patients vary. Lim and Walters showed a diminished left ventricular performance evidenced by systolic time intervals using echocardiography.

Larkin and coworkers did not find significant differences in the functional status of the left ventricle, as evidenced by echocardiographically derived EF between normotensive and hypertensive pregnant patients. On the other hand Beneditti and coworkers, Rafferty and Bercowits using flow directed pulmonary artery catheter for the study of severe preeclampsia found a hyperdynamic left ventricular function in terms of the relationship between the stroke work index and pulmonary capillary wedge pressure.

J Kuzniar et al reported that the left ventricular systolic function was well preserved in most of the preeclamptic women. Only 7% of them showed depressed left ventricular performance as evidenced by percentage fractional shortening.

In our study it is found that in most of the preeclampsia patients the indices of left ventricular systolic function fell within normal range. These observations indicate good cardiac contractile function. Even though the left ventricle operate on the ascending limb of the left ventricular function curve only 4 out of 54 patients of this group had depressed LV systolic function. These findings correlate well with the J Kuzniar et al study.

The mechanism of this LV systolic dysfunction may be related to high afterload. None of the above studies addressed the issue of diastolic dysfunction.

Diastolic dysfunction is a well described clinical entity in essential hypertension especially in patients with left ventricular hypertrophy as the hypertensive heart has

less relaxing capacity. Williams C madie et al found that among the 25 preeclamptic women who developed pulmonary edema during antepartum and postpartum period, 12 (48%) showed diastolic dysfunction as evidenced by reversed E/A ratio with normal LV systolic function.
Desai DK et al reported that among the 16 preeclamptic patients who developed pulmonary edema in the antenatal period 12 (75%) patients showed LV diastolic filling abnormalities in significant proportion with preserved systolic function compared to control normotensive group.LV diastolic dysfunction as evidenced by reversed E/A ratio and prolonged IVRT and DT occurred in 17/54 (30%) of our preeclampsia patients. In our study it is interesting to note that only 2 out of 17 patients had LV hypertrophy.
Due to increased TPR in preeclampsia there is an increase in afterload and the left ventricle becomes stiff. This may be the possible mechanism by which diastolic dysfunction develops independent of LV hypertrophy.

Pulmonary edema occurs when the forces favouring fluid retention within the pulmonary intravascular space (Plasma oncotic pressure and interstitial hydrostatic pressure) are overwhelmed by those driving fluid from intravascular to the interstitial space (pulmonary capillary hydrostatic pressure and interstitial oncotic pressure). Accordingly decrease in the plasma oncotic pressure by relative (dilutional) or absolute reduction of plasma colloid by transfer of plasma colloid to the interstitial space (capillary leak) or increase in capillary hydrostatic pressure (volume expansion or cardiac dysfunction) is frequently the cause, singly or in combination. All these factors have been implicated in the genesis of pulmonary edema associated with pregnancy. Heart failure and pulmonary edema due to primary cardiac disease have traditionally been thought to be mediated by impaired left ventricular systolic function. Reduced systolic function results in incomplete left ventricular emptying, elevated left ventricular diastolic pressure and ultimately increased pulmonary capillary hydrostatic pressure. However recent evidence has indicated that over one third of cardiogenic pulmonary edema is due to abnormal left ventricular diastolic filling with normal or even hyperdynamic systolic function. In these cases increases in residual left atrial volume raise the left atrial pressure which is inturn transmitted to the pulmonary capillary bed. Impaired filling may be due to impaired ventricular filling in early diastole, reduced myocardial compliance (the stiff heart syndrome) or combination of the two.
Patients with heart failure due to impaired left ventricular contractile function have a relatively poor prognosis. When ventricular filling is impaired, relatively small increments in intravascular volumes translate into relatively large increases in left ventricular end diastolic and pulmonary capillary hydrostatic pressure. This state of affairs make these patients extraordinarily sensitive to volume loading and changes in capillary permeability.
MANAGEMENT OF HYPERTENSION IN PREECLAMPTIC PATIENTS

All these patients in group1 were put on antihypertensive drugs like alpha methyldopa and nifedipine. All the cases of mild preeclampsia were put only on alpha methyldopa 250mg eight hourly and advised atleast 8 hours of sleep in the night and 2 hours of rest in the afternoon in left lateral position. With this treatment blood pressure has been controlled to systolic pressure ranging 110 to 120 and diastolic pressure ranging from 70 to 84 mmHg. In severe preeclampsia group all patients were on tab methyldopa 250mg 8th hourly and cap nifedipine 10ms 8^{th} hourly. One patient showed uncontrolled blood pressure and it was fluctuating. Blood pressures of other patients were reduced

from severe to mild degree. Their proteinuria also showed improvement from 3+ to 1+.

PERIPARTUM MANAGEMENT OF PATIENTS WITH LV DYSFUNCTION:

Among the 17 patients with LV dysfunction 10 patients had mild preeclampsia and 7 patients had severe preeclampisa. Since the development of pulmonary edema and cardiac failure are more common during labour and immediate postpartum, careful monitoring and appropriate management of these patients is crucial. Among the 10 mild preeclamptic patients with LV dysfunction 6 patients had normal vaginal deliveries. 4 patients went in for LSCS, 2 indicated by previous LSCS and 2 for fetal distress.

Among the 7 patients with severe preeclampsia 4 patients had normal vaginal deliveres. 1 patient developed imminent eclampsia and was started on magnesium sulphate regimen and her pregnancy was terminated by cerviprime induction. She expelled a dead born fetus weighing 650gms. Other 2 patients had LSCS one for fetal distress and the other indicated by breech presentation.

All these patients were admitted in Intensive Care Unit (ICU) during labour. Sedation was given with Injection Diazepam 10mg to alleviate pain, anxiety which may lead to tachycardia. They were kept in propped up position. Pulse rate, temperature and respiratory rate were recorded ½ hourly. B.P. and unrine output recorded hourly. Spo_2 was monitered with pulse oximeter, and oxygen by mask was administered at the rate of 4-6 litres/min, when Spo_2 falls below 90%. Frequent auscultation of lung bases was done. IV fluids when used was maintained at the rate of 75-100ml/hour so as to maintain an adequate urine output of 1ml/kg/hour.

Jugular Venous Pressure (JVP) was measured 4^{th} hourly to detect fluid overload. Ideally patients with Lvdysfunciton should be monitored with Central Venous Pressure (CVP) to detect changes in Pulmonary Capillary Wedge Pressure (PCWP). In the absence of invasive hemodynamic monitoring JVP can be used. Normal right atrial pressure is 0 to 5 mm Hg which equals CVP thereby indicating PCWP. Since JVP reflects changes in right atrial pressure it can be used in the absence of invasive hemodynamic monitoring. Normal JVP is up to 3 cms measured from the Angle of Louis. Every 1 cms increase in JVP is equal to 0.68mmHg increase in CVP. Even 1 cms increase in JVP indicates significant volume overload. Injection Frusemide 40mg i.v is given when there is fluid overload as indicated by elevated JVP and basal lung crepitations.

Two patients with severe preeclampsia had severe uncontrolled B.P. during labour and nitroglycerine drip was started at the rate of 2.5 µg/kg body weight and dosage titrated to maintain systolic B.P around 120-130 mmHg and diastolic B.P around 80-90mmHg. Infusion was stopped when systolic B.P was <100mmHg. Since Nitorglycerine is a venodilator it reduces preload thereby preventing pulmonary edema. One patient with severe preeclampsia had uncontrolled B.P with tachycardia, during labour. She was treated with i.v. labetolol starting with a 20mg bolus i.v and if not effective within 10 minutes followed by 40mg then 80mg every 10minutes but not to exceed total dose of 220mg.

For mild preeclamptic patients tablet alphamethyl dopa 250mg 8^{th} hourly was continued and their B.P. was under control. Since tachycardia reduces the diastolic filling time and precipitates pulmonary edema, the control of heart rate has a key role in the

management of patients with LV dysfunction.

For patients who underwent LSCS, the mode of anaesthesia was discussed with anaesthetist. Since GA may cause sudden hypertension and tachycardia due to sympathetic stimulation caused by tracheal intubation, regional anaesthesia was used in preference; These patients were monitored in ICU for 72 hours postpartum, and then transferred to high risk postnatal ward.

None of the 17 patients developed complications like pulmonary edema or cardiac failure. Their B.P returned to normal within 15 days postpartum.

FOETAL OUTCOME:

Among the 53 patients who had live child, 28 were female children and 25 were male children. The average weight of the babies was 2.6 kg and the finding of LV diastolic dysfunction was found to have no corrletation with foetal outcome.

FOLLOW UP:

Out of the 17 patients who showed diastolic dysfunction, 2 patients were lost for follow up. 15 patients were followed up with echocardiography after a mean period of 28 days. They were found to have normal diastolic function.

Characteristic of the patients who showed LV diastolic dysfunction

S.No	*Name/ IP No*	*Clinical Data*			*Systolic Function*		*Diastolic Function*			*Parity*	*Mode of Delivery*	*Fetal Weight (kg)*
		Age	*BP*	*U/A*	*EF*	*FS*	*E/A ratio*	*IVRT msec*	*DT msec*			
1.	Bhuvaneshwari/1333	21	170/110	3+	56	27.22	0.9	135	243	Multi para	Lab. Nat	2.7
2.	Jabin/ 11413	21	180/110	3+	52	47.62	0.8	127	245	Primi para	DeadBorn	0.65
3.	Tamilselvi/13079	25	150/100	1+	54	24.37	0.7	136	248	Multi para	Lab. Nat	2.5
4.	Jayalakshmi/6247	18	150/100	1+	57	31.19	0.8	135	250	Primi para	Lab. Nat	2.4
5.	Mohana.6397	26	140/90	Trace	60	37.25	0.7	128	246	Primi para	Lab. Nat	2.9
6.	Poongodi/6705	18	180/110	3+	53	38.46	0.8	125	241	Multi para	LSCS	3
7.	Alamelu/10140	35	160/110	2+	62	25.50	0.9	124	244	Primi para	Lab. Nat	2.2
8.	Vidhya/10286	19	160/110	3+	56	39.22	0.8	12	239	Multi para	LSCS	2.5
9.	Roshini / 10521	35	160/110	2+	51	26.37	0.8	120	244	Primi para	Lab. Nat	2.6
10.	Aysha/ 10571	28	150/100	1+	55	30.14	0.7	135	242	Primi para	Lab. Nat	2.4
11.	Nadiya/ 10680	22	140/90	1+	0	33.33	0.8	123	238	Primi para	LSCS	3

S.No	*Name/ IP No*	*Clinical Data*			*Systolic Function*		*Diastolic Function*			*Parity*	*Mode of Delivery*	*Fetal Weight (kg)*
		Age	*BP*	*U/A*	*EF*	*FS*	*E/A ratio*	*IVRT msec*	*DT msec*			
12.	Nirmala/ 10723	20	150/100	1+	60	32.17	0.9	134	250	Primi para	Lab. Nat	2.1
13.	Guna / 10721	26	150/100	1+	58	32	0.8	140	260	Multipara	LSCS	3.4
14.	Mahalakshmi/ 10922	20	170/110	3+	65	36.36	0.7	120	258	Primipara	Lab. Nat	2.5
15.	Chitra/ 11139	25	150/100	1+	23	32.28	0.8	135	234	Multi para	Lab. Nat	3
16.	Maheswari/ 11121	20	150/90	1+	64	31.25	0.8	132	245	Multi para	LSCS	3.1
17.	Pasupathy. 11356	18	140/100	1+	55	25	0.7	130	240	Multi para	LSCS	2.5

CONCLUSION

It was observed that

1. LV systolic function was within normal limits in most of the preeclamptic patients
2. Statistically insignificant number of patients 4/54 (7.4%) had LV systolic dysfunction p value > 0.05.
3. Statistically significant LV diastolic dysfunction was seen in 17/54 (31.48%) of preeclamptic patients p < 0.001.
4. LV hypertrophy was a rare finding occurring only in two patients of the study group.
5. Appropriate management of the patients who showed LV dysfunction prevented pulmonary edema and cardiac failure.

CLINICAL IMPLICATIONS:

While overt cardiac failure associated with preeclampsia is rare subclinical LV dysfunction is being increasingly recongnised as in this study. Abnormal diastolic function is the most common cause of heart failure in patients with normal LV systolic function. This can be easily diagnosed by echocardiography with evidence of abnormal relaxation, increased filling pressure and decreased compliance as well as normal LV dimensions and preserved LV ejection fraction.

Inspite of having high afterload, the systolic function is well preserved in preeclampsia. But in contrary diastolic dysfunction was observed in significant number which is a predisposing factor for pulmonary edema. Even in asymptomatic patients the presence of diastolic dysfunction portend poor clinical outcome. Maintaining normal diastolic function may be the key to cardiac immortality. In the normal population with normal diastolic function, filling pressures increases slightly with exercise. It seems that diastolic dysfunction leads to development of exercise induced high filling pressures. In these patients cardiac output is increased at the expense of increased filling pressures. The sustained cardiac changes during pregnancy are similar to acute changes reported for moderate to strenuous exercise. Preeclamptic patients with grade 1 diastolic dysfucntion are asymptomatic as long as the diastolic filling period is sufficiently long to accommodate the delay in myocardial relaxation. The key to management is the prevention of tachycardia and the control of factors that further aggravates diastolic dysfunction which include management of hypertension, control of obesity, management of diabetes mellitus if associated. During labour betablocker therapy is helpful in minimising tachycardia.

It is to be expected that the additional stress caused by labour uncontrolled fluid therapy or general anaesthesia may be potentially dangerous in these patients with subclinical left ventricular dysfunction. It may also be of interest to know that left ventricular dysfunction associated with preeclampsia is reversible. These may shed light on the pathogenesis of peripartum cardiomyopathy. There is also a link between preeclampsia and future cardiovascular disease.(Journal of Clinical Endocrinology and Metabolism vol89;2004). Women with a history of preeclampsia demonstrate altered expression of angiogenesis related proteins and increased insulin resistance more than one year post partum. These factors may contribute to their risk of future cardiovascular disease. Further studies are required addressing this issue.

PROFORMA

NAME : AGE : IPNO : DOA:

SOCIAL STATUS OBSTETRIC HISTORY : LMP: EDD:

PAST HISTORY :

FAMILY HISTORY :

PHYSICAL EXAMINATION – Wt : kg BUILD : NUTRITION : ANAEMIA

EDEMA LEGS : PULSE: /mt, BP: mmHg, JVP: CVS: RS

OBSTERIC EXAMINATION:

P/A:

P/V:

INVESTIGATION: URINE-ALBMIN SUGAR- DEPOSIT –

HB- gm/dl, BLOOD SUGAR - mg/dl, BLOOD UREA - mg/dl

SERUM CREATNINE - mg/dl SERUM URIC ACID -mg/dl

BT - , CT- PLATELET COUNT -

LFT:

FUNDUS EXAMINATION:

TREATMENT :

MANAGEMENT DURING LABOUR

MODE OF LABOUR: APGAR OF BABY :

POSTNATAL PERIOD :

ECHOCARDIOGRAPHIC DATA

M-mode :

AORTA:	cm,	LA :	cm,				
LV-ESD :	cm,	LVEDD :	cm	EF :	%		
LVPW :	cm,	IVS :	cm,	FS:			

2D & DOPPLER:

MV:	AV:	PV:	TV:				
Evel -	A vel	E/A:	DT:	ms,	IVRT:	ms	

OTHER DEFECTS:

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