DISCUSSION

Pregnancy is a physiological condition of increased stress and adaptation, characterized by significant changes in breathing, mechanics of respiration and lung volumes, as a result of the hormonal stimuli beginning at conception; the increasing oxygen requirements and the mechanical effects of the enlarging uterus in later weeks. Maternal respiratory alterations will naturally influence fetal blood gases and acid base balance, as the mother and her fetus represent a biologic unit whose direct gas and substrate exchange takes place in the placenta. Any factor or respiratory conditions adversely affecting the lung functions is known to produce hypoxemia and maternal hypoxemia is associated with impaired fetal growth and development. Thus the respiratory problems in pregnancy must be approached with an understanding of the unique, interdependent physiological status of the mother and the fetus.

The present work is taken up in an attempt to elucidate the relevance of the normal physiological changes and the changes occurring in pregnant women with respiratory complaints / problems/history to their perinatal outcome, with special emphasis on the neonatal birth weight. One hundred and fifty pregnant women were included in this study and they were grouped as group I-P (n=93) consisting of normal pregnant women and group II-P (n=57) consisting of pregnant women with respiratory complaints/problems/history of active or passive smoking. The same subjects in their postpartum stage were termed as group I-PP (control group) and group II-PP (experimental group).

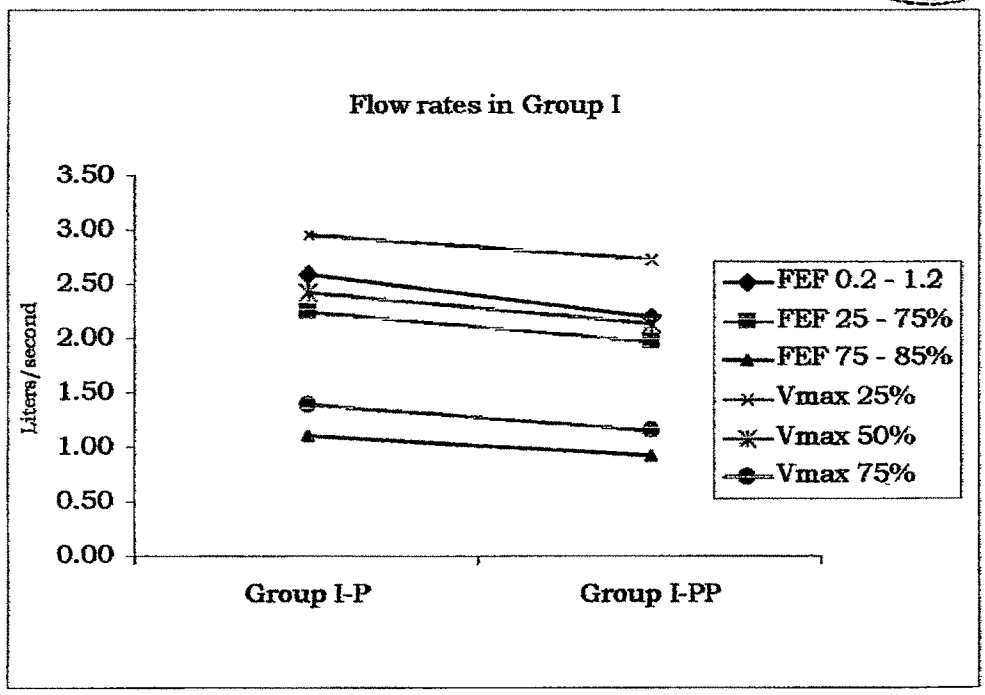
The physical characteristics of the pregnant subjects during their mean 32 weeks gestation in both the groups are presented in table 1. The mean age recorded was 23.75 ± 3.84 years and 23.56 ± 3.00 years in group I-P and group II-P respectively. The

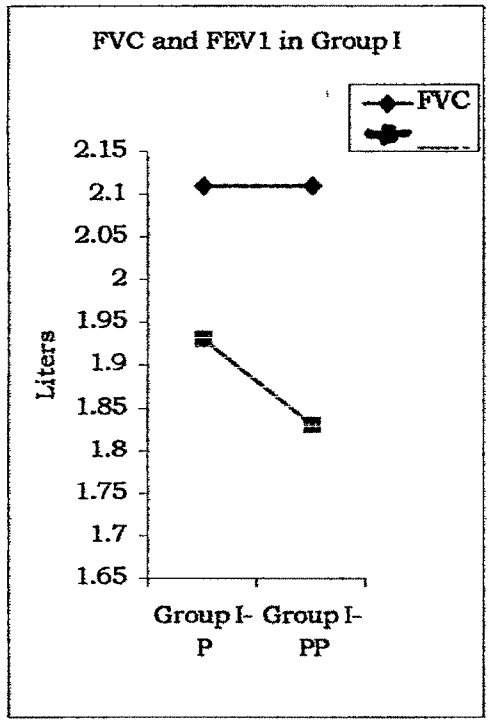
difference of mean age between the two groups derived statistically was not found to be significant (P>0.1). The mean height noted was 149.94 ± 5.31 and 151.67 ± 5.50 (cm) in group I-P and group II-P respectively was found to be statistically insignificant (P>0.05). The mean weight obtained was 49.67 ± 9.00 and 48.67 ± 7.29 (kg) in group I-P and group II-P respectively with no statistically significant difference (P>0.1). Thus, both group I and group II were well comparable with respect to their mean age, height and weight.

Table 2 shows the mean ± SD values of physiological parameters like arterial systolic blood pressure and diastolic blood pressure in mm Hg, pulse rate in rate/min, oral temperature in Fahrenheit and Hb in gm % at their mean 32 weeks of gestation. The subjects were recruited between 29th and 35th week of gestation i.e. third trimester, the mean age being 31.94 ± 2.48 and 31.73 ± 2.40 in group I-P and group II-P respectively. The mean difference calculated between the two groups for the mentioned parameters were found to be statistically insignificant and these values correspond to the associated physiological variations expected in pregnancy.

Earlier cross-sectional and longitudinal lung function studies nvolving all the three trimesters of pregnancy have reported the airways function to be unaltered throughout pregnancy (Baldwin et al, 1977; Milne et al, 1977; Alaily and Carrol, 1978 and Das et al, 1991). Considering this fact the forced spirometry was conducted only once during pregnancy in the third trimester while their postpartum values were considered as their controlled values (non-pregnant data). A graphical representation of lung functions parameter conducted in both groups as a part of present study is shown in chart 1 and 2. Evidently higher values are obtained in both the groups during pregnancy as compared to their postpartum values but the difference is not significant.

CHART 1





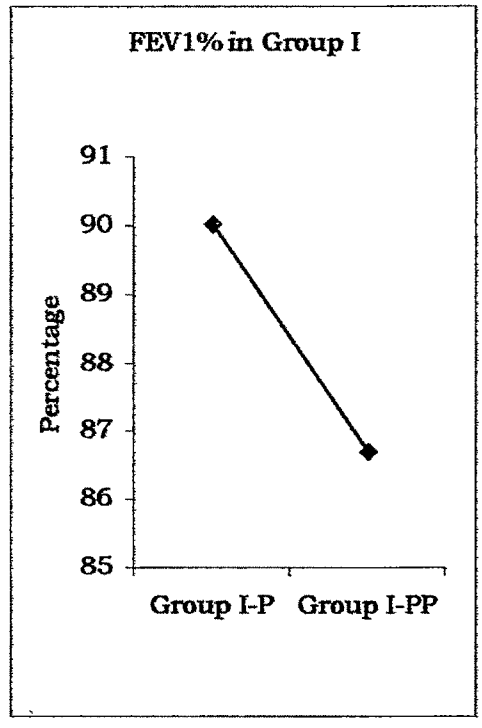
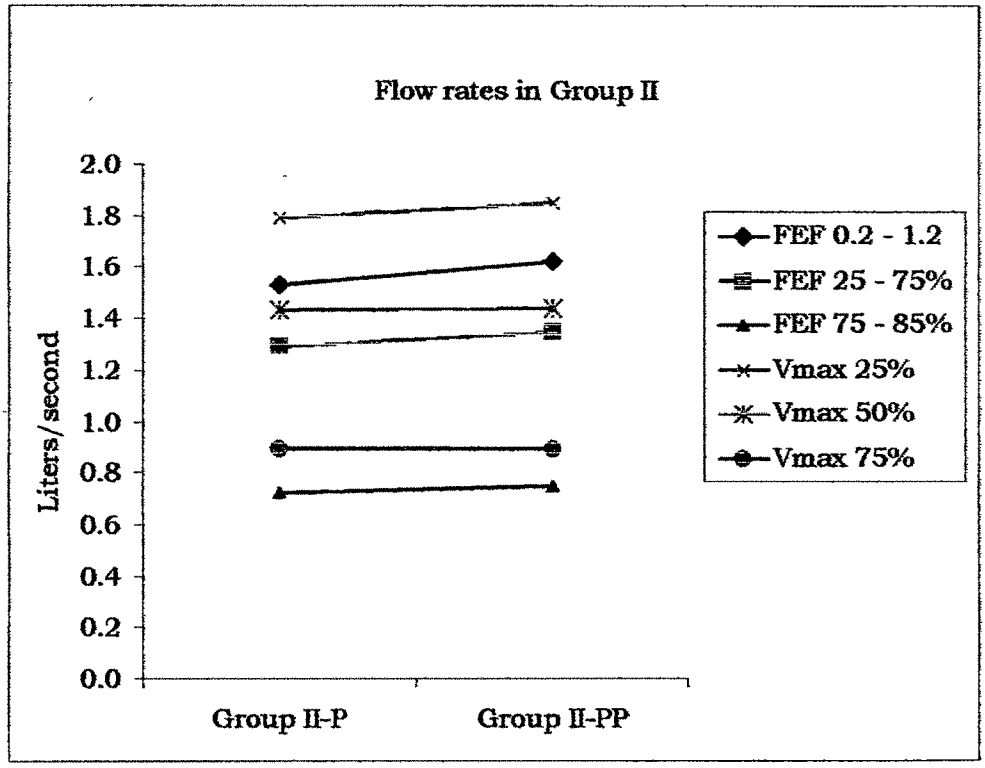
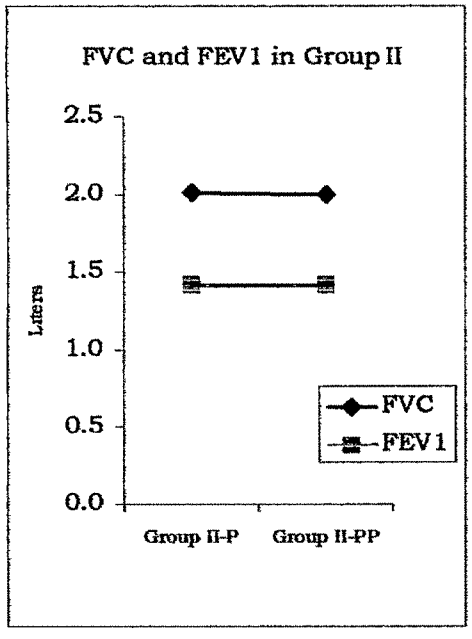
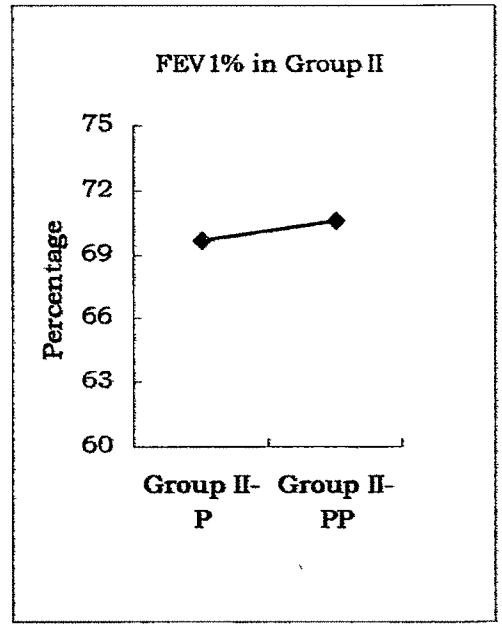


CHART 2


Flow rates in Group II
2.0
1.8
1.6
1.4
1.2
1.0
0.8
0.6
0.4
0.2
0.0
Liters/second
Group II-P
Group II-PP
FEF 0.2 - 1.2
FEF 25 - 75%
FEF 75 - 85%
Vmax 25%
Vmax 50%
Vmax 75%


FVC and FEV1 in Group II
2.5
2.0
1.5
1.0
0.5
0.0
Liters
Group II-P
Group II-PP
FVC
FEV1


FEV1% in Group II
75
72
69
66
63
60
Percentage
Group II-P
Group II-PP

The mean FVC recorded during pregnancy was 2.11 ± 0.28 and 2.014 ± 0.43 in group I-P and group II-P respectively, while in postpartum they were 2.11 ± 0.41 and 2.005 ± 0.34 in group I-PP and group II-PP respectively. There was some increase observed in mean FVC during pregnancy, but whatsoever increase occurred was not significant. The insignificant increase in mean FVC was also noted during pregnancy Root and Root, 1923; Enright et al, 1935; Widlund et al, 1945 and Gazioglu et al, 1970. The present observation of unaltered FVC can probably be explained as follows.

It has been shown by Kountz and Alexander (1934) that the VC in patients with emphysema can be increased by elevating the diaphragm by means of a specially designed abdominal belt. Thus it can be seen that an elevated diaphragm does not invariably reflect reduced VC, as commonly supposed, but may actually tend to increase it. Such a factor could be operating in pregnancy. The mechanism by which this preservation is brought about is not known but may be associated with reduction in the volume of residual air as suggested by Hasselbach (cited in Thomson and Cohen, 1938).

The relative mobility of the thoracic cage has been shown to be one of the factors for preservation of VC. Thomson & Cohen (1938) pointed out that there is more mobility in the strenomanubrial joint during pregnancy than in the puerperium. This may be due to "relaxin", the hormonal substance shown first by Hisaw (1925) to affect the symphysis pubis and later purified by Fevold et al, 1930. Further evidence that change in the chest configuration is possibly under hormonal control is the observation that subcostal angle begins to widen, as did the symphysis pubis in the studies of Abramson, et al (1934) earlier in pregnancy than could be accounted for satisfactorily by mechanical pressure on an enlarging uterus.

The possible effect of decreasing VC due to decrease in vertical diameter of the thoracic cage as a result of upward movement of diaphragm is compensated by an increase in width of thoracic cage. By the end of the third trimester the average circumference of the maternal thoracic cage is increased by 5-7 cm, this includes both the transverse and anteroposterior diameters. An increase of the substernal angle from 68^0 to 103.5^0 is also noted (Thomas and Cohen, 1938; Beck and Rosenthal, 1955; Marx and Orkin, 1958 and Hellman and Pitchard, 1971).

Despite alteration of the resting position of the diaphragm by pressure from the uterus, diaphragmatic motion is not impaired. Diaphragmatic excursions with tidal breathing were actually greater in pregnancy than in puerperium. During fluoroscopic examination of the thorax, the diaphragm, although elevated during quiet respiration, were seen to be capable of moving with apparently normal excursion during forced inspiration and expiration, although at a somewhat higher level than in the non pregnant state (McGuinty, 1938; Stewart, 1951 and Weinberger et al, 1980). However, relaxation of abdominal musculature and the ligamentous attachment of the ribs may result in unaltered lung volumes and chest mobility (Bonica, 1967).

Again, the preservation of FVC could be achieved by a corresponding increase in the IC and IRV despite of increase in intra-abdominal pressure exerted by gravid uterus (Marx and Orkin, 1958; Moya, 1963).

The measurement of FEV_1, $FEF_{0.2-1.2}$ and $V_{max\ 25\%}$ are important to detect large airway function. The measurement of FEV_1 and $FEF_{0.2-1.2}$ in pregnancy have not shown any significant change (chart 1), when compared to their postpartum values. The little change observed in both the parameters remained within normal range. Therefore, from the above observation, it is seen that the large airway function during

pregnancy almost remains unaffected. Similar observations were made by Cameron et al, 1970; Sims et al, 1976; Milne et al, 1977; Garrad et al, 1978 and Das et al. 1991). However there exists a controversy regarding the changes in airways resistance in pregnancy measured during quiet respiration. It has been variably shown to increase (Briscoe and Dubois, 1958 and Newhouse et al, 1964), decrease (Rubin et al, 1956) or remains unchanged throughout pregnancy (Cugell et al, 1953).

Pregnancy is associated with enlarged gravid uterus causing upward displacement of diaphragm and it tends to increase the flow resistance of airways by reduction in resting lung volume that is FRC (Briscoe and Dubois, 1958). Similarly it has been shown that reduction in P_ACO_2, such as occurs in pregnancy, leads to an increase in airways resistance (Newhouse et al, 1964) caused by bronchial smooth muscle constriction. Since in this study we have not found any significant change in large airway function by assessing timed VC and $FEF_{0.2-1.2}$, therefore, there may be a mechanism operating in pregnancy to dilate the bronchi. Progesterone is known to have a widespread smooth muscle relaxing effect on number of structures (Hytten and Leitch, 1971). In addition there is evidence that progesterone increases β adrenergic activity (Raz et al, 1973), which is present in large pulmonary airways. An increase in β adrenergic activity, however could cause bronchodilation and an increase in conductance and decrease in resistance. Thus, lack of change in large airway function in pregnancy may be related to the opposing effects of hypocapnia and increased β adrenergic activity induced by hormone progesterone.

Obstruction to airflow in the large airways was considered to be a simple phenomenon investigated by the techniques described earlier. However, the work of Macklem and Mead (1967) has shown

that airway resistance is partitioned into large and small airways. Hogg et al (1968) stated that there may be extensive disease or dysfunction of the smaller airways before it becomes detectable by the standard methods for assessment of airways function. It is now possible to investigate the function of these peripheral or smaller airways with the use of measurements such as $FEF_{25-75\%}$, $FEF_{75-85\%}$, $V_{max50\%}$ and $V_{max75\%}$, which is an "effort independent" portion of FVC maneuver. Studies so far have found this parameter too, remains unchanged (Gazioglu et al, 1970; Baldwin et al, 1977 and Das et al, 1991). Our results relate to above studies (chart). Thus, inspite of compressed lung bases and increased angulation of bronchi (Rubin et al, 1956), the peripheral airways function remains unaffected during pregnancy. It is postulated that this may be due to increase in airways cross sectional areas (Gee et al, 1967). Progesterone is known to have widespread smooth muscle relaxing effect and causes increase in airways cross sectional areas. Again Woolcock et al, 1969 suggested that β adrenergic activity of progesterone is more marked in peripheral airways as opposed to large airways and causes easy flow of air. It would seem likely that the constancy of large and small airways function in pregnancy is a balance between factors tending to increase and those tending to decrease airways resistance.

It has long been recognized that pregnancy can alter lung function and the natural maternal history of certain pulmonary diseases. Often preexisting pulmonary diseases are present when pregnancy occurs in most of the cases. Patients with disease of respiratory system generally present symptoms or an abnormality of chest radiography or both. A set of diagnostic possibilities are often suggested by the initial problems at presentation, including particular symptom(s) and appearance of any radiographic abnormalities. The differential diagnosis is then refined on the basis of additional

information gleamed from physical examinations, pulmonary function tests, additional imaging studies and bronchoscopic examination.

Clinically dyspnea and cough are the primary presenting symptoms for patient with respiratory system disease. The presence of sputum accompanying the cough often suggests airway disease. Rarely hemoptysis and chest pain are presented. Additionally, historical information about the risk factor for lung disease can be explicitly explored. A history of current and past smoking, especially of cigarettes, should be sought from all patients, as COPD, neoplasia, spontaneous pneumothorax etc. are associated with smoking. A history of second hand (passive) exposure to smoke, whether at home or workplace should be investigated, as it is also considered a risk factor for neoplasia or an exacerbating factor for airway diseases. Further detailed information of occupational, personal and home environment may prove helpful too.

The two major patterns of abnormal ventilatory functions measured by spirometry are restrictive and obstructive pattern. In obstructive pattern the hallmark is reduction in expiratory flow rates. Sometimes in early obstructive disease; which originates in the small airways, FEV_1/FVC may be normal. The only abnormality noted on routine findings may be depression of $FEF_{25-75\%}$ and an abnormal configuration in the terminal portion of the forced expiratory flow volume curve. While, with fully established disease, the ratio FEV_1/FVC is decreased as is $FEF_{25-75\%}$.

Based on the past history of common respiratory symptoms, complaints, past history of obstructive lung disorder and that of smoking and passive smoke exposure an inclusion criterion was designed. History of acute bronchitis, asthma (clinically stable with no requirement or minimal requirement of medications), smoking and passive smoke exposure was sought. The smoking history included

number of years of smoking, the intensity (cigarettes/bidis per day) and in case of passive smokers the exposure to occupational or vocational determinants and concerned environmental factors in terms of ventilation as well as duration of exposure per day was noted. The pathophysiological sequel of the above conditions or factors is airways function impairment, an airways obstruction with a universal finding of hypoxemia.

The hallmark of asthma is a reduction in airways diameter brought about by contraction of smooth muscle, vascular edema of the bronchial wall and thick tenacious secretions. The net result is an increase in airway resistance, a decrease in forced expiratory volumes and flow rates, hyper-inflammation of the lungs and thorax, increased work of breathing, alterations in respiratory muscle function, changes in elastic recoil, abnormal distribution of both ventilation and pulmonary blood flow with mismatched ventilation-perfusion ratios.

Acute bronchitis is an inflammatory condition of the trancheo-bronchial tree. Cough, hoarseness and fever are the most common symptoms. Soar throat and rhinitis resolves within 3-4 days, but cough persists and may last as long as 3 weeks with sputum production. Chronic bronchitis is defined as a productive cough from excessive secretions for at least 3 months of the year from two consecutive years and is associated with other chronic pulmonary disease like COPD.

Bronchitis results in airways narrowing which is often associated with both an increase in airways resistance and a diminution in maximal expiratory flow rates. There are occasions in which a normal or only slightly elevated airways resistance is accompanied by reduced MEFR. Under such conditions, an increase in the dynamic collapsibility of intra-thoracic airways during forced exhalation is a possible explanation.

COPD is defined as a condition, in which there is chronic obstruction to airflow, small airways being major site of airway obstruction. The currently accepted use of this term is in the description of chronic bronchitis, emphysema or small airway disease, entities that coexist in the same patient. In the overwhelming majority of cases COPD is directly attributed to a history of cigarette smoking.

Cigarette smoking is the most commonly identified correlates with both, chronic bronchitis during life and extent of emphysema at postmortem. Experimental studies have shown that prolonged cigarette smoking impairs ciliary movement, inhibits function of alveolar macrophages and leads to hypertrophy and hyperplasia of mucus secreting glands. Inhaled cigarette smoke can produce an acute increase in airways resistance due to vagally mediated smooth muscle constriction, presumably by way of stimulating submucosal irritant receptors. Increased airway responsiveness is associated with more rapid progression in patients with chronic airways obstruction. Obstruction of small airways ($\leq$ 2mm) is the earliest demonstrable mechanical defect in young cigarette smokers, at times in the presence of normal FEV_1 (Buist,1989).

Cigarette smoking has adverse effects on female reproductive life. Evidence suggests that nicotine and other alkaloids in tobacco, not only inhibit the formation of estrogen but also accelerates its catabolism by hydroxylation (Benowitz, 1988). However, both estrogen and progesterone may be required to achieve large ventilatory changes observed during pregnancy (Regensteiner et al, 1989). Therefore in spite of smooth muscle relaxation and the bronchodilator activity of progesterone, cigarette smoking causes deterioration of airways function, similar to the effect observed in non-pregnant smokers (White and Froeb, 1980 and Das et al. 1991).

Passive smoking or sidestream smoking or second hand smoking appears to be hazardous in its own right (Jarvis et al, 1983). Levels of various smoke contaminants have been shown to be higher in sidestream than in mainstream smoke (Martin and Bracken, 1986). The passive smoking associations may be modified by some unknown factors like socioeconomic status, which might be related to poor housing and ventilatory conditions causing increased exposure to passive smoke and occupational exposure. Several studies have shown that respirable particulate load in any household directly proportional to the number of cigarette smokers living in the house (Yerushalmy et al, 1962; Rubin et al, 1986 and Martin and Bracken et al, 1986). When smokers reside in the household, the problem is potentially greater. Increase in prevalence of respiratory illness and reduced level of pulmonary function measured with simple spirometry have been found in children of smoking parents in number of studies (Yerushalmy, 1966, Underwood et al, 1967 and Terri and Gold, 1969)

In the present study the dynamic airway functions were recorded in a group I-P (n=93) and were compared with those recorded in group II-P (n=57). The mean ± SD of FVC, FEV_1 and $FEV_{1\%}$ of two groups, their unpaired "t" value and significance level (set at 5% level) is presented in table 3. The mean FVC values recorded were 2.11 ± 0.28 airway diseases 2.01 ± 0.43 (liters) in group I-P and group II-P respectively. The mean difference between the two groups evaluated statistically was not found to be significant ($P>0.1$). An insignificant but a little reduction is found in group II-P. As this group is associated with airways obstruction, the change is related to narrowing or closure of small airways, because 20% of FVC is thought to be related to these structures (Das et al, 1991). Loss of elastic recoil pressure of the lungs may also cause the FVC to fall a little. Similar FVC findings during the third trimester of pregnancy were reported like 2.1 ± 0.24 liters (Puranik et al, 1994) and 2.07 ± 0.28 liters (Chhabra et al,

1998). Das et al, (1991) have recorded mean FVC value as 3.88 ± 0.48 and 3.76 ± 0.51 in non smokers and light smoker pregnant women respectively. The significant difference in their study may be due to more number of cigarettes smoked and years involved.

The mean FEV_1 noted were 1.93 ± 0.26 and 1.41 ± 0.37 liters in group I-P and group II-P respectively. The mean difference calculated between the two groups was found to be significantly lower in group II-P as compared to group I-P ($P<0.001$). Mean $FEV_{1\%}$ calculated were 90.03% and 69.67% in group I-P and group II-P respectively. The statistical mean difference was also found to be significantly and abnormally reduced in group II as compared to group I ($P<0.001$). Significant reduction in both FEV_1 and $FEV_{1\%}$ suggests an overall airways obstruction, although neither bronchoconstriction, compliance nor elastic recoil was actually measured. Since FEV_1 is the volume of air recorded in first second of FVC and constitutes 80-90% of FVC, a highly significant reduction in FEV_1 reflects small airway change in addition to large airway changes. Schatz (1990) have noted significantly reduced FEV_1 in asthmatic pregnant women. Das et al. (1991) have reported mean FEV_1 and FVC in normal non smoking and light smoking pregnant women as 3.36 ± 0.39 and 3.09 ± 0.45 and 85.65 ± 4.13 and 82.10 ± 6.23 respectively. Parre et al (1995) have reported progressive loss of airway distension due to loss of lung elastic recoil in asthmatics.

The mean values of expiratory flow rates and instantaneous flow rates are presented in table 4 and table 5 respectively. The mean $FEF_{0\,2\text{-}1\,2}$ were 2.59 ± 0.87 and 1.53 ± 0.59 and $V_{max25\%}$ were 1.40 ± 0.49 and 0.89 ± 0.36 in group I-P and group II-P respectively. The mean difference calculated statistically was seen to be significantly reduced ($P<0.01$) in group II-P as compared to group I-P. The decreased flow observed in $FEF_{0\,2\text{-}1\,2}$ and $V_{max25\%}$ reflects large airways

resistance or a decrease in effort as, much of these flows are effort dependent. Although subjects performed tests using maximum effort, a decrease in the expiratory muscle power among smokers cannot be entirely ruled out.

In contrast $FEF_{25-75\%}$, $FEF_{75-85\%}$, $V_{max50\%}$ and $V_{max75\%}$ are independent of effort and are important to detect small airway patency, particularly in airways with a diameter ≤ 2 mm. The mean $FEF_{25-75\%}$ and $V_{max50\%}$ observed were 2.24 ± 0.61 and 1.29 ± 0.43 (lit/sec) and 2.42 ± 0.74 and 1.43 ± 0.54 (lit/sec), while $FEF_{75-85\%}$ and $V_{max75\%}$ values were 1.10 ± 0.36 and 0.72 ± 0.27 and 2.96 ± 0.92 and 1.79 ± 0.68 in group I-P and group II-P respectively. The mean difference calculated statistically between groups in each parameter was found to be significantly ($P<0.001$) lower in group II-P as compared to group I-P. $FEF_{25-75\%}$ is said to be a very sensitive parameter to detect airflow in peripheral airways where the diseases of chronic airflow obstruction are thought to begin (McFadden and Linden, 1972; Coiso et al, 1978; Buist, 1989 and Das et al, 1991). A 25% reduction in $FEF_{75-85\%}$ is considered to be clinically abnormal (Morris et al, 1975), however the sensitivity of $FEF_{25-75\%}$ to dysfunction is not necessarily greater within and between individual variability (Buist, 1989). A reduced $V_{max75\%}$ is strongly suggestive of early small airway disease even though the subjects were asymptomatic at the time of recruitment. In group II-P the contribution of small airway resistance to total airway resistance is normally expected to increase at lower lung volumes, even if FEV_1 would not have been clinically abnormal. As FEV_1 is abnormally low, the small airway contribution is attributable for the same. Their findings are comparable to those obtained by Das et al. (1991) who found a significant reduction in both $FEF_{25-75\%}$ and $FEF_{75-85\%}$ ($P<0.001$)in smoker group to that of nonsmokers.

Although a direct airway resistance and elastic recoil pressure or lung compliance were not measured, it is assumed that the observed decrease in flow rates at low lung volume is caused by increased resistance to flow rather than a decrease in driving force. Alteration in lung recoil in group II-P can not be entirely ruled out; however in normal pregnant women lung elastic recoil should not be altered because lung compliance has been shown to remain unchanged during pregnancy compared with the postpartum period (Gee et al, 1967).

The lung functions conducted 6-8 weeks postpartum (equivalent to pre-pregnant state) in the same subjects served as the control data for the respective subjects. The physiological changes in lung function occurring during pregnancy in normal subjects and in subjects with respiratory problems can be compared. Group I-P and group II-P subjects in their postpartum state were designated as group I-PP and group II-PP, the mean values of each parameter are shown in table 6, 7 and 8).

The mean FVC recorded within 6-8 wks postpartum were 2.11 ± 0.41 and 2.00 ± 0.34 lit in group I-PP and group II-PP respectively. The difference in their mean was not found to be statistically significant ($P<0.1$), though a little lower mean value was seen in group II-PP as compared to group I-PP. The narrowing or closure of small airway and loss of elastic recoil pressure of the lung, related to the varied etiological factors considered in selection or inclusion criterion of group II-PP subjects might explain the lower FVC in this group.

The mean FEV_1 noted were 1.83 ± 0.33 and 1.42 ± 0.33 lit in group I-PP and group II-PP respectively and the mean difference derived statistically between the two groups has shown a significantly reduced FEV_1 ($P<0.001$) in group II-PP, as compared to group I-PP ($P<0.001$). Thus the statistical mean difference of $FEV_{1\%}$ (X = 86.7 ±

8.36 and X = 70.6 ± 9.11 in group I-PP and group II-PP respectively) was found to be significantly lower in group II-PP (P<0.001). This finding suggests an overall airways obstruction and probably confirms its existence preconceptionally and independent of pregnancy.

The mean $FEF_{0\,2\text{-}1\,2}$ and $V_{max25\%}$ noted in group I-PP and group II-PP were 2.49 ± 0.84 and 1.62 ± 0.80 and 2.73 ± 0.96 and 1.85 ± 0.89 respectively and the statistically difference of their means showed a significant decrease in both parameter of group II-PP (P<0.001) showing large airway function reduction.

The $FEF_{25\text{-}75\%}$ and $V_{max50\%}$ obtained in group I-PP and group II-PP were 1.97 ± 0.62 and 1.35 ± 0.56 and 2.14 ± 0.72 and 1.44 ± 0.56 respectively. The difference of mean determined statistically showed a significantly reduction (P<0.001) in group II-PP. Since this effort independent portion of FVC curve reflects small airway function, hence significantly reduced small airway function is indicated.

The mean $FEF_{75\text{-}85\%}$ and $V_{max75\%}$ recorded in group I-PP and group II-PP were 0.93 ± 0.32 and 0.75 ± 0.29 and 1.15 ± 0.40 and 0.89 ± 0.39 respectively. The difference of means in each parameter was found to be significantly decreased (P<0.001) in group II-PP, suggesting a significant reduction of peripheral airways flow rate.

The observed decrease in expiratory flow rates and instantaneous flow rates at lower lung volumes suggests an increase resistance in flow rather than a decrease in driving force. The physiological changes associated with pregnancy tend to preserve the lung compliance against gross mechanical opposing forces, but as the postpartum period obviates this protective change and the preexisting elastic recoil pressure may dominate which might have been reflected as a decrease in flow rates observed in group II-PP. Thus these postpartum findings of lung function confirm the existence of airways

obstruction prior to pregnancy in group II-PP. And the within variability of parameters in group II (group II-P and group II-PP) indicates that the bronchodilatation brought about by increased level of progesterone in pregnancy, specially near term may not be sufficient to overcome the deleterious effect in airways function.

Maintenance of normal lung function in pregnancy is important for maternal gas exchange across the alveolar-capillary membrane and thus also on the maternal oxygen saturation. Despite of remarkable reserve of pulmonary function in normal pregnant women, pregnancy can tip the balance towards deterioration when underlying lung disease is present (Greenberger, 1992). Thus pregnant women with respiratory problem pose a special challenge.

During normal pregnancy the biochemical and mechanical changes occur which alter gas exchange and pulmonary functions. Progesterone stimulates respiration and increases minute ventilation upto 40% by increasing TV without increasing the respiratory rate. Because of the increased body surface area of the mother and the energy demands of the fetus, oxygen consumption is increased by approximately 20% (Cugell et al, 1953). This disproportionate increase in ventilation compared with oxygen consumption results in physiological hyperventilation and a mild respiratory alkalosis. The other notable alteration in pulmonary mechanics is a decrease in airway resistance, which is likely due to progesterone, induced bronchial smooth muscle relaxation (Gee et al,, 1967).

The mechanical effect of the gravid uterus results in small changes in lung volumes, which are clinically insignificant except for the 20% decrease in FRC at term (Gee et al, 1967). Normally, during tidal breathing, the end tidal volume (or FRC) will exceed the CV when the small airway (≤2mm diameter) in the dependent portion of the lung begins to collapse. During the third trimester, particularly in the

supine position, CV can exceed FRC indicating premature closing of the airways, creating a mismatch between ventilation and perfusion, and causing arterial hypoxemia (Muller et al, 1980).

Pregnancy induced hyperventilation results in a decreased P_{aCO2}, but the blood pH is maintained in a slightly alkalotic range by renal compensation. As a result of the decrease in P_{aCO2} and hence, P_{ACO2} alveolar P_{O2} increases and P_{aO2} therefore also increases. However, an abnormally high (A-a) P_{O2} near term, presumably related to airway closure, partially offsets the increase in P_{Q2} expected from hyperventilation. In subjects without underlying pulmonary disease, these changes in P_{aO2} appear to have little clinical significance.

Pulse oximeter is safe, noninvasive monitoring technique that measures changes in transmission of red light that passed through a vascular bed to determine the arterial hemoglobin saturation (S_aO_2). It is useful in patients with altered lung function at risk for hypoxemia and may obviate frequent arterial blood gas sampling.

S_aO_2 has been defined as the ratio of oxygenated hemoglobin to total hemoglobin expressed as percentage and falls within ± 2% of that measured with arterial blood gas analysis (Cohen et al, 1988). It does not take into account hemoglobin concentration and therefore is not equivalent to oxygen content. A normal saturation of hemoglobin again does not guarantee that oxygen exchange will occur but in most circumstances it correlates well with tissue oxygenation and thus serves as an indirect indicator of maternal respiratory function more so, in patients with normal hemoglobin concentration. The oxygen hemoglobin dissociation curve is to be referred to relate S_aO_2 to oxygen tension. The normal oxygen saturation determined by pulse oximetry in non-pregnant and pregnant women breathing room air is approximately 96-100%, no significant change is seen in pregnancy (Porter et al, 1988 and VanHooke et al, 1996). Because of the shape of

oxyhemoglobin dissociation curve the S_aO_2 will not fall significantly P_{aO2} drops to 60-70 mm Hg (Nobel et al, 1988).

The preservation of the maternal oxygen in face of a normal hemoglobin concentration is postulated to be due to the increased concentration of progesterone (Brownell et al, 1986). But even in a normal pregnancy hypoxemia was noted at term related to postural change i.e. from sitting to supine posture (Awe et al, 1979 and Norregaard, 1989). Norregaard (1989) related the increased risk of hypoxemia to the decrease in FRC and also that the closing capacity approaches the FRC, as supine posture is associated with airways closure above FRC mainly due to increased diaphragmatic pressure, caval compression and hemodynamic changes. Ang et al (1969) found a 13 mm Hg decrease in capillary P_{O2} in women near term on a result of a change from sitting to supine position and Awe et al, (1979) discovered an increase in (A-a) P_{O2} from 14.3 to 20 mm Hg due to postural change (sitting to supine). There is a definite relationship between oxygenation during wakefulness and that during sleep (Douglas, 1992). There appears to be the reason for concern about potential nocturnal hypoxemia during pregnancy, both as a primary event and as consequence of sleep apnea (Feinsilver and Hertz, 1992). Thus any condition that causes maternal hypoxemia while awake will worsen it during sleep, particularly in supine posture. The respiratory obstruction related to the decreased airways function will cause ventilation-perfusion mismatch and would subsequently cause maternal hypoxemia. Several studies have been conducted to seek the association between maternal respiratory dysfunction and hypoxemia and have put forward relevant explanations.

Hypoxemia was noted in patients with COPD being severely affecting during sleep (Wyne et al, 1979; Douglas et al, 1979 and Douglas, 1992). The major cause of hypoxemia during REM sleep in

patients with COPD is hypoventilation adding to the prevailing decreased FRC and ventilation perfusion mismatch. In cigarette smokers CO causes a left shift in the oxygen hemoglobin dissociation curve, resulting in an increased affinity of the maternal hemoglobin and a decreased release to fetal hemoglobin. CO dissociates slowly from hemoglobin, with a half life of 4 hours in an awake, sedentary adult breathing room temperature. Sleeping is found to increase this period to 10 hours (Pearce and Jones, 1984). A typical puff of cigarette smoke contains up to 5% CO, 10% O_2 and 9.5% CO_2 (Conroy, 1969). The affinity constant of hemoglobin for CO is more than 200 times that of oxygen; even small amounts of CO are profoundly bound and tightly held. Typical hemoglobin CO concentration in nonsmokers range up to 2.5%; in smokers they may reach 15% (Pearce and Jones, 1984). Again nicotine is a potent adrenergic agonist, producing significant elevations in arterial blood pressure, heart rate and peripheral vascular resistance for 20-30 minutes after exposure. The net effect of smoking is nicotine induced increase in O_2 consumption with a reduced O_2 supply secondary to CO (Jones et al, 1987). VanHooke (1996) recorded significant lower S_aO_2 (<96%) in smokers while S_aO_2 in chronic smokers was found to artificially increased by an increase of HbCO levels by 1%-2% (Kendrick, 1996).

Arterial oxygen saturation (table 9) was carried out to learn about the differences in oxygenation status in the normal group and in subjects with airways obstruction, both during pregnancy and postpartum.

The mean SpO_2 recorded were 98.99 ± 1.08 and 97.92 ± 0.82 percent in group I-P and group II-P respectively. The mean difference evaluated statistically was found to be significantly lower in group II-P compared to group I-P ($P<0.001$), though the mean values noted in both the cases fall within the normal range. But mean value in group

II is seen to fall in the lower limit of normal range (97 – 100 %). Since decreased airway functions were noted in this group the ventilation-perfusion mismatch seems to be the major factor responsible. The cause of the same is likely to be related to hypoventilated alveoli being continuously perfused.

The mean SpO_2 recorded at postpartum were 97.08 ± 0.74 and 95.98 ± 0.85 in group I-PP and group II-PP respectively and their difference calculated statistically was found to be abnormally lower in group II ($P<0.001$), same ventilation-perfusion mismatch may be the factor responsible for this change. Considering the inclusion/exclusion criterion that is normal hemoglobin content, no cardiovascular, renal or musculoskeletal diseases, as well the airway functions were found to be reduced in this group, the lower S_aO_2 indicates respiratory obstruction as the only possible cause of these findings.

While comparing the pregnant SpO_2 means in both groups to their respective means at postpartum shows lower values in postpartum that instigates the bronchodilatory effect of progesterone in improving lung function and thus the maternal oxygenation. Moreover, all the subjects were breastfeeding at the time of postpartum study whereby the progesterone concentration would be still lower. Though the levels were not measured the protective role of progesterone cannot be ruled out.

Maternal oxygenation is the prime determinant of fetal oxygenation which in turn affects the fetuses' development and growth. Maternal P_{aO2} and thus S_aO_2 affect the oxygen content of the uterine artery blood flow (Parer, 1987).

Variation in maternal pH also influences oxygen delivery (Novy et al, 1967 and Parer, 1987). Alkalosis causes vasoconstriction of the

uterine artery resulting in decreased fetal oxygen delivery. This effect is magnified by leftward shift in the maternal oxyhemoglobin saturation curve which increases oxygen affinity and consequently decreases oxygen transfer to the umbilical vein. Although mild maternal acidosis does not enhance uterine blood flow because the uterine vasculature is maximally dilated already, it shifts the maternal oxyhemoglobin saturation curve to the right, which leads to decreased oxygen affinity and increased fetal oxygen delivery (Novy et al, 1967). Maternal hypotension and increased sympathetic stimulation (exogenous or endogenous) both cause uterine arterial vasoconstriction.

The interaction of the maternal and fetal circulations in the placenta most likely follows a concurrent exchange mechanism (Meschia,1979 and Parer, 1987), which partly explains presence of lower fetal umbilical vein P_{O2} (32 mm Hg) than the uterine vein P_{O2} and why increased inspired oxygen increases oxygen tension but does not cause major increase in umbilical vein P_{O2}.

Despite of low umbilical vein P_{O2}, fetal oxygen content is actually quite close to maternal oxygen content because of the shape of the oxyhemoglobin saturation curve for fetal hemoglobin (Metcalfe, 1972 and Sullivan and Ramnathan, 1985). This is one of the major protective mechanism for fetal oxygenation. In addition, the fetal oxyhemoglobin saturation curve is relatively unaffected by changes in pH; although acidosis may decrease maternal hemoglobin oxygen affinity, fetal oxygen affinity is unchanged.

The fetal arterial blood has even lower P_{O2} than umbilical vein blood, because oxygenated umbilical vein blood is mixed in the fetal inferior venacava with deoxygenated systematic venous blood before delivery to the systematic circulation. This is compensated in part by a high fetal cardiac output relative to oxygen consumption thus

enhancing oxygen delivery to tissues (Longo et al, 1977). In addition, the fetal circulation appears to have the ability to autoregulate in the face of hypoxemia, thus protecting brain, adrenal glands and heart (Peeters et al, 1979). But the most rising question is – how long this adaptation can be dependent on before organ damage occurs in the underperfused areas? And how well do the compensatory mechanisms that provide adequate oxygen supply to the fetus under normal conditions manage during maternal hypoxia?

The fetus like the newborn, responds in various stereotypic manners to stress particularly by a decrease in growth rate. This response may be seen in its reaction to many pathophysiologic insults. When faced with growth retarded fetus, an attempt is made to ascertain the nature of insult responsible for the observed abnormality. The underlying etiology may have important ramification regarding fetal prognosis and pregnancy management. IUGR represents a continuum of condition that ultimately results in the failure of the fetus to attain its inherent growth potential putting it at risk for increased perinatal morbidity and mortality.

The growth process is complex and delicate and may be viewed as having three consecutive phases (Winick, 1971). The first phase is referred to as the phase of cellular hyperplasia and encompasses the first 16 weeks of gestation. During this phase, a rapid increase in cell number occurs. The second phase known as the phase concomitant hyperplasia and hypertrophy occurs between the 16th and 32nd weeks and involves increase in cell size and number. The third and final phase referred to as the phase of cellular hypertrophy occur between 32nd week gestation and term and is characterized by a rapid increase in cell size. It is in this phase that most fetal fat deposition is thought to occur the baby may gain as much as seven ounces / week. The fetus can suffer from the restricted

growth if the delicate process of development and weight gain is disturbed or interrupted

IUGR is defined as the baby weight at or below the 10th percentile for his or her gestational age. It is clinically clarified on the basis of the morphometric pattern of fetal growth, as symmetrical (type I) and asymmetrical (type II). The symmetrical IUGR is associated with reduction of all the external measurement (weight, length and head measurement), thus producing a normal fetal ponderal index (PI). Whereas asymmetrical (type II) IUGR is associated with the head circumference and length being relatively normal for the gestational age, but with a reduced weight/length ratio and thus the PI.

Birth weight is known to be the prime determinant of fetal growth and the strongest known indicator of perinatal mortality. Obviously birth weight depends both on gestational age and fetal growth. Infants of low birth weight thus may be classified as belonging to one of the following three groups (1) preterm or born before 37 weeks' of gestation and appropriate weight for gestational age, (2) preterm and growth retarded or born before 37 weeks' of gestation and weighing less than appropriate for gestational age. (3) term and small-for-gestational-age. The importance of analyzing birth weight as a function of gestational age is confirmed by the fact that this relationship has a direct effect on perinatal morbidity and mortality. It was shown that, within fixed gestational age strata perinatal mortality is related inversely to birth weight (Williams et al, 1982). The increase in perinatal mortality associated with growth retardation is correlated with increases in fetal and neonatal mortality rates. Although perinatal mortality is an outcome variable that is both clinically relevant and readily ascertainable, the morbidity associated with IUGR is also significant.

The growth and development of fetus in utero reflects a balance between the fetus, the placenta and the mothers. The fetus depends on both on adequate maternal fuel supply and the ability of the maternal vascular tree to deliver an adequate fuel supply to fetoplacental unit. Any impairment in maternal homeostasis may have an adverse effect on fetal growth and development.

The present study involves subjects with lung disorder associated with significant airway obstruction which leads to maternal hypoxemia. So the attention is being currently focused on the underlying maternal conditions/factors that may reduce the fetal birth weight. Low birth weight is resultant of either due to preterm birth or IUGR.

Obstructive pulmonary disease like asthma has been studied without clear conclusions. Some studies have noted low birth weight may be due to preterm delivery with no difference in perinatal mortality to that of known asthmatics (Fittzsimmons et al, 1986; Lao and Huengsburg, 1990; Perlow et al, 1992 and Kelly et al, 1995). Increase incidence of prematurity low birth weight and neonatal mortality in asthmatics was reported by Bahana and Bjerkedal (1972). While no difference in gestational age, birth weight or perinatal mortality in asthmatics compared to non-asthmatics was reported by few (Dombrowski et al, 1986; Stennius-Aariala et al, 1988; Mabie et al, 1992 and Park and Chazotte, 1994). The etiology of birth weight was mostly explained on the basis of maternal hypoxemia caused by asthma, Schatz (1990) specifically related it to lower maternal gestational FEV_1.

Smoking is associated with low birth weight due to IUGR (Gluckman and Liggins, 1954; Davis and Gray, 1976; Miller and Hassanein, 1964; Pearson et al, 1978; Pirani, 1978; Naeye, 1978; Christianson, 1979; Wouters et al, 1987; Wertelecki et al, 1987;

Usandizaga et al, 1987; Aronson et al,, 1993; Cnattingius et al, 1997; Horta et al, 1997; Seeker-Walker et al, 1997 and Das et al, 1998), preterm delivery (Kirstein et al, 1996) and both (Shi Wu Wen et al, 1990 and Kalinka and Hanke, 1996).

The effect of smoking on birth weight appears in particularly to be mediated through vascular constriction and decreased utero-placental blood flow. The smoke contaminants include CO, nicotine and cyanide, the action of which is explained as follows.

CO from cigarette smoke will not only cause fetal hypoxia but also blunt the adaptive response that would normally occur due to the insult. That fetus has limited ability to respond to hypoxia because it already has a high hematocrit and its cardiac output is almost at the maximum. HbF has even greater affinity for CO than HbA. Thus, though combination of CO and HbF results in increased hemoglobin oxygen binding the oxygen availability at the tissue level is decreased (left shift of O_2 dissociation curve). Moreover, CO stays elevated longer in the fetus as it is less able to metabolize it (Longo, 1977). A woman who smokes two pack of cigarettes/day during pregnancy will have 10% concentration of carboxyhemoglobin, which is equivalent to a 60% reduction in blood flow (Longo, 1977 and Longo and Hill, 1977).

The physiologic effect of nicotine is an increase in the circulating levels of norepinephrine, epinephrine and acetyl choline through its action on the adrenal gland (Manning, 1978 and Monheit, 1983). This sympathetic response leads to a decrease in utero placental perfusion. Nicotine crosses the placenta and either directly or through that adrenal gland causes a rise in blood pressure and respiratory rate of the fetus (Sachs et al, 1989).

The placental changes noted in smokers were the same as seen in cases of chronic hypoxia or ischemia. These were relative

hypertrophy of the placenta, a decrease in vasculosyncytial membrane, a decrease in cytotropholastic cell proliferation, decreased pinocytosis and trophoblastic secretor activity and focal syncytial necrosis (Sachs, 1989). These microscopic changes are consistent with the pathophysiological effects of CO and nicotine on pregnancy.

Increased levels of smoke contaminants metabolites after maternal exposure were shown in both the mothers and the fetus (Astrup et al, 1972; Jarvis and Russell, 1984 and Lynch, 1984). Passive smoking during pregnancy was found to double the non smokers risk of delivering IUGR infant (Martin and Bracken, 1986), and is approximated to be similar to those found for cigarette smoking mothers. Effect of paternal smoking on fetal weight gain have been studied widely and were shown to reduce birth weight (Yerushalamy, 1962; McMohan et al, 1966 and Rubin et al, 1986) while no significant effect of paternal smoking on having low birth weight infant was found (Terri and Gold, 1969). Pedrira et al (1985) studied the association of passive smoking and childhood respiratory illness where they found a direct relation. Passive smoke exposure was significantly related to delivering a low birth weight new born at term (Martin and Bracken, 1986; Jedrochowski and Flak, 1996; Ahluwalia et al, 1997 and Dejin-Karlson et al, 1998). The basic mechanism of the fetomaternal interaction due to passive smoke exposure in pregnancy is believed to be similar to that studied in active smokers and is mediated through vascular constriction and decreased utero-placental blood flow.

Though the growth of the human fetus was a subject of extensive study, prior to advent of ultrasonography, physicians interested in the growth process of the fetus could only assess by historical dating (last menstrual period), serial fundal height measure and comparison with the actual size of the neonate at birth. Based on this observation, clinicians were able to categorize fetuses in very

general terms taking into account their age and size. For e.g., fetuses were classified as preterm if they were born before 37 menstrual weeks, term if they were born from 38 and 42 menstrual weeks and post term if they were born beyond 42 menstrual weeks. Similarly fetuses were classified by birth weight as small-for-gestational-age, appropriate for gestational age and large for gestational age. These categories allowed clinicians to recognize preterm or post term fetuses and fetuses born too small or too large for menstrual age (Pollack and Divon, 1992 and Campbell, 1998).

Obstetric ultrasonography has enhanced the ability to detect growth abnormalities, thus directing more intensive antepartum care with an improvement in perinatal outcome. Routine ultrasound used for dating prior to 20 weeks' gestation provides an extremely accurate gestational age (± 7 days) and allows more confident diagnosis of growth abnormalities if at all they occur (Hadlock, 1987 and Campbell, 1998). In the present study the gestational age (fetal dating) was confirmed in all subjects by an ultrasound examination conducted at the mean age of 12 weeks gestation (11-14 weeks) with that noted through historical dating (LMP). The subjects in whom LMP and approximated ultrasound gestation dating did not relate were excluded from this study.

The common morphometric parameters applied in the assessment of fetal growth and also used in the present study are BPD, HC, AC, FL and their ratios like HC/AC, BPD/FL and FL/AC were noted (table 10 and 11). An estimated fetal weight using formula involving the above mentioned biometric parameter was determined to evaluate the fetal growth.

The mean BPD recorded in the present study was 8.06 ± 0.68 and 7.91 ± 0.64 cm in group I-P and group II-P respectively. The difference of mean was found to be statistically insignificant (P>0.1),

but a little lower mean is observed in group II-P. Queenan et al (1976) determined BPD serially in 468 normal obstetric patients and noted mean BPD at 32 weeks approximately 7.9 cm (7.2–8.6 cm), which is in agreement to that seen in the present study. They reported that though the accuracy and reproducibility of ultrasound fetal BPD is established due to the variations in the normal growth of fetus, it poses difficulty in detecting IUGR. The range of normal was found to be relatively large for each week of gestation therefore, a single value is seldom useful.

The mean HC recorded was 29.59 ± 2.26 and 29.33 ± 2.65 cm in group I-P and group II-P respectively. The mean difference calculated was found to be statistically insignificant. The mean HC noted in group II-P was found to be slightly lower as compared to group I-P. Zimmer and Divon (1992) have suggested that the measurement of HC is more representative of the brain size for diagnosing IUGR. Hadlock (1982) also supports this view and has further reported that in symmetric IUGR, fetal head size will frequently be compromised early in pregnancy, while in asymmetric IUGR the growth of the fetal head is typically normal till late pregnancy because of preferential blood flow to the brain at the expense of other organ systems. Since HC values in both groups are within normal range the fetal head growth seems to be normal in the present study.

The mean AC recorded was 26.76 ± 2.78 and 25.97 ± 2.90 cm in group I-P and group II-P respectively. The mean difference noted statistically was found to be insignificant but a little lower value is noted in group II-P. This measurement was first proposed by Campbell and Thoms as a means of estimating fetal weight and to identify the growth retarded fetuses. Actually this measurement is the best analyzer of fetal trunk size and soft tissues. The AC is decreased

in both symmetric and asymmetric growth retardation. One point to remember, however, is that the AC has the greatest interobserver and intraobserver variability of all other fetal measurement (Campbell, 1998). Considering the above fact in the present study a single observer and determination using same instrument was stressed upon. The values of AC in both groups were found to be within normal range thus reflecting normal abdominal girth growth.

The mean FL noted in the present study was 6.09 ± 0.78 and 6.02 ± 0.57 cm in group I-P and group II-P respectively. A slightly lower but insignificant difference is reported on statistically evaluation in group II-P. FL is reproducible from 15 weeks gestation to term to represent linear growth (Campbell, 1998). The degree of compromised FL in IUGR is inconsistent. The femur growth lag is common early with symmetric IUGR but is less predictable with asymmetric IUGR. Early asymmetric IUGR may be associated with a normal FL, but, as the pregnancy continues, a lag may become apparent (Hadlock, 1984). Perhaps the major role of FL in the growth profile is in the evaluation of the fetal weight and body proportionality. The value of FL in both groups falls in a normal range indicating a linear growth.

The earliest fetal morphometric ratio used in detection of IUGR was the HC/AC ratio described by Campbell and Thoms. With an accurate knowledge of gestational age the values are found to be >1 until 34 to 36 weeks gestation, after which they decrease ≤1 until delivery (Campbell, 1998). An abnormal HC/AC ratio may predict some IUGR fetuses especially in situations of placental insufficiency resulting in asymmetric fetal growth related to third trimester. The BPD/FL in itself may report fallacy resulting from a large or small fetal head with a normal AC, but when used in study along with other measurement might aid in identification of IUGR.

Another morphometric ratio applied for the identification of fetal growth retardation is FL/AC ratio. It is the gestational age independent and remains constant after 20 weeks. Most practitioners have used the 90th percentile (23.5) as the upper limits to detect IUGR. Limitations in the use of this ratio include its applicability only to asymmetric IUGR (Campbell, 1998).

The morphometric ratios calculated in this study are presented in table 11. The mean BPD/FL recorded was 1.31 ± 0.08 and 1.30 ± 0.10 and mean HC/AC was 1.11 ± 0.08 and 1.14 ± 0.09 and mean FL/AC was 23.11 ± 1.62 and 23.42 ± 1.33 in group I-P and group II-P respectively. The difference of mean of each ratio was found to be statistically insignificant (p> 0.1).

Currently, the most common method of screening for and identification of the small-for-gestational-age and suspected IUGR infant is estimation of fetal weight. An accurate knowledge of gestational age is essential for proper interpretation of estimated fetal weight (Ott, 1997). If the estimated fetal weight decreases below the 10th percentile the fetus is designated as small-for-gestational-age and is at risk for IUGR.

The mean estimated fetal weight derived in this study was 1808.97 ± 498.79 and 1683.33 ± 511.3 gm in group I-P and group II-P respectively. The mean difference between the two groups was found to be statistically insignificant (P>0.1). The mean values are found to meet the normal standards for EFW put forward by Hadlock et al (1991) fall between the 10th and 50th percentile of their mean gestational age of 32 weeks. But insignificant lag of weight is noted in group II-P.

The morphometric measurement reported in the present study are found to relate to the normal values derived and standardized by

several workers and so does the biometric ratios and EFW. But the lower values noted in group II-P as compared to group I-P needs to be considered though this difference was statistically found to be insignificant, it needs to be related to the neonatal outcome parameter for further evaluation. It is well documented that the current ultrasound methods are more useful for including the possibility of abnormal fetal growth rather than for confirming it Zimmer and Divon, 1992). The parameter like AC and FC are associated with an apparent lag only with advanced pregnancy, probably the measurement by 34 weeks' gestation may be more applicable for detection of IUGR (Warsof, 1986 and Campbell, 1998). Screening at an approximately 34 weeks is related to sensitivity of approximately 70% and a predictive value of 50% (Campbell, 1998). Thus probably lower values found in the morphometric parameters and EFW in group II-P may be just the indication of the onset of fetal growth lag.

The neonatal outcome data was collected from the hospital records. The noted mean gestational age at delivery were 38.41 ± 1.21 and 38.42 ± 0.77 weeks in a group I and group II respectively, which reflects deliveries of both groups. The physical characteristics of neonate are presented in table 12. The commonly studied variables/parameters are included in the study viz CC, HC, height/length, birth weight and neonatal PI.

The mean neonatal CC recorded were 32.0 ± 1.52 and 31.75 ± 1.45 cm, the mean neonatal HC were 33.93 ± 1.66 and 33.73 ± 1.39 cm and the mean neonatal height was 47.90 ± 1.81 and 47.70 ± 1.53 cm in group I-P and group II-P respectively. The mean difference was found to be statistically non significant (P>0.1) though a little lower mean values of each parameter was found ing2p as compared to group I-P. all these mean average relate to the mean values documented in standard Indian books of pediatrics (Ghai).

But the mean PI of these neonatal was found to be significantly lower in group II-P as compared to group I-P. The mean PI recorded was 2.40 ± 0.29 in group I-P and 2.13 ± 0.15 in group II-P. PI is birth weight/length^3 x100. The lower birth weight in face of the normal height is found to be responsible for this discrepancy. The mean birth weight noted was 2643.97 ± 368.81 in group I-P and 2319.29 ± 270.20 gm in group II-P. The mean difference calculate statistically between them was found to be significantly lower in group II-P as compared to group I-P ($P<0.001$). it is established and documented that the asymmetrical (type 1) IUGR is associated with the HC and length being relatively normal for the gestational age but with a reduced weight/length ratio and PI (<2.2) as given by Van Geijn et al (1980) and Schatz et al (1990). Thus the decreased mean birth weight and PI found in group II-P of our study indicates asymmetrical IUGR. The growth retardation seen at birth could not be detected by the ultrasound conducted in the present study probably because the growth lag in asymmetrical IUGR is believed to occur in late gestation (>32 weeks). This fetal growth phase is commonly referred to as phase of cellular hypertrophy and is characterized by a rapid increase in cell size and it is in this phase in which most fetal fat deposition is thought to occur (Pollack and Divon, 1992). Thus the lower birth weight noted in the group II-P compared to group I-P, though not significant, was probably an indicator of the onset of growth lag. The neonatal data of significantly reduced birth weight or low birth weight (<2500 gm) and PI (<2.2), indicates an established asymmetrical IUGR. Progressive uteroplacental insufficiency is believed to cause asymmetrical IUGR (Wheeler et al, 1996 and Cnattingius et al, 1997).

Thus from the above discussion, it is apparent that the prevalence of the obstructive disorders affecting small airway function in pregnant women leads to maternal hypoxemia. Maternal hypoxemia affects the fetal growth causing growth retardation. Although, infants

with asymmetric IUGR may be at a greater risk for perinatal hypoxia and neonatal hypoglycemia but have good long term prognosis with appropriate management (Pollack and Divon, 1992). The early detection of growth lag by serial ultrasound determination might prove beneficial.