

## A LONGITUDINAL STUDY OF PULMONARY FUNCTION TESTS DURING PREGNANCY

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**Abstract :** The study deals with evaluation of pulmonary function status in fifty normal pregnant women tested monthly. The parameters studied were Vital Capacity (VC) Forced Vital Capacity (FVC) and Forced Expiratory Volume in 1st second ( $FEV_1$ ) using Vitalograph Spirometer; tidal volume ( $V_T$ ), inspiratory capacity (IC) and expiratory reserve volume (ERV) using Expirograph and resting minute ventilation ( $V_E$ ) using Tissot's spirometer. Control values were obtained in the same subject 8-10 weeks after delivery. The increase seen in  $V_T$ ,  $V_E$  and IC was very highly significant. The small increment in frequency of respiration is significant and the declining trend observed in ERV is very highly significant. VC and FVC were maintained by the rise in IC and a concomittant fall in ERV. Rise in VC is attributed mainly to rise in  $V_T$  than rise in frequency. The results suggest that though pulmonary function is altered during pregnancy, it is not compromised and hence does not induce any mechanical stress on the respiratory efficiency of the pregnant woman.

**Key words :** pregnancy lung function

### INTRODUCTION

A number of anatomical, biochemical and hormonal changes occur in a normal woman during the course of pregnancy; including changes in both pulmonary function and ventilation. The anatomic changes consist of increased transverse diameter of the chest due to widened subcostal angle (1,2). This compensates for the level of diaphragm which is raised by the enlarging uterus.

The hormonal changes mainly comprise of increased levels of progesterone (2). Progesterone exerts an influence on total minute ventilation and its subcomponents; tidal volume and frequency. It has been advocated unanimously that the most important feature is a rising minute ventilation and tidal volume during pregnancy.

The biochemical changes are of compensated respiratory alkalosis i.e. the respiratory alkalosis

produced by hyperventilation is compensated by metabolic acidosis to maintain pH at 7.44 (2).

Besides understanding the physiology of lung function during pregnancy the study also provides a control, on the background of which any respiratory problem which may appear during pregnancy can be evaluated with greater precision. Only few studies have reported average pulmonary functions during pregnancy in Central India (6,11). Of which one is a cross sectional study on different women (11) and other though longitudinal LFTs were recorded in each trimester of pregnancy (6). Hence we endeavoured to do a more extensive study by observing the changes in LFT in each month of pregnancy in the same woman. In the present study serial monthly estimations of LFT of pregnant woman in the same subjects have been made.

### METHODS

Fifty pregnant women were selected in their 3rd

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month of gestation from out patient Department of Gynaecology and Obstetrics, Government Medical College, Nagpur. Through history taking and a thorough clinical examination cardiopulmonary diseases were excluded. All women were in the age group 20-28 yrs (average age 24 yrs) and height between 130-160 cms (average height 154.5 cms). The haemoglobin level of all women was above 10 gms/dL (Sahli Hellige's method). An informed consent was given by every woman. Each subject was examined at monthly interval and again 8-10 wks postpartum which was taken as control. Thus each subject formed her own control. The complexity of the study precluded serial evaluation of the subjects.

Pulmonary function tests were carried out in a postabsorptive state at least 12 hours after last meal. A rest period of 30 min preceded the test with 5 min between every reading. Discrete explanation along with demonstration of each test was given to the subjects. Maximum effort and full co-operation was urged each time. All tests were carried out with the subjects in standing position except VE (L/M) which was done in sitting position. Three readings were taken for every parameter and maximum of the three was recorded (Vitalograph S model spirometer manual of instructions). The same conditions were followed strictly during each serial testing in order to avoid technical variations. All observations were corrected to B.T.P.S. The observations during pregnancy were compared with the control readings which were taken as defined above and significance of changes was tested statistically using Student's t test.

*Instruments used were* : Vitalograph S model spirometer with function analyser (Vitalograph Ltd., Buckingham) for recording VC, FVC and FEV<sub>1</sub>.

Expirograph (Rajdhani Scientific Instruments Co., New Delhi) for recording V<sub>T</sub>, IC and ERV after volume calibration of 33 ml/div along vertical axis. Tissot's spirometer for recording V<sub>E</sub>.

## RESULTS

It was observed that VC, FVC and FEV<sub>1</sub> remained unchanged throughout pregnancy. Slight variations observed were statistically insignificant. The opposing changes in IC and ERV were statistically very highly

significant ( $P < 0.001$ ) and were responsible for maintaining the VC. The increment in V<sub>E</sub> as well as V<sub>T</sub> was statistically very highly significant and the small increase in frequency was statistically significant.

## DISCUSSION

An extensive serial estimation of the changes in lung function in 50 normal pregnant women was done using the methods accepted by American Thoracic Society.

*Vital Capacity* : VC remained unchanged throughout pregnancy. The low results of VC reported in our study was because of the low socio-economic status and poor nutrition of the subjects. Unchanged VC was reported by some (4,5,6) whereas decline in VC (7,8,9) and rise in VC (10,11) was also noted. Conflicting results could be due to observations on different subjects from different socio-economic status at different periods of gestation and postpartum period. The maintenance of VC was due to the increment in IC being compensated by the reduction in ERV.

*Forced Vital Capacity* : No change was observed in FVC (12,13,14,15). Significant reduction in FVC may be obtained due to restrictive effect of the enlarging uterus (15). Despite the progressively diminishing abdominal compliance maintenance of VC and FVC is attributed to augmentation of rib cage volume displacement, relative mobility of thoracic cage and unimpaired diaphragmatic movements.

*Forced Expiratory Volume in 1st second* : No significant change was observed in FEV<sub>1</sub>. No change was observed in  $\frac{FEV_1}{FVC}\%$  as shown in Table I (4,7,9, 10, 13, 14). The mechanical disadvantage to the respiratory apparatus induced by advancing pregnancy is well compensated by decrease in airway resistance and improved airway conductance due to smooth muscle relaxation produced by progesterone, corticosteroids and relaxin (2).

*Inspiratory Capacity* : IC increased during pregnancy (4,6,8,13). This is due to the altered thoracic configuration and also to heightened sensitivity to the nervous stimuli required to produce muscular contraction.

*Expiratory Reserve Volume* : A decline in ERV

was observed. (4,6,8,10,11,13). Reduction in power of the muscles of expiration due to the stretching of the abdominal wall with the progress of pregnancy is the main cause.

*Resting Minute Ventilation and its subcomponents :* Increased VT during pregnancy was observed (6,10,11,13). Together with the smooth muscle relaxation; a direct effect of progesterone increasing the sensitivity of respiratory centre to carbon dioxide is the probable cause of this rise (2). The altered thoracic configuration also contributes (1). Progesterone may exert its influence by modifying the permeability of the chemoreceptor cells or directly stimulating central

the placenta to unload its CO<sub>2</sub> (17). This manoeuvre satisfies the foetal needs of gas exchange and increases the margin of safety by protecting the foetus from high levels of CO<sub>2</sub> : but it causes considerable inconvenience to mother. To maintain the plasma pH increased excretion of sodium bicarbonate occurs. Thus the respiratory alkalosis is compensated. In this way hyperventilation in the mother is beneficial to the foetus as better oxygenation of maternal blood is possible and it also facilitates foeto-maternal gas exchange.

Thus though the pregnant woman has apparent handicaps causing restrictive changes in the respiratory apparatus; the anatomical, physiological and hormonal

TABLE I : Showing mean values of VC, FEV<sub>1</sub>, FEV<sub>1</sub>%, IC and ERV in litres during pregnancy ( $\bar{x} \pm S.D.$ ).

Month	VC	FVC	FEV <sub>1</sub>	FEV <sub>1</sub> /FVC%	IC	ERV
3	2.53 ± 0.12	2.19 ± 0.25	2.00 ± 0.09	92.60 ± 5.01	1.61 ± 0.22	0.64 ± 0.11
4	2.54 ± 0.35	2.19 ± 0.43	2.05 ± 0.37	96.36 ± 4.12	1.76 ± 0.33	0.52 ± 0.17
5	2.51 ± 0.14	2.14 ± 0.14	2.10 ± 0.20	98.06 ± 2.83	1.75 ± 0.33	0.53 ± 0.22
6	2.48 ± 0.34	2.10 ± 0.28	2.07 ± 0.25	98.63 ± 3.54	1.83 ± 0.42	0.48 ± 0.18
7	2.48 ± 0.21	2.10 ± 0.31	2.08 ± 0.33	99.32 ± 2.33	1.99 ± 0.39	0.41 ± 0.19
8	2.49 ± 0.31	2.21 ± 0.27	2.15 ± 0.21	97.51 ± 4.04	2.14 ± 0.40	0.34 ± 0.18
9	2.50 ± 0.15	2.17 ± 0.24	2.10 ± 0.18	97.37 ± 3.42	2.29 ± 0.29	0.27 ± 0.11
PP	2.53 ± 0.29	2.20 ± 0.26	2.20 ± 0.22	99.10 ± 2.37	1.61 ± 0.22	0.75 ± 0.13

respiratory or hypothalamic neurones in contact with blood (3). Progesterone may increase facilitating stimuli from higher centres eventhough at high concentrations it has anaesthetic effect (3). Frequency of respiration though increased with advancement of pregnancy; all the obtained values were within the normal range of frequency. Thus rise in V<sub>E</sub> is mainly due to rise in V<sub>T</sub>.

Persistent increment in V<sub>T</sub> has been observed in pregnancy (4,6,8,10,11,13). The pregnant woman overbreathes. The rise in VT is at the expense of ERV. So fresh air inspired with each breath is much less diluted by the gas in the lung. This reduces the alveolar and arterial pCO<sub>2</sub>. PaCO<sub>2</sub> reduces from 38 to 32 mmHg (16). The foetus using progesterone as manipulator, "resets" the respiratory centre to the new low level of CO<sub>2</sub> which is preserved so that it may itself enjoy a normal pCO<sub>2</sub> and still have a substantial gradient across

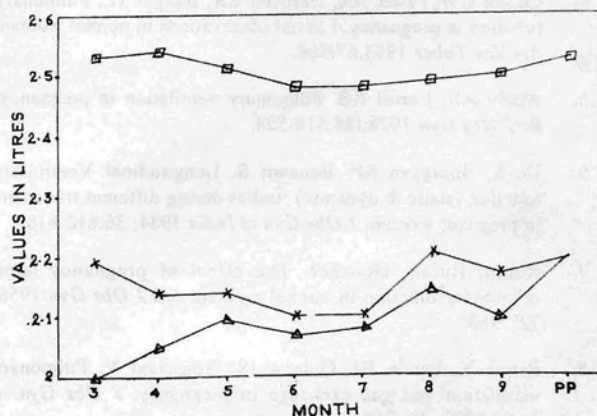


Fig. 1 : Showing mean values of VC (□), FVC (x), and FEV<sub>1</sub> (Δ) in litres in different months of pregnancy.

TABLE II : Showing mean values of  $V_E$ ,  $V_T$  in litres and per min during pregnancy ( $\bar{x} \pm S.D.$ ).

Month	$V_E$	$V_T$	$f$
3	7.91 $\pm$ 1.67	0.79 $\pm$ 0.16	11.00 $\pm$ 1.51
4	9.00 $\pm$ 2.00	0.78 $\pm$ 0.27	12.40 $\pm$ 2.98
5	9.33 $\pm$ 2.75	0.71 $\pm$ 0.30	14.50 $\pm$ 2.15
6	10.87 $\pm$ 4.81	0.83 $\pm$ 0.25	14.30 $\pm$ 1.72
7	13.02 $\pm$ 3.77	0.92 $\pm$ 0.15	14.83 $\pm$ 3.50
8	16.70 $\pm$ 4.85	1.03 $\pm$ 0.23	15.89 $\pm$ 3.71
9	18.49 $\pm$ 3.70	1.24 $\pm$ 0.45	16.50 $\pm$ 2.33
PP	7.30 $\pm$ 1.55	0.74 $\pm$ 0.13	12.00 $\pm$ 1.22

changes compensate for them causing no discomfort to the pregnant woman.

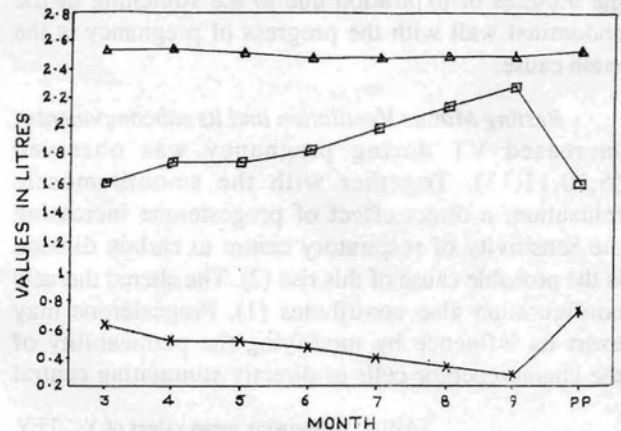


Fig. 2 : Showing mean values of IC (□), ERV (×) and VC (Δ) in litres in different months of pregnancy.

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