PEAK EXPIRATORY FLOW RATE AND SOME ASPECT OF ECG AND HAEMATOLOGICAL VARIABLES IN NON-PREGNANT AND PREGNANT WOMEN AT SECOND AND THIRD TRIMESTERS OF PREGNACY IN KURA LOCAL GOVERNMENT AREA, KANO STATE, NIGERIA.

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DECLARATION

I declare that the work in the project/ thesis entitled peak expiratory flow rate and some aspect of ECG and haematological variables in non-pregnant and pregnant women at second and third trimesters of pregnancy in Kura local government area of Kano state has been performed by me at Kura General hospital under the super vision of Dr. A. B. Adelaiye. The information derived from the literature has been duly acknowledged in the text and a list of references provided. No part of this project was previously presented for another degree or diploma at any university.

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OCTOBER, 2007

CERTIFICATION

This research project titled PEAK EXPIRATORY FLOW RATE AND SOME ASPECT OF ECG AND HAEMATOLOGICAL VARIABLES IN NON-PREGNANT AND PREGNANT WOMEN AT SECOND AND THIRD TRIMESTERS OF PREGNANCY IN KURA LOCAL GOVERNMENT AREA OF KANO STATE by Ahmed Ibrahim Salisu meets the regulations governing the award of the Degree of Master of Science of Ahmadu Bello University Zaria, and is approved for its contribution to scientific knowledge and literary presentation.

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Dean, Postgraduate School Date

DEDICATION

I have dedicated this project work to my mother Hajiya Hassana Danjuma for the moral upbringing, support, courage and love she has for me.

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I thank Almighty Allaah so much for enabling me to carry out this research project work. I am forever grateful to my major and minor supervisors; Dr. A. B. Adelaiye and Dr. M. Babashani for their constructive criticism, comments and suggestions as academicians and physicians which have really improved the quality of this thesis. I am grateful to Dr. Aliyu Mohd for his cooperation as the Head of Department. I am also grateful to all the staffs of the Human physiology department especially Professor Abid Ali and Dr. Shola for their constructive criticism and supply of some relevant literatures. My appreciation also goes to Dr. Dauda Maigatari and Mallam Tanko Yusif for the moral and psychological support. I am forever grateful to Dr. Auwal Ibrahim Yola for sourcing the Electrocardiographic machine for me to carry out this research work. I must also register my appreciation to the Technical crew members that helped me to carry out this work, they are; Mallam Hassan Yusuf, Nura Danjuma, Shafa'atu Aminu, and Habiba Musa. The assistance of Dr. Lawan Umar, Dr. Isah Abubakar and Dr. Zubairu Ilyasu in the statistical analysis of the data is highly appreciated. I am grateful to Mallam Yusuf Gezawa of chemical pathology department of Murtala Mohd specialist hospital and Dr. Mohd Atiku Head of Biochemistry department, Bayero University, Kano, for their immense contribution to the laboratory works. Special thank goes to the staffs of the Kura comprehensive hospital especially the matron of the antenatal clinic, and OPD staff for helping me in data collection. My appreciation also goes to Mallam Bashir Chedi for his support and assistance to see this work completed. I am very grateful to my father in-law for sourcing the facility to process this work. Lastly, I must register my appreciation to my wife and my children for their patience and understanding during the period of project work.

ABSTRACT

Values of peak expiratory rate (PEFR), ECG and Haematological variables in 250 females at their reproductive ages, made up of 123 pregnant subjects and 127 non-pregnant control groups, living and/or working in Kura local government area and its environs were obtained.

Pearson's correlation coefficient between PEFR and ages as well as ECG, Haematological and other anthropometric measurements were calculated in both groups.

Multiple regression analyses were made to obtain the best prediction formulae using the measured variables in pregnant and non-pregnant groups. Furthermore, graphs were plotted to show the variation of PEFR with age in the subjects and controls with specific weights. Data for plotting the graphs were generated using the prediction formulae generated from this study and other prediction formulae generated from previous studies among Nigerians, whose best prediction formulae incorporated age and height. The graph illustrated the limitations of applying formulae derived from a different race, tribe and/or generation to an entirely different people with dissimilar characteristics. This lends credence to the need to make use of recent generated formulae for local predictions.

TABLE OF CONTENTS

CHAPTER		Page
	Title page	i
	Declaration	ii
	Certification	iii
	Dedication	iv
	Acknowledgements	v
	Abstract	vi
	Table of contents	vii
	List of Tables	x
	List of Figures	xi
	Appendix	xii
1	**INTRODUCTION**	1
1.0	Introduction	1
1.1	Statement of Research Problem	3
1.2	Justification	4
1.3	Aims and Objectives	4
1.4	Physical and Morphological Features of Respiratory System	5
1.4.1	The Air Passage	5
1.4.2	Respiratory Bronchioles (Seventeenth to Nineteenth Generations)	6
1.4.3	Alveolar Ducts (Twentieth to Twenty Second Generations)	6
1.4.4	Alveolar Sacs (Twenty third Generation)	7
1.4.5	Alveolar Surfactant	9
1.4.6	Lung Volumes and Capacities	10
1.4.7	Elastic Recoil of the Lungs	11
1.4.8	Compliance of the Lungs	12
1.4.9	The Nature of Forces Causing Recoil of the Lungs	13

1.5	Control of Breathing	15
1.5.1	Respiratory Centre	15
1.5.2	Neural Control of Breathing	16
1.5.3	Medullary Systems	17
1.5.4	Pontine and Vagal Influence	18
1.5.5	Chemical Control of Breathing	18
1.5.6	Carotid and Aortic Bodies	19
1.5.7	Chemoreceptor in the Brain Stem	19
1.5.8	Ventilatory Response to Changes in Acid-Base Balance	21
1.5.9	Ventilatory Response to Carbon Dioxide	21
1.5.10	Ventilatory Response to Oxygen Lack	21
1.6	Basic Electrophysiology of the Heart	22
1.6.1	Mechanical Properties of the Cardiac Muscle	24
1.6.2	Conduction System of the Heart	26
1.6.3	The Nomenclature of the Electrocardiograph Deflection	26
1.6.4	Lead Placement and Recording	27
1.6.5	The Limb Leads	27
1.6.6	Augmented Leads	27
1.6.7	Chest Leads	28
2	**LITERATURE REVIEW**	30
2.0	Pulmonary Function Overview	30
2.1	Anthropometric Data	30
2.2	Peak Expiratory Flow Rate	30
2.3	The Electrocardiograph (ECG)	33
2.4	Lipoproteins	35
2.5	Haematological Variables	36

3	**MATERIALS AND METHOD**	39
3.0	Equipment	39
3.1	Study Procedure	39
3.2	Methodology	42
3.2.1	The Basic Anthropometric Data	42
3.2.2	Blood Pressure	42
3.2.3	The Peak Expiratory Flow Rate	42
3.2.4	The ECG	43
3.2.5	Blood Sample	45
3.3	Limitations	45
3.4	Data Analysis	45
4	**RESULT**	46
5	**DISCUSSION**	59
6	**SUMMARY AND CONCLUSION**	64
6.1	Summary	64
6.2	Conclusion	65
6.3	Recommendations	66
REFERENCES		67
APPENDIX		73

LIST OF TABLES

Table 3.1	-	12-Lead ECG electrodes and leads	43
Table 4.1	-	The means values of all the measured variables in The subjects and controls	49
Table 4.2	-	Grouping of Subjects according to the number of Children.	50
Table 4.3	-	Grouping of Subjects according to the stage of Pregnancy.	50
Table 4.4	-	Grouping of Subjects and Controls according to their level of education	51
Table 4.5	-	Grouping of Subjects and Controls according to their occupation	51
Table 4.5	-	Age grouping with observed mean values of PEFR among each group.	52
Table 4.6	-	Pearson's correlation coefficient between PEFR and measured variables both subjects and controls	53
Table 4.7	-	Linear regression coefficient for predicting PEFR from variables in the subject grouping	54
Table 4.9	-	Liner regression measured variables in the controls.	55

LIST OF FIGURES

Figure 1.1	-	Lung Volumes and Capacities	11
Figure 1.2	-	Compliance of the Lungs	13
Figure 1.3	-	Action Potential of the Cardiac Muscle	24
Figure 1.4	-	Conduction system of the heart	25
Figure 1.5	-	Nomenclature of the ECG deflections	27
Figure 1.6	-	Limb leads and precordial leads	28
Figure 4.1	-	Variation of PEFR with age among the subjects	56
Figure 4.2	-	Variation of PEFR with age among the controls	56

ABREVIATION

AHA - American Heart Association

ERV - Expiratory Reserve Volume

FRC - Functional Residual Capacity

HDL - High Density Lipoprotein

IRV - Inspiratory Reserve Volume

LDL - Low Density Lipoprotein

NS - Not significant

P - Statistical level of significance

PCV - Packed Cell volume

R - Correlation Coefficient

RV - Residual Volume

T C - Total Cholesterol

TLC - Total Lung Capacity

TV - Tidal Volume

VC - Vital Capacity

WBC - White Blood Cell Count

CHAPTER ONE INTRODUCTION

1.0 Introduction

Ventilatory function tests enable a better understanding of pulmonary physiology in subjects of all ages, sex, professions and occupational groups, Ali (1983). The test can identify abnormalities of lung function that might otherwise be overlooked and can exclude the possibility of some respiratory disorder such as chronic obstructive pulmonary disease, Robert and Crapo (1994). Serial Measurement of lung function may be useful in tracking pulmonary expressions of diseases, quantifying responses to the therapy, and making early diagnosis of lung injury after occupational exposures and drug or radiation therapy. Many diseases, including heart failure, rheumatoid arthritis, inflammatory bowel disease, and vasculities, affect the lungs either directly or as a result of the adverse effects of treatment, Bates (1989). The changing pattern of environment, occupations, personal factors like education, and social factors, which come with development, are known to affect respiratory function, Melville et. al., (1981). Test quality remains the most important concern in lung function testing. Variability is greater in pulmonary function tests than in most other clinical laboratory tests because of the inconsistency of efforts by patients, Becklake (1986). Peak expiratory flow rate, which is measured with a simple, portable, relatively in expensive equipment that is independent of electricity, is still a suitable test for some important parameters of lung function useful in some parts of Africa where availability of more sophisticated medical facilities are poor Elebute & Femi (1971).

Importance of Pulmonary Function Test

i. It is useful in diagnosing and managing pulmonary diseases, e.g. asthma.

ii. It provides objective and quantifiable measures of lung function.

iii. It is used to evaluate and monitor diseases that affect the heart and lungs functions.

iv. It is used to monitor the effects of environmental, occupational, and drug exposures on the lung.

v. It is used to assess the risks of surgery.

vi. It assists in medical evaluation before employment or for insurance purposes.

Electrocardiogram

The electrocardiogram (ECG) is a graphic recording of the myocardial electrical potentials produced during the cardiac cycle. The cardiac muscle is unique among the muscles of the body in that it is functionally syncytial and contracts rhythmically in the absence of external innervations owing to the presence in the myocardium of pacemaker cells Sino- Atrial Node (SA Node) that discharges spontaneously. The impulses produced from the (SA Node) result in excitation of the muscle fibres throughout the myocardium. Impulse formation and conduction produce weak electric currents that spread through the entire body. By applying electrodes to various positions on the body and connecting these electrodes to an electrocardiography apparatus, the ECG is recorded. The connections of the apparatus are such that an upright deflection indicates positive potentials and a downward deflection indicates negative potential, Goldman (1976).

Because human body is a volume conductor, fluctuations in the electric potential that represent the algebraic sum of the action potentials of myocardial fibres can be recorded extra cellularly. Conventionally, 12 LEAD SYSTEM is recorded. These are

leads I, II, III, AVR, AVL, AVF, $V_1 - V_6$. They are put around the arms and legs and also on the chest to record the electrical activity in the heart.

Uses of ECG

i. It is a non-invasive method for the diagnosis and monitoring of cardiac arrhythmias, Braunwald (1997).

ii. It guides therapy and risk stratification for patients with suspected acute myocardial infarction, AHA Guidelines (2005).

iii. It helps detect electrolyte disturbances e.g. hyperkalamia and hypokalemia, Clinical value (2004).

iv. It allows for the detection of conduction abnormalities e.g. right and left bundle branch block, Braunwald (1997).

v. It is used as a screening tool for ischaemic heart disease during cardiac stress test, Braunwald (1997).

1.1 Statement of Research Problem

Pulmonary disease is a major cause of ill-health throughout the world, Sovijarrni et. Al., (2000).Cardiopulmonary diseases associated with pregnancy in the United Kingdom, contributes significantly to maternal morbidity and mortality, Mc Fall (1988). In the last three decades in UK, the number of deaths due to cardiac diseases complicating pregnancy was comparable to those from thromboembolic disorders and greater than those due to ante partum haemorrhage and hypertensive disorders combined, (Danny, 1996). From the unpublished medical records of the Murtala

Mohd Specialist hospital in Kano State, it was shown that the number of deaths due to pre eclampsia in the year 1995 is up to 2.3%.

1.2 Justification

Limited knowledge is available on normal values of cardiopulmonary parameters of pregnant women in Northern Nigeria and Kano State in particular. Therefore, this study will provide some data on basic cardiopulmonary and haematological parameters of pregnant women in this part of the country. It will also provide some input into normal reference values among pregnant women in Kano State.

1.3 Aims/Objectives

1. To determine normal values of PEFR in healthy pregnant women in Kura Local Government Area, Kano State.
2. To assess the effect of pregnancy on PR- interval, QRS complex, QTc duration and Electrical axis of the ECG recordings of apparently healthy pregnant women in Kura LGA.
3. To assess the effect of pregnancy on Packed cell volume (PCV), White cell count WBC) and Serum Lipids.
4. To find out whether there is an association or not between the various anthropometric parameters and the measured PEFR.
5. To obtain the prediction formula for the PEFR in our environment from the locally generated data.

1.4 Physical and Morphological Features of Respiratory System

1.4.1 *The Air Passages*

Simplified accounts of lung function distinguish sharply between conducting air passages and areas in which gas exchange takes place. In fact, no such sharp demarcation occurs and the air passages gradually change their character showing a transition from the trachea to the alveoli, with the role of conduction gradually giving way to the role of gas exchange Nunn (1993).

Between the trachea and the alveolar sacs, the airways divide into 23 generations. The first 16 generations of the air passages form the conducting zone of the airways that transports gas from and to the exterior. They are made up of the trachea, bronchi, bronchioles, and terminal bronchioles. The remaining seven generations form the transitional and respiratory zones where gas exchange occurs and are made up of respiratory bronchioles, alveolar ducts, and alveoli. These multiple divisions greatly increase the total cross-sectional area of the airways, from $2.5cm^2$ in the trachea to $11{,}800cm^2$ in the alveoli, Ganong (2005).

The trachea and bronchi have cartilages in their walls but relatively little smooth muscle. They are lined by ciliated epithelium that contains mucous and serous glands. Cilia are present as far as the respiratory bronchioles, but glands are absent from the epithelium of the bronchioles and terminal bronchioles, and their walls do not contain cartilage. However, their walls contain more smooth muscle, of which the largest amount relative to the thickness of the wall is present in the terminal bronchioles.

The walls of the bronchi and bronchioles are innervated by the autonomic nervous system. There are abundant muscarinic receptors, and cholinergic discharge causes broncho constriction. There are β_2 adrenergic receptors in the bronchial epithelium and smooth muscles. The β_2 receptors mediate bronchodilation and increase bronchial secretion, while α adrenergic receptors inhibit secretion.

1.4.2 *Respiratory Bronchioles (Seventeenth to Ninetieth Generations)*

Down to the end of the bronchioles, the function of the air passages is solely conduction and humidification. At the next generation (first order respiratory bronchioles) gas exchange occurs to a small extent and this function increases progressively through the three generations of respiratory bronchioles until, in the first order alveolar duct (twentieth generation), the entire surface is devoted to gas exchange. The epithelium is cuboidal between the mouths of the mural alveoli but becomes progressively flatter until it finally gives way entirely to alveolar epithelium in the alveolar ducts. The respiratory bronchioles are embedded in lung parenchyma and rely upon tissue traction for maintenance of their lumen. There is well-marked muscle layer and the muscle forms bands, which loop over the opening of the alveolar ducts and the mouths of the mural alveoli.

1.4.3 *Alveolar Ducts (Twentieth to Twenty-Second Generations)*

Alveolar ducts arise from the terminal respiratory bronchioles from which they differ by having no mucosa at all. The walls are in fact composed entirely of alveoli, which open widely on to the lumen of the alveolar duct and are capable of contraction causing considerable narrowing of the lumen of the duct. About half of the total

number of alveoli arises from ducts and some 35 percent of the alveolar gas resides in the alveolar ducts and the alveoli which arise directly from them, Nunn (1993).

1.4.4 *Alveolar Sacs (Twenty-Third Generation)*

The last generation of the air passages is designated alveolar sacs although they are functionally identical to alveolar ducts except for the fact that they are blind. The mean number of alveoli ranges from about 200 to 600 million, correlating with the height of the subject, Angus and Thurlbeck (1972). The size of the alveoli is proportional to the lung volume; however, it is not uniform throughout the lungs but is largest in the uppermost part of the lung and smallest in the most dependent parts, Glazier et al, (1967).

Alveolar walls, which separate two adjacent alveoli, consist of two layers of alveolar epithelium on separate basement membranes enclosing the interstitial space. This space contains the pulmonary capillaries, elastic and collagen fibres and nerve endings. The interstitial space is asymmetrically disposed with relation to the capillaries, Nunn (1993).

On the side of the capillary, the space tends to be more than 1-2um thick and to contain abundant collagen fibres. This may be considered as the ‘service side’ of the capillary and forms the connective tissue framework, which maintains the geometry of the alveolus. On the other side of capillary, endothelium and epithelium are closely apposed and the total thickness of the alveolar-capillary membrane is usually less than o.4 μm. This may be considered as the ‘active side’ of the capillary and the gas exchange function is presumably more efficient on this side. The distinction between

active and service sides has considerable pathophysiological significance, since the active side tends to be spared in pulmonary edema.

The alveolar epithelium is covered with a very thin film of alveolar lining fluid, which forms an interface with the alveolar gas. Surface tension at the interface tends to make the alveolar septa contact and this constitutes a major fraction of the lung 'elasticity'. The surface tension is, however, modified in a most important manner by a lipoprotein secreted by the alveolar epithelium called surfactant.

A number of structurally distinct cells may be identified at the alveolar level, these include:

1. *Capillary endothelial cells*: These are continuous with the general circulatory endothelial cells and, in pulmonary capillary bed, have a thickness of only about 0.1μm, except where thickened to contain nuclei.

2. *Alveolar squamous epithelial cells*: Type1. These cells constitute the outermost layer of the parenchyma of the lungs exist as a tin sheet approximately 0.1μm in thickness except where expanded to contain the nuclei. Type 1 cells have a relatively clear cytoplasm, which is devoid of organelles other than pinocytic vesicles.

3. *Type II alveolar cells*: (syn. Cuboidal or septal cells). These cells are probably the main site of release of the surfactant which modifies the surface tension of the alveolar lining fluid. They are characteristically sited at the junction of septa and have large nuclei and microvillus on their surfaces. They also

contain large osmiophilic striated organelles, which are probably concerned with storage of the phospholipids.

4. *Type III alveolar brush cells*: These are only rarely seen and their function is not yet established.

5. *Alveolar macrophages*: These are phagocytes, which are active in combating infection and in scavenging foreign bodies such as dust particles. The alveolar macrophages pass through the alveolar epithelium and lie within the alveolar living fluid. They contain a variety of destructive enzymes and their death presents a danger of auto digestion of the alveolar septa, but this is normally prevented by the protease transport system.

6. *Mast cells*: In common with other organs, the lungs contain numerous mast cells, which are located in the sub pleural area. They have well-developed organelles which secrete heparin, histamine, dopamine and 5-hydroxytryptamine (serotonin).

1.4.5 *The Alveolar Surfactant.*

The strange features of the surface tension of the alveolar living fluid are due to the presence of a surface active material called surfactant. In fact, a group of phospholipids are responsible for this activity and the most important is lecithin, but sphingomyelin, phosphatidyl inositol and phosphatidyl dimethylethanolamine are also found Gluck (1971). Pulmonary surfactant is highly insoluble and floats on the surface of the alveolar lining fluid, where it offers no appreciable resistance to the

diffusion of gases such as oxygen, carbondioxide and anaesthetics. Mainly the type II alveolar lining cells secrete it. Surfactant exerts a surface pressure which counteracts the surface tension of the alveolar living fluid itself, and this effect is greater when the surface film is reduced in area and the concentration of lecithin at the surface is raised.

It is now well established that surfactant levels are diminished in respiratory distress syndrome of the newborn, formerly known as hyaline membrane disease, Avery and Mead (1959); pattle et al., (1962). The quantity of lecithin in the amniotic fluid rises sharply late in gestation and, if the child is born before this occurs, there is a risk of respiratory distress syndrome, Gluck et al., (1971). In contrast the amount of sphingomyelin remains constant and the lecithin/sphingomyelin (L/S) ratio in the amniotic fluid is a useful measure of the level of lecithin.

1.4.6 *Lung Volumes and Capacities*

The lung volume is considered in relation to three volumes which are relatively fixed for a particular subject under particular conditions. These volumes are:

1. Total lung capacity (TLC), which is the volume of gas in the lungs at the end of a maximal inspiration.
2. Functional residual capacity (FRC), which is the volume of gas in the lungs at the end of a normal expiration.
3. Residual volume (RV), which is the volume of gas in the lungs at the end of a maximal expiration

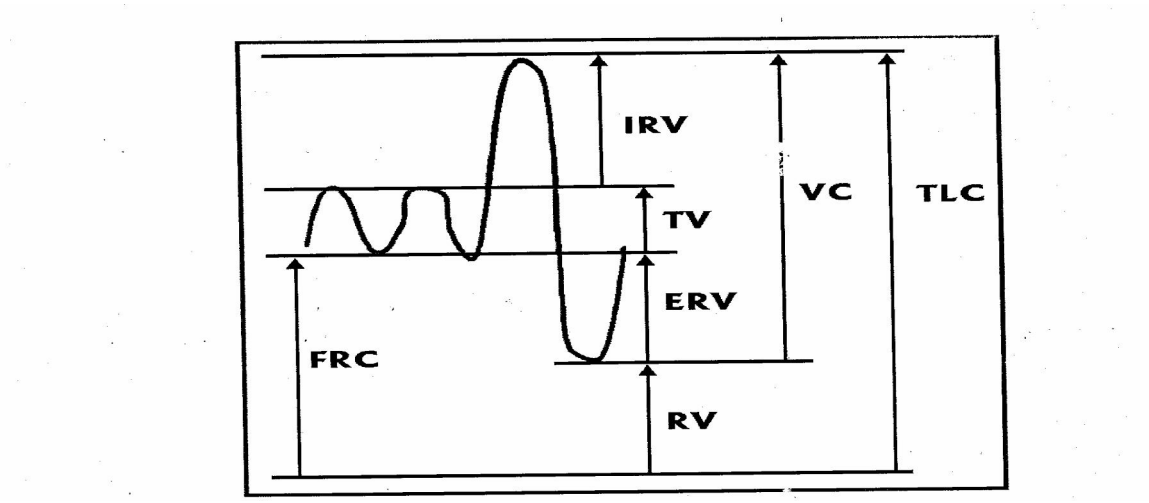


Figure 1: Adapted from Nunn (1993).

1.4.7 *Elastic Recoil of the Lungs*

The Transmural Pressure Gradient: At all lung volumes, the pressure within the alveoli is greater than that surrounding the lungs, Nunn (1993). The difference (the transmural pressure gradient) is related to the lung volume, and as the lung volume diminishes, three important changes take place.

1. The Trans mural pressure gradient diminishes.
2. The small air passages become narrower and one by one become totally obstructed. Airway closure commences at the closing capacity.
3. There is a tendency for alveoli to collapse.

Closure of the smaller air passages tends to prevent total collapse of the alveoli, unless the sequestered gas trapped therein can be removed by the pulmonary blood flow, which continues to perfuse the alveoli after the air passages are collapsed. This is most likely to happen if the alveoli contain high concentrations of oxygen or soluble anesthetic gases such as cyclopropane and nitrous oxide. Absorption is very slow if the trapped alveoli contain air, Webb and Nunn (1967).

1.4.8 *Compliance of the Lungs*

The extent to which the lungs expand for each unit increase in trans pulmonary pressure is called their compliance. The total compliance of both lungs together in the normal adult human being averages about 200 milliliters of air per centimeter of water trans pulmonary pressure, Guyton (2000). That is, every time the trans pulmonary pressure increases by 1 centimeter of water, the lung volume expands 200 milliliters.

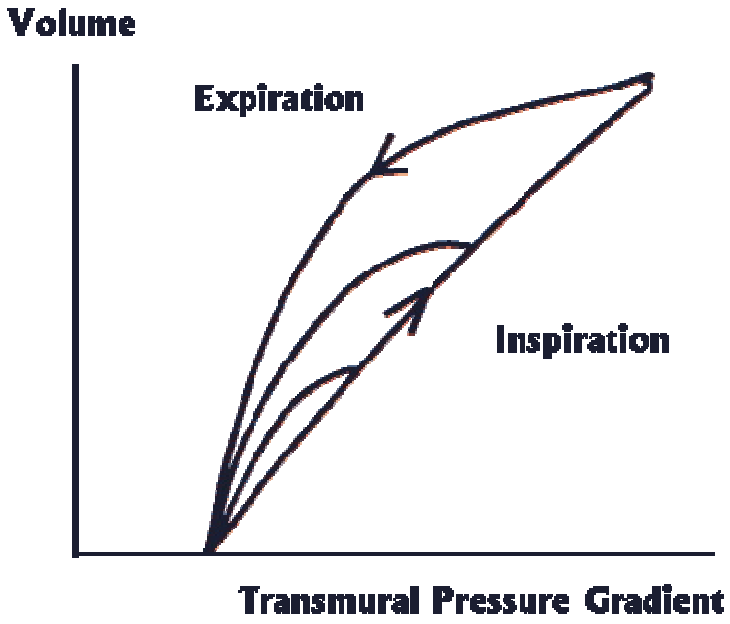


Figure 2: Adapted from West (1990).

The relationship shown above between lung volume and trans mural pressure gradient is typical of an elastic structure. Hooke's law states that stress is proportional to strain. SIn this case the stress is the change in the trans mural pressure gradient, and the strain is the change in the lung volume. The relationship is valid over limited ranges, such as that encountered in a normal breathing and may be stated as follows.

$$\frac{\text{Change in lung volume}}{\text{Change in trans mural pressure gradient}} = \textbf{a constant}$$

The constant is known as the **compliance.**

1.4.9 *The Nature of Forces Causing Recoil of the Lungs*

For many years it was believed that the recoil of the lungs was due entirely to stretching of the yellow elastin fibres present in the lung parenchyma. However, VON Negard (1929), showed that much of the elastic recoil' was due to surface tension acting throughout the vast air/water interface lining the alveoli. Surface tension at an air/water interface produces forces which tend to reduce the area of the interface. Thus, in a bubble the gas pressure within the bubble will be higher than the surrounding pressure, because the surface of the bubble is in a state of tension. Alveoli resemble bubbles in this respect but differ in their being connected with the exterior through the system of air passages.

The pressure in a bubble is above ambient by an amount depending on the surface tension of the liquid and the radius of curvature of the bubbles, according to the laplace equation.

$$P = \frac{2T}{R}$$

Where P is the pressure within the bubble (dyn/Cm^2), T is the surface tension of the liquid (dyn/Cm), and R is radius of the bubble (Cm).

Although it appears inevitable that surface tension must make a contribution to the retractive forces of the lung, two difficulties must be resolved. The first problem is

that the pressure in small bubbles is higher than in large bubbles, a conclusion that stems directly from the laplace equation. From this one might expect that the pressure in a small alveolus would be greater than in a large one and would result in a progressive discharge of alveolus into a larger one, until eventually only one gigantic alveolus would be left. Instability would be inevitable except in the unlikely event of all the alveoli being exactly the same size. Such a state of affairs would not, of course, be compatible with life, and no such instability exists in the normal lung.

The second problem concerns the relationship between lung volume and the trans mural pressure gradient. According to the laplace equation, one would expect the retractive forces of the lung to increase as the lung volume decreased, a relationship, which is certainly true of a bubble. If this were true of the lung, one would expect the lung to decrease in volume with tendency to collapse increasing progressively as the lung volume diminished. This of course, dose not happen in the normal lung and the retractive forces decrease as the lung volume is reduced.

The problems were resolved by von Neergard (1929), who concluded that the surface force in the alveolar lining fluid must be less than would be expected from the properties of simple liquids and, furthermore, that the surface tension must be variable. The original observations of pattle (1995) on bubbles in lung froth and later studies on alveolar extracts; Brown, Johnson and Clements (1959), have demonstrated that the surface tension of the alveolar lining fluid is variable and decreases as its surface area is reduced, to attain very low levels which are well below the normal range for body fluids such as plasma.

1.5 Control of Breathing

The nervous system normally adjusts the rate of alveolar ventilation almost exactly to the demands of the body so that the partial pressure of oxygen (PO_2) and that of carbondioxide (PCO_2) in the arterial blood are hardly altered even during exercise and most other types of respiratory stress, Wasserman (1986).

1.5.1 *Respiratory Centre*

Conventionally, the neural mechanisms that regulate ventilation are considered to be a hierarchy, with more primitive processes regulated by the higher centres Euler (1986). The central feature of this scheme is the respiratory pattern generator, which is believed to lie within the medulla oblongata, Richter (1982). Here, at least three groups of neurons receive inputs from important chemoreceptors and mechanoreceptors. Those going to the dorsal respiratory group are predominately inspiratory but are not influenced by stretch receptor inputs. Those in the ventral respiratory group are also thought to be inspiratory, at least in the upper part of this complex, but activate expiratory muscles in the lower part. Finally, neurones in the pontine respiratory group appear to cover the period during late inspiration and early expiration and are important in terms of respiratory rhythm generation. A basic rhythmic oscillatory pattern is developed between these neuronal groups, which acts as a form of respiratory "pacemaker" but this is greatly modulated by the nature and severity of the inputs from the other peripheral sensors, Remmers (1999). Mechanical and chemical inputs arise from changes in chest-wall movement and/or blood gas tensions provide the feedback, which modify the intensity and timing of the respiratory neural outputs, Bisgard (1995).Spontaneous respiration is produced by rhythmic discharge of motor neurons that innervate respiratory muscles. This

discharge is totally dependent on nerve impulses from the brain. The rhythmic discharges from the brain that produce spontaneous respiration are regulated by alterations in arterial PO_2, PCO_2 and H^+ concentration, and this chemical control of breathing is supplemented by a number of non chemical influences, Richter (1982).

1.5.2 *Neural Control of Breathing*

Two separate neural mechanisms regulate respiration. One is responsible for voluntary control and the other for automatic control, Ganong (2005).SThe voluntary system is located in the cerebral cortex and send impulses to the respiratory motor neurons via the corticospinal tracts.

The nerve fibres mediating inspiration converge on the phrenic motor neurons located in the ventral horns from C3 to C5 and the external intercostals motor neurons in the ventral horns throughout the thoracic cord. The fibres concerned with expiration converge primarily on the internal intercostal motor neurons in the thoracic cord, Ganong (2005).

The motor neurons to the expiratory muscles are inhibited when those supplying the inspiratory muscles are active, and vice versa. Although spinal reflexes contribute to these reciprocal innervations, it is due primarily to activity in descending pathways. Impulses in these descending pathways excite agonists and inhibit antagonists, Guyton (2000).

1.5.3 *Medullary Systems*

Rhythmic discharge of neurons in the medulla and pons produces automatic respiration. Brain stem respiratory neurons are of two types: those that discharge

during inspiration (I neurons) and those that discharge during expiration (E neurons). Many of these discharge at increasing frequencies, and some discharge at the same high rate during inspiration or expiration. However, expiration is passive during quire breathing, and E neurons are quite: they become active only when ventilation is increased, Ganong (2005).

The area in the medulla that is concerned with respiration has classically been called the respiratory centre, but there are actually two defined groups of respiratory neurons: The dorsal group of neurons is located in and near the nucleus of the tractus solitarius. The ventral group is a long column of neurons that extends through the nucleus ambiguous and the nucleus retroambiguus in the ventrolateral medulla. The dorsal groups is made up primarily of I neurons, some of which project monosynaptically to the phrenic motor neurons Ganong (2005).

They probably receive afferents from the airways and carotid and aortic bodies, which terminate in the nucleus of tractus solitarius. The ventral group has E neurons at its caudal end, I neurons in its mid portion, and E neurons at its rostral end. Some of these neurons project to respiratory motor neurons. Those in the rostral end of the group appear to inhibit I neurons during inspiration.

1.5.4 *Pontine and Vagal Influences*

Although the rhythmic discharge of medullary neurons concerned with respiration is spontaneous, it is modified by neurons in the pons and afferents in the vagus from receptors in the airways and lungs. An area known as the pneumotaxic centre in the medial parabrachial nucleus of t he dorsolateral pons contains I and E neurons as well as neurons that are active in both phases of respiration. The function of the

pneumotaxic centre is probably involved in switching between inspiration and expiration Ganong (2005).

Stretching of the lungs during inspiration initiates impulses in afferent pulmonary vagal fibres. These impulses inhibit inspiratory discharge.

1.5.5 *Chemical Control of Breathing*

The ultimate goal of respiration is to maintain proper concentrations of oxygen, carbon dioxide, and hydrogen ions in the tissues. It is fortunate, therefore, that respiratory activity is highly responsive to changes in each of these, Kafer (1975).

Excess carbon dioxide or excess hydrogen ions in the blood mainly act directly on the respiratory centre itself, causing greatly increased strength of both the inspiratory and the expiratory motor signals to the respiratory muscles, Kafer (1975).

Oxygen in contrast, does not have a significant direct effect on the respiratory centre of the brain in controlling respiration, Aubier et al., (1980). In stead, it acts almost entirely on the peripheral chemoreceptors located in the carotid and aortic bodies, and these in turn transmit appropriate nervous signals to the respiratory centre for control of respiration, Aubier et al., (1980).

1.5.6 *Carotid and Aortic Bodies*

There is a carotid body near the carotid bifurcation on each side, and there are usually two or more aortic bodies near the arch of the aorta. These are special nervous chemical receptors that are located outside the brain. They are especially important for detecting changes in oxygen in the blood, although they also respond to a lesser

extent to changes in carbon dioxide and hydrogen ion concentration. The chemoreceptors in turn transmit nervous signals to the respiratory centre in the brain to help regulate the respiration, Poon (1989).

The carotid bodies are located bilaterally in the bifurcations of the common carotid arteries, and their afferent nerve fibres pass through Hering's nerves to the glossopharyhgeal nerves and then to the dorsal respiratory area of the medulla. The aortic bodies are located along the arch of the aorta; their afferent nerve fibres pass through the vagi also to the dorsal respiratory area, Ganong (2005).

1.5.7 *Chemoraceptors in the Brains Stem*

If alveolar ventilation is inadequate and arterial oxygen tension PO_2 falls then the peripheral chemoreceptors at the junction of the external and common carotid arteries increase their discharge rate Bisgard (1995). One of the most consistent findings in animal studies is the relationship between the PO_2 and the carotid sinus nerve traffic and this clearly shows that these receptors are only significantly activated once the PO_2 falls below 8.0 kPa, Biscoe et al. (1970). However, some tonic activity does persist as this can be abolished by oxygen therapy and this may be particularly important during exercise. Increases in arterial CO_2 tension PCO_2 stimulates both peripheral, but especially central chemoreceptor, firing rate Bruce (1987). It is still difficult in humans to distinguish the effect of CO_2 on the central chemoreceptors from those on the peripheral chemoreceptors and brief stimulation, as used in many tests of ventilatory control, may notgive an appropriate index of more sustained hypercapnia, as happens in disease, Easton (1986).The chemoreceptors that mediate the hyperventilation produced by increases in arterial PCO_2 after carotid and aortic

bodies are denervated are located in the medulla oblongata and consequently are called medullary chemoreceptors. They are separate from the dorsal and ventral respiratory neurons and are located on the ventral surface of the medulla.The chemoreceptors monitor H^+ concentration of CSF, including the brain interstitial fluid. CO_2, readily penetrates membranes, including the blood-brain barrier, where as H^+ and HCO_3^- penetrate slowly. The CO_2 that enters the brain and CSF is promptly hydrated. The H_2CO_3, dissociates, so that the local H^+ concentration rises. The H^+ concentration in brain interstitial fluid parallels the arterial PCO_2. Experimentally produced changes in the PCO_2 of CSF have minor, variable effects on respiration as long as the H^+ concentration is held constant, but any in crease in spinal fluid H^+ concentration stimulates respiration. The magnitude of t he stimulation is proportionate to the rise in H^+ concentration. Thus, the effects of CO_2 on respiration are mainly due its movement into the CSF and brain interstitial fluid, where it increases the H^+ concentration and stimulates receptors sensitive to H^+.

1.5.8 *Ventillatory Responses to Changes in Acid-Base Balance*

The degree of acid base compensation within the cerebrospinal fluid is an important determinant of the effects of raised carbon dioxide and this is relevant for those individuals who develop persistent hypercapnia or slowly worsening hypercapnia in the course of an episode of respiratory failure, Bledsoe (1981).In metabolic acidosis due, for example, to the accumulation of the acid ketone bodies in the circulation in diabetes mellitus, there is pronounced respiratory stimulation (Kussmaul breathing). The hyperventilation decreases alveolar PCO_2 (blows off CO_2) and thus produces a compensatory fall in blood H^+ concentration. Conversely, in metabolic alkalosis due, for example, to protracted vomiting with loss of HCL, from the body, ventilation is depressed and the arterial PCO_2 raise, raising the H^+ concentration toward normal. If

there is an increase in ventilation that is not secondary to a rise in arterial H^+ concentration, the drop in PCO_2 lowers the H^+ concentration below normal (respiratory alkalosis); conversely, hypoventilation that is not secondary to a fall in plasma H^+ concentration causes respiratory acidosis, Bledsoe (1981).

1.5.9 *Ventilatory Responses to CO_2*

The arterial PCO_2 is normally maintained at 40mm Hg. When there is a rise in arterial PCO_2 as a result of increased tissue metabolism, ventilation is stimulated and the rate of pulmonary excretion of CO_2 is increased until the arterial PCO_2 falls to normal, shutting off the stimulus. The operation of this feedback mechanism keeps CO_2 excretion and production in balance, Kafer (1975).

1.5.10 *Ventillatory Response to Oxygen Lack*

When the O_2 content of the inspired air is decreased, there is an increase in respiratory minute volume. The stimulation is slight when the PO_2 of the inspired air is more than 60mmHg, and marked stimulation of respiration occurs only at lower PO_2 values. However, any decline in arterial PO_2 below 100mmHg produces increased discharge in the nerves from the carotid and aortic chemoreceptors. There are two reasons why in normal individuals this increase in impulse traffic does not increase ventilation to any extent until the PO_2 is less than 60mmHg. Because Hb is a weaker acid than HbO_2, there is a slight decrease in the H^+ concentration of arterial blood when the arterial PO_2 falls and haemoglobin becomes less saturated with O_2, Bledsoe (1981). The fall in H^+ concentration tends to inhibit respiration. In addition, any increase in ventilation that does occur lowers the alveolar PCO_2, and this also tends to inhibit respiration. Therefore, the stimulatory effects of hypoxia on ventilation are not

clearly manifest until they become strong enough to override the counter balancing inhibitory effects of a decline in arterial H^+ concentration and PCO_2.

1.6 Basic Electrophysiology of the Heart

Resting membrane and action potentials of cardiac electrical activity is determined by trans membrane potential. This is the potential difference between the intracellular and extracellular environments. The potential difference exists because of the selectively permeable cardiac cell membrane. This membrane is a lipid bilayer containing special proteins allowing passage of certain ions at certain times between the intra-and extra cellular spaces.

The resting membrane potential of individual mammalian cardiac muscle cells is about -90Mv, interior negative to exterior, Ganong (2005). Stimulation produces a propagated action potential that is responsible for initiating contraction. Depolarization proceeds rapidly, and an overshoot is present, which is followed by a plateau before the membrane potential returns to the baseline.

As in other excitable tissues, changes in the external K^+ concentration affect the resting membrane potential of cardiac muscle, where as changes in the external Na^+ concentration affect the magnitude of the action potential. The initial rapid depolarization and the overshoot (phase 0) are due to opening of voltage-gated Na^+ channels similar to that occurring in nerve and skeletal muscle. The initial rapid depolarization (phase 1) is due to closure of Na^+ channels. The subsequent prolonged plateau (Phase 2) is due to a slower but prolonged opening of voltage-gated Ca^{2+} channels. Final depolarization (Phase 3) to the resting membrane potential (phase 4) is

due to closure of the Ca^{2+} channels and K^+ efflux through various types of K^+ channels.

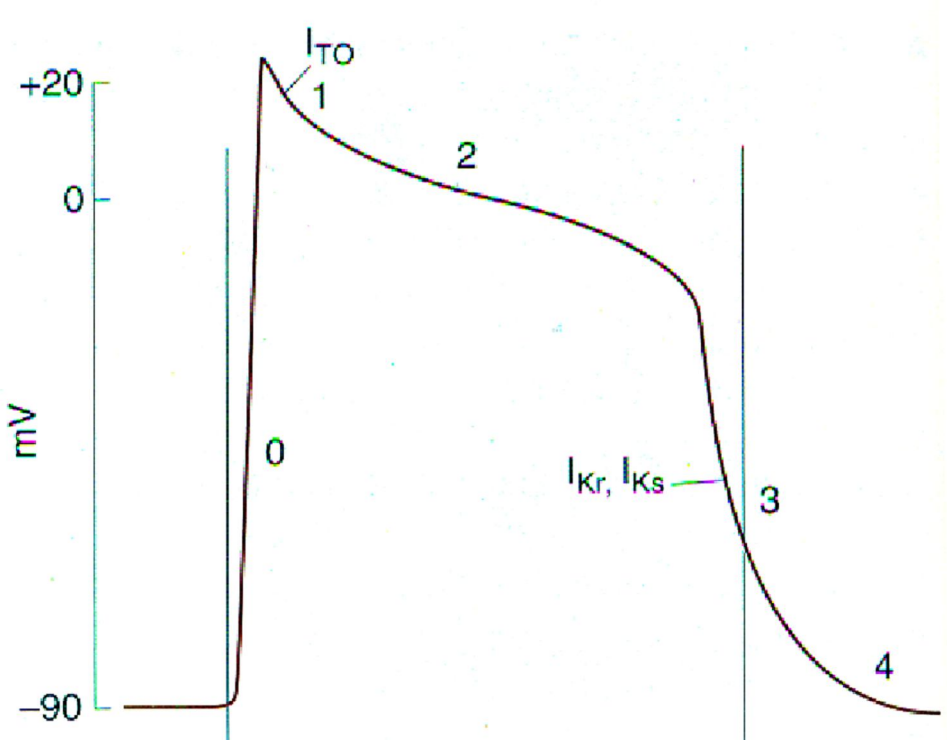


Figure 3: Action potential of a cardiac muscle fibre. Key: O, depolarization; 1, initial rapid depolarization; 2, plateau phase; 3, late rapid depolarization; 4, baseline. Adapted from Ganong (2005).S

1.6. Mechanical Properties of the Cardiac Muscle

The contractile response of cardiac muscle begins just after the start of depolarization and lasts about 1.5 times as longs as the action potential. The role of Ca^{2+} in excitation contraction coupling is important in strengthening of cardiac muscle contraction.

During phases (0-2) and about half of phase 3 (until the membrane potential reaches approximately -50mv during repolarization), cardiac muscle cannot be excited again; i.e., it is in its absolute refractory period. After, it remains relatively refractory until phase 4. Therefore, tetanus of the types seen in skeletal muscle cannot occur.

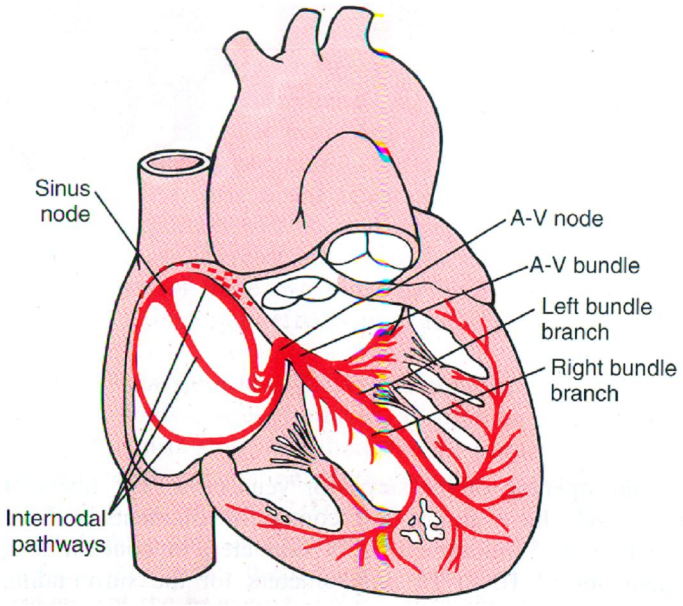


Figure 4: Sinus node of the Purkinje system of the heart. Adapted from Guyton 2000.

1.6.2 *Conduction System of the Heart*

The parts of the heart normally beat in orderly sequence: Contraction of the atria (atrial systole) is followed by contraction of the ventricles (ventricular systole), and during diastole all four chambers are relaxed. The structures that make up the conduction system are Sino atrial node (SA node), the inter nodal atrial pathways, the atrioventricular node (AV node), the His bundle and its branches, and the purkinje system.

Sino atrial node (SA node): This is the cardiac pacemaker. It is located at the junction of the superior vena cava with the right atrium. Blood supply is from the right

coronary artery in 55% of cases and the circum flex branch of the left coronary artery in 45%. Impulses spread from the SA node over the atria from right to left and top to bottom. They complete the atria depolarization in 80 – 100ms.

Atrioventricular (AV) node: The AV node is located in the right posterior portion of the intertribal septum. It is normally the only conducting pathway between the atria and ventricles and continuous with bundle of His. Blood supply is from the right coronary artery in 90% of cases.

HIS-purkinje system and ventricle: Rapid spread of impulses through the ventricles is mediated by cells of the HiS-Purkinje system (HPS). The AV node terminates on top of the HiS-bundle, which then divides in to a left and right bundle branches. The left bundle branch divides into an anterior fascicle and a posterior fascicle. The branches and fascicles run sub endocardially down either side of the septum and come into contact with the purkinje system, whose fibres spread to all parts of the ventricular myocardium. The entire mass of ventricular myocardium is depolarized in about 80-100ms. HPS blood supply is almost entirely from the left anterior descending artery; the proximal bundle may have a right coronary supply.

S1.6.3 *The Nomenclature of the Electrocardiograph Deflections*

The normal electrocardiogram is composed of a P wave, a QRS Complex, and a T wave. The QRS complex is often three separate waves, the Q wave, the R wave, and the S wave, but not always, Guyton and Hall (2000).

Waves and intervals

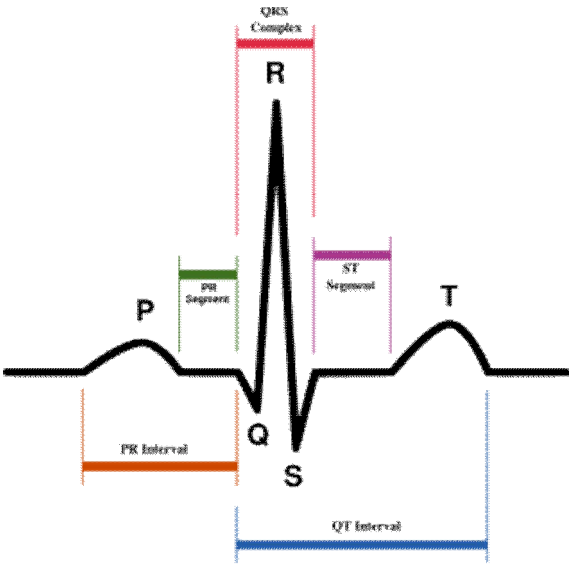


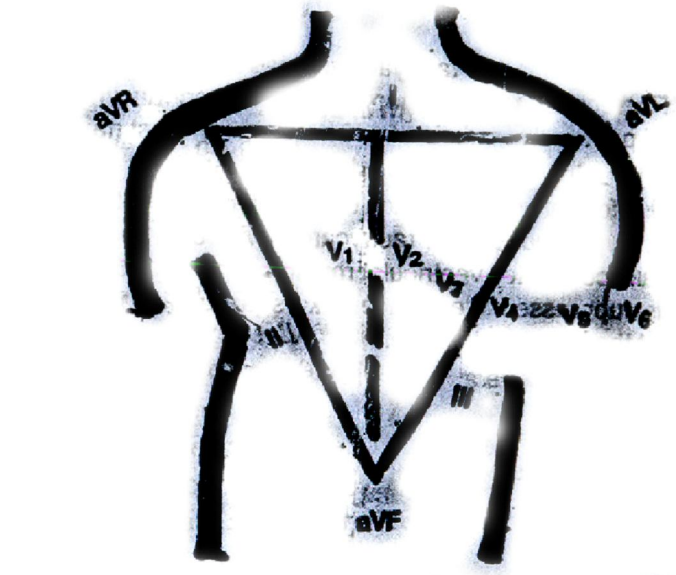
Figure 5: Nomenclature of ECG Deflections. Adapted from Sabatine (2000)

The P wave is caused by electrical potentials generated when the atria depolarized. This normally precedes atrial contraction. The QRS complex is caused by potentials generated when the ventricles depolarize before their contraction that is, as the depolarization wave spreads through the ventricles. Therefore, both the P wave and the components of the QRS complex are depolarization waves. The T wave is caused by potentials generated during ventricular repolarization.

1.6.4 *Lead Placement and Recording*

Conventionally, 12 LEAD SYSTEMS are recorded. These are leads I, II, III, AVR, AVL, AVF, $V_1 - V_6$.

The two arms and the two legs are connected through leads to the ECG machine. The right foot does not take part in the recording, so it acts as a negative (zero potential) lead. The three leads. Right arm, left arm and left foot when connected form an equilateral triangle with the heart in the centre as described by Einthovens.



Einthovens Equilateral Triangle and the Precordial Leads.

Figure 1.6: Relationship of the limb leads and precordial leads. Adapted from Olarenwaju P (2001).

1.6.5 *The Limb Leads*

Leads I, II and III measure differences of electrical potentials between two selected points, that is, they are bipolar leads. Lead I records potential difference between left arm (LA) and right arm (RA). In this case, the positive electrode is on LA while the negative is on RA.

Lead II records potential difference between LL and RA Lead III records potential difference between LL and LA. The augmented limb leads (AVR, AVL, and AVF) and chest leads ($V_1 - V_6$) are unipolar leads.

1.6.6 *Augmented Leads*

The arrangement of the augmented leads is such that the RA, LA and LL are connected through a 5000hms resistance to cancel out the potentials from them, resulting in a common Terminal of Willis, which is an indifferent electrode or inactive electrode. Another electrode can now be connected as an exploring electrode, which functions as active electrode (VR, VL or VF). By special mechanism the voltage obtained is augmented to obtain the AVR, AVL and AVF. The augmentation allows significant visibility of the voltage of the heart, which is at a distance from the foot (AVF) or right arm (AVR).

The standard limb leads I, II, III and augmented leads AVR, AVL and AVF measure activities of the heart in frontal plane.

1.6.7 *Chest Leads*

The chest leads are placed on the horizontal plane of the body. The American Heart Association recommendations on the precordial leads are as follows:

V_1: 4th Intercostals space, right of sternum

V_2: 4th intercostals space, left of the sternum

V_3: Midway between V_2 and V_4.

V_4: 5th intercostals space, at cardiac apex

V_5: 5th intercostals space, left anterior axillary line.

V_6: 5th intercostals space, left mid-axillary line

CHAPTER 2 LITERATURE REVIEW

2.0 Pulmonary Function Overview

Pulmonary function tests provide objective, quantifiable measures of lung function. They are used to evaluate and monitor diseases that affect heart and lung function, to monitor the effects of environmental, occupational, and drug exposures, to assess risks of surgery, and to assist in evaluations performed before employment or for insurance purposes.

Lung function tests included in many evaluations of fitness and some routine physical examinations. Such testing may provide interesting information about the physiological state of an individual. Monitoring is useful only when adequate base-line studies are available for comparison.

2.1 Anthropometric Data

Anthropometric measurements such as age, height, weight and diastolic blood pressure affect the lung function, Onadeko, falase and Ayeniyi (1976). Similarly, the changing pattern of environment like urbanization, occupations, personal factors like education and exercise and social factors like cigarette smoking, all of which have come with development, are known to affect respiratory, function, Rossiter (1974).

2.2 Peak Expiratory Flow Rate

Substantial racial differences in various aspects of respiratory function have been noted in various studies, with Blacks and Asiatics having lower values than their Caucasians counter parts; Schoenberg et al., (1978), Wodcock et. Al., (1972) and Miller et al., (1972). Published work on peak expiratory flow rate (PEFR) on

Africans; Oduntan (1970), Femi-pearse and Elebute (1971), Onadeko, Falase and Ayeniyi (1976); and studies on Nigerians living in Port harcort and its environs Njoku & Anah (1999) and in Zaria, Ali (1983) show similar trend. Several hypotheses have been put forward for the smaller lung volumes among normal black subjects. These include; Genetic (sex, ethnic group); Altitude of residence; Activity, training, muscular development, atmospheric pollution and smoking. Danny (1966) found that black army vehicle drivers had a 20% smaller chest expansion when compared to height-matched white drivers. He also found a 3% shorter sitting height and trunk height among the black drivers, but felt that this was insufficient to account for the nearly 13% difference in vital capacity. It thus appears likely that there is a difference in lung size between black and white subjects, the former having smaller lungs given the same standing height. Paul, Felcher and Addison (1960) measured chest size and took chest x-rays of 150 black and 150 white miners in Zambia. The Europeans were 4% taller, while the chest circumference was 9% bigger, the maximum lung field width was 10% bigger and the lung height 8% bigger in Europeans.

Similarly, Ali (1990) revealed significantly higher values of lung function tests for Nigerians compared to Indians resident in Zaria, Nigeria, which was attributed to the increased height of Nigerians, as the ventilator functions studies are largely dependent on the body size. Within similar ethnic groups, environmental factors may be important in determining ventilatory functions, Ali (1983), because the vital capacities of the Negroes in the United States was similar to those of Europeans, where as African populations Abramowitz et al (1965) had much lower values and Indians living in Guyana had lower values than Indians living in Indian miller et al., (1970).

The values of forced expiratory volumes and flows are significantly higher in men than in women and are also significantly higher in younger than in older age groups Ali (1983). Thoracic length showed no significant correlation with PEFR in female subject while chest circumference correlated positively and significantly with PEFR in males and negatively in females Njoku and Anah (1999).

Decline in values in PEFR is faster in blacks after 40years of age when compared with Caucasians Balchum et al., (1962). Also, PEFR values declined significantly throughout the pregnancy in all maternal positions, Hassan et al., (2005). The rate of decline for the supine position was higher than for the standing and sitting positions, Nunn and Gregg (1989). In normal position, variation of PEFR is largely the result of age, sex, race, height weight body surface area and smoking, Gregg and Nunn (1989). Values of PEFR by recent workers Njoku and Anah (1999) in Nigeria are higher than those recorded in earlier studies, Elebute and Femi-Pears (1971). Improvement in the environment generally and in education may be contributory to this trend. Subjects used in recent studies were on the average taller than those used in earlier studies and this may be another contributory factor. In the study by Njoku and Anah (1999), height correlated positively and significantly with PEFR in both male female subjects, but age correlated negatively and insignificantly with PEFR. In the study by Jaja and Ojo (1983) age had a positive correlation with PEFR in male. The difference in correlation is largely due to age difference, as subjects in the study of Njoku and Anah (1999) are older than those of Jaja and Ojo (1983). The values of forced expiratory volumes and flows are significantly higher in younger than in older age groups Ali (1983). Elebute and Femi-Pearse (1971), have suggested that anthropometric measurements such as height and weight cannot explain the difference in males and females. Other factors such as muscular strength, size and shape of thoracic cage and

elasticity of the lungs should be given due consideration. PEFR values also depend on effort and respiratory muscle strength as well as patient motivation, Hassan et al., (2005).

2.3 The ECG

The existence of racial variants in the electrocardiogram and the possibility of their confusion with truly abnormal tracings have been stressed by Goldman (1953). Indeed several authors have recorded variants of the normal electro cardiogram in African Grusin (1954); Somers & Ranklin (1962); Seriki and smith (1966); Goldman (1953), and other non-European races srikantia, palmavati and Gopalan (1964); Dharmadasa & Nadarajah (1968), and Nigerians Araoye (1984).

There are distinct racial differences in wave amplitude with the Nigerians scaling higher voltages than Caucasians of the same age and sex Araoye (1984). In his work, Araoye (1984) indicated P-wave amplitude in the range of 0.06 to 0.25 MV in the limb leads, and 0.04 to 0.14MV in the precordial leads. The highest P-voltage was seen in lead 2. There is no sex variation but the P-voltage increases with age in the inferior leads while it diminishes with age in aVL. Araoye (1984), documented that Q wave in excess of 0.2MV is rare in any lead except aVL 3% and aVR 75-82% in his study. He stressed that there is no significant sex variation in R wave amplitude in the limb leads and V6, but in V1 to V5, males have higher voltages than age-matched females. Araoye (1984), found that 40-75% of adults have no S wave in the limb leads. About 30-45% others have low S amplitude (less than 5mm). However, S waves dominated the pattern without exhibiting sex variation in V1 to V4, but there is a significant diminution in amplitude with age.

J- point (beginning of ventricular repolarisation) is characteristically elevated in Negroes Golman (1953). Levels up to 4mm (04MV) have been reported; and Araoye (1982) reported J-point level of 5mm in V2 in young Nigerians. J-point elevation is commoner in males in males than females. J-point elevation is of such universal occurrence that its absence in the mid-precordial leads of any adult Nigerian should in fact raise suspicion that one is dealing with an abnormal ECG Araoye (1982). S-T segment elevation equal to or greater than 2mm, with an upward slope ending in a tall positive T-wave is common Oyediran et al., (1972). Seriki and Smith (1966) documented S-T segment elevation in excess of 2mm in lead 3 in 34% of fifty Nigerian male students. The common occurrence of S-T segment elevation in the precordial leads has also been reported from other parts of Africa Walker and Walker (1969).The significance of this S-T segment elevation in apparently healthy individuals is obscured. Its high prevalence, especially in young adualt males, needs to be recognized, as it may cause considerable diagnostic confusion Oyediran (1972). The T-wave amplitude presents tremendous age, lead, race and sex variation Araoye (1984). In all leads, it is higher in males than females of the same age group. In V3 and V4, T-wave amplitude in males was more than double that of the age-matched female and young Nigerian have taller T-waves than Caucasians of same age Aroaye (1984).

Normal QRS axis (0 to $+90^0$) occurs in over 95% of normal Nigerians Aroaye (1984); left axis deviation (0 to -90^0) was seen in 2.9% and 0.1% had right axis deviation ($+90^0$ to ± 180), no indeterminate avis (-90^0 to ± 180) was recorded.

Not only constitutional variables (age, sex, and body habitus), but also ethnic factors affect ECG measurements and morphology, therefore separate criteria are needed for different races, sex and age groups, Araoye (1984).

2.4 Lipo proteins

Hyperlipidaemia is a characteristic feature of human pregnancy peters et al., (1951); Oliver and Boyd (1955). It has been reported that the distribution of lipoprotein levels in the gereral population are different from the bell-shaped curve of serum cholesterol, Dionyssiou-Asteriou and Rizos (1996). During pregnancy, the serum levels of cholesterol, triacylglycerol, very low density lipoproteins (VLDL), low density lipoprotein are increased, while the serum high density lipoprotein cholesterol level is decreased Tayeau et al, (1982), Ordovos (1981). The increased concentration in serum triacylglycerols is to provide the mother with enough fatty acids to be burned, sparing carbohydrate and protein for foetal use, Knoop (1973).

In a study of serum lipids and fatty acid composition in pregnant Nigerian women, it was reported that the lipidaemia associated with pregnancy was less pronounced in Nigerian women compared with women from western countries Taylor (1972). This low serum lipid level in Nigerian women from low in come class, was attributed to their inadequate caloric intake.

Sex difference in the serum lipid and lipoprotein levels was reported in earlier studies Miller et al., (1979). The observed higher mean serum HDL-CH levels in females than in males may be secondary to differences in hormonal levels, such as estrogen, which when given exogenously tend to increase HDL-CH Gustatson and Svanborg (1972). Ononogbu (1979) reported a mean serum Total cholesterol (TC) level of

181mg/dl for Nigeria subjects aged 20-29 years resident in Nsukka. Miller et al., (1979) found mean serum Total cholesterol (TC) and HDL-CH values of 201.2mg/dl and 77.40mg/dl and 197.4mg/dl and 61.9mg/dl in white male and female subjects aged 25-29 years respectively. The variation of serum lipid and lipoprotein pattern on racial/ethnic lines has been attributed to differences in dietary pattern Ononogbu (1988). Wide spread interest has grown in the determination of the serum lipid and lipoprotein pattern of various communities because of the relationship that has been found to exist between the serum lipid levels and the incidence of coronary heart disease (CHD), Seymour (1979). Result obtained elsewhere Bang et al., (1971), has shown that in communities with low incidence of CHD, HDL-CH appears to be high in comparison with communities in which CHD is common. Brewer (2004) added that even in those communities with normal levels of total cholesterol, risk for myocardial infarction is high when HDL-CH is low.

2.5 Haematological Variables

Hematological variables differ significantly between Europeans and Blacks, Araba (1973). Because of malnutrition resulting in decreased production of red cells and increased loss of blood due to various infestations in the tropics, the majority of the populations in tropical Africa with some exceptions suffer from some degree of anaemia, which is worse in women and young children, Trowell (1960).

In the savanna of northern Nigeria, as in other parts of tropical Africa, anaemia has been reported in about 50% of pregnant women Fleming (1984). It is most frequent in women of low socioeconomic status, in primigravidae and during the second trimester of pregnancy. The most important causes are plasmodium falciparum malaria, iron and folate deficiency Fleming (1984). These aetiological factors interact with one another in a complex manner, affecting both mothers and infants Fleming (1986). The

most serious pathological consequence in northern Nigeria is an increased prevalence and density of P. falciparum parasitaemia; this is most marked at about 20 weeks of pregnancy and is found especially in primigravidae Brabin (1983). Malaria, complicated by folate deficiency, is the most important cause of severe anaemia where transmission is holoendemic or hyperendemic: it has become much less common in cities, where mosquito avoidance is practiced and women and obstetricians are aware of the benefits of malaria prophylaxis during pregnancy. Iron deficiency anaemia is the most common form of anaemia encountered during pregnancy, Hesham (2002).The prevalence of iron deficiency anaemia is determined by diet, parity and the transmission of hookworm Fleming (1982). Cereals are poor sources of bioavailable iron: sorghum, millet and maize are eaten in the savanna, while imported rice and wheat have become common foods in urban areas. In contrast, diets based on tubers, including yams and sweet potatoes in the forest region, appear to provide sufficient bioavailable iron to meet most physiological demands. The frequency of iron deficiency rises with parity, especially if pregnancies succeed one another at short intervals Isha (1985). Social customs determine the prevalence of hookworm anaemia, which is extremely rare among Yoruba and Muslim women in the savanna, who do not work in the fields and so escape exposure Fleming (1982). But women from Gambia to Ghana, and in most parts of tropical Africa east of the river Niger, perform farmwork, and for them iron deficiency is a serious and common problem Fleming (1982). The commonly eaten foods in the savanna region are poor sources of folates, whereas yams and plantains of the forest region are rich in them. Folates are destroyed by the prolonged cooking and reheating to which African soups are subjected to Fleming (1982).

Rigorous selection made among African subjects by workers in French West Africa, South Africa and Uganda, Lehman (1949) showed that the hemoglobin concentration is the same as in other races. Araba (1973) showed that; the white blood count is lower in Nigerians than in Europeans. Levels that could be regarded as showing leucopenia are normal amongst our healthy subjects. The leucocyte count is higher in the female than in male subjects.

CHAPTER 3 MATERIALS AND METHOD

3.0 Equipments

1. Wright's peak flow meter
2. ECG machine, sphygmomanometer, and stethoscope
3. Bathroom scale, meter rule and measuring rape.
4. Centrifuge machine, Neumann's counting chamber, heparinized capillary tubes, lancets, cotton wool, Leishman's solution, Acetone (decolorizing agent), Glacial acetate acid solution and antiseptic solution.

3.1 Study Procedure

3.1.1 ***Setting*****:** This is a cross sectional study to determine some aspects of cardiopulmonary and some haematological parameters of apparently healthy non - pregnant and pregnant women at second and third trimesters of pregnancy in Kura LGA of Kano State.

3.1.2 ***Study Site*****:** The study was conducted in Kura town, in the antenatal wing of their comprehensive hospital. Kura town is one of the 44 LGAs in Kano State. Based on the 1991 census, Kano State has a population of 8 million people and 297, 480 of these live in Kura LGA. Most of the men in Kura town are subsistent farmers. Kura LGA has a comprehensive hospital that handles all the referrals from the primary health centers within the LGA.

3.1.3 *Study Population*

The study population are the women of child bearing age, from 15-49years, comprising of both primipara and multipara and a control group of non-pregnant women from other clinics in the same hospital that match age and height.

Sample Size.

A total of 250 both pregnant and non-pregnant women were recruited for the study. This was determined by the use of the formula; $N = \frac{Z^2 p q}{d^2}$ Where

N – Sample size required

Z – Standard normal deviate = 1.96

P – Prevalence or proportion of the parameter in the population for the study

q– Complimentary probability (1-p)

d– Precision with which you carry out the test i. e same as level of significance = 0.05.

No of attendance to ANC in a year =8100 (Kura Ante natal Clinic record 2004)

Women of child bearing age =92,600

$P = \frac{8100}{92600} = 0.087$

$Q = 1 - 0.087 = 0.913$

$N = \frac{(1.96)^2 \times 0.087 \times 0.913}{0.0025}$

$= 122.006$

$= 122$

3.1.5 *Consent for the Study*

Ethical approval from Ahmadu Bello University, Zaria (ESC/2005/00227) was sought for prior to the beginning of this project work. Consent form was also read for the cohorts and their approval obtained prior to the commencement of the procedure everyday until the work was completed.

3.1.6 *Inclusion Criteria*

a. Willingness and ability to participate fully in the study.
b. Certified medically fit by a physician
c. No history of hypertensive disorders i.e. BP > 140/90 $_{MMHG}$
d. No history or sign of pre eclampsia
e. No history of pulmonary disease e.g. asthma or pneumonia
f. No history of recent ingestion of drugs that may affect their cardio respiratory systems.
g. Not alcohol or cigarette users
h. Absence of any chest or physical deformities and other signs of systemic disease on physical examination.

3.2 Methodology

A random sampling method was used to select the cohorts on first contact with research team and/or the supporting staffs in the hospital. After selecting the study population everyday, a questionnaire was completed for each subject.

The data collected include.

3.2.1 *The basic anthropometric data*

Weight (Kilogram) On bare feet with light closing, using a bathroom scale (Beexon, Hungry); height (centimeter) on bare feet using standard measuring meter rule or a calibrated wall; chest circumference (centimeter) in expiration at the level of the 4^{th} intercostals space; and thoracic length (centimeter) measured at the back from the root of the neck to the last rib with an inelastic tape measure (gold fish brand superior tailoring rule chine; body mass index, calculated by using the measured height and weight from this study.

3.2.2 *Blood pressure*

This was taken using the manual Accoson mercury sphygmomanometer. The cuff was tied around the mid arm region of the left arm and its bulb inflated until the radial pulse was no longer felt. Thereafter the pressure was slowly deflated. The first sound heard was taken as the systolic blood pressure while the level of disappearance of the last sound was considered as the diastolic blood pressure.

3.2.3 *Peak expiratory flow rate (PEFR)*

This was determined by the portable Wright's peak flow meter (clement Clarke international Harlow England) whose calibration was checked at regular interval. It

has a central graduation and a pointer. The pointer moves in proportion with the volume and pressure of the expired gas from the mouth. The apex of the flow meter is connected to a disposable mouthpiece. Several mouthpieces were used. Each sterilized at the end of use by one subject, using a cotton wool and chlorohexidine solution. Explanation of the purpose of study and a demonstration of the method of performing the test were given prior to the commencement of the test. Subjects were then made to stand erect, all tight buttons, belt or girdles (as may apply) were loosened. Each subject was made to hold the flow meter lightly with fingers of both hands, emphasis placed on not occlude the movement of the pointer. They, then blow the expired gas from the month with full pressure into the month pieces. Three readings were taken at one-minute interval. The highest of the readings was taken as the representative value.

3.2.4 *ECG*

This was done with the Russian made ECG 3 KIT – 03M2 machine. The instrument is a single electric or battery-powered two-speed (25 and 50mm/second) electrocardiograph.

The electrocardiograph consists of five (5) electrodes, three of which constitute the standard limb leads (I, II and III) and the Goldberger limb leads (AVR, AVL and AVF). The fourth electrode is a neutral or indifferent electrode and the fifth is the Wilson's primordial electrode. All the electrodes were supplied with a 4.8 by 1-8cm Aluminum coated so as to facilitate which electrode is for which extremity.

Table 3.1: 12-lead ECG Electrodes and leads

Colour Code	Letter Code	Connect to
Red	"RA"	Right Arm
Yellow	"LA"	Left Arm
Green	"LL"	Left Leg
Black	"RL"	Right Leg
White	"C"	Chest

Adapted from Schiller ECG 3KIT – 03M2 Machine, Russia.

Each electrode is connected through an outlet wire cable to a multiple lead switch so that the standard limb lead and the precordial leads may be taken successively by moving the dial of the switch without disturbing the connections to the patient. The recording pen is of the direct-writing types with adjustable stylus heat and three levels of sensitivity calibrated at 5, 10 and 20 millimeters per millivolt (HMV).

The paper chart is a heat-sensitive recording roll with a width of about 50mm, and a direct current micro motor with electronic speed control to drive the chart under the stylus.

Effective contact between electrode and skin is essential. ECG gel was used. Electrode jelly contains electrolytes and an abrasive; the abrasive is intended to breakdown the waterproof horny layer of the skin so that the electrolytes of jelly and body may form a continuous conductor.

The jelly was rubbed briskly, not directly smeared, on the skin before the electrolyte is applied. After the test, the leads were removed and ECG jell was wiped off.

3.2.5 *Blood Sample*

Venous blood sample, (5mls) was taken on each subject and control according to standard methods Bachorik (1982) after an overnight fast and the serum separated by centrifugation at room temperature for 1 – 2 hours. Serum total cholesterol level was determine by the method of Zlatkis, et al., (1953), while serum HDL – CH concentration was determined after precipitating out VLDL and LDL according to the procedure of Lopes – Vitrella, et al., (1977).The blood analysis took place in the laboratories of Murtala Mohammad Specialist Hospital, and department of Biochemistry, Bayero University Kano, both of which are about 40kilometers from the study area respectively.

3.3 Limitations

1. Poor effort of blowing the expired air through the peak flow meter by some of the respondents.
2. Inability to obtain the exact ages of some of the respondents.

3.4 Data Analysis (Statistical Methods)

The data was analyzed using simple percentages, range, mean and standard deviation as appropriate using EPI – info 6.0 statistical software package (CDC Atlanta, Georgia, USA). Microsoft Word and Excel were used for the tabulations. The student t-test was used to compare means and the chi-square test for testing the significance of association between the categorical variables. The level of significance is set at $P<0.05$.

CHAPTER 4 RESULTS

Table 4.1: Mean and standard error of mean of the measured variables in the subject and control groups.

Variable (Unit)	Subjects Mean (SEM)	Controls Mean (SEM)
Age (Yrs)	22.7 ± 0.54	25.7 ± 0.64
Height (cm)	156.9 ± 0.005	157.5 ± 0.005
Weight (kg)	52.8 ± 0.60	51.5 ± 0.89
Chest circumference (cm)	81.6 ± 0.42	80.1 ± 0.60
Thoracic Length (cm)	38.0 ± 0.18	38.7 ± 0.24
BMI	21.5 ± 0.23	20.73 ± 0.31
PR interval (ms)	0.2 ± 0.002	0.2 ± 0.002
QRS duration (ms)	0.6 ± 0.002	0.7 ± 0.002
QTC (ms)	0.4 ± 0.0034	0.4 ± 0.004
Electrical Axis	34.6 ± 1.9	32.1 ± 3.7
PCV (%)	32.7 ± 0.52	35.7 ± 0.46
WBC (cm^3)	6.3 ± 0.15	6.4 ± 0.15
TC (mg/dl)	163.8 ± 3.5	161.9 ± 2.5
HDL (mg/dl)	34.9 ± 0.98	32.2 ± 0.49
LDL (mg/dl)	127.2 ± 2.8	130.1 ± 2.0
PEFR (L/min)	375.2 ± 3.1	371.2 ± 4.3

The mean value of PEFR among those studied was 375.2 ± 3.1 L/min in subjects and 371.2 ± 4.3 L/min in the control group.

Table 4.2: Grouping of subjects according to the number of childbirth.

	Number	Percent
Primiparous	40	32.5
Multiparous	83	67.5
Total	123	100

Multiparous women attend the Antenatal clinic more than the primips.

Table 4.3**:** Grouping of subjects according to the stage of pregnancy

	Number	Percent
Second trimester	43	35.0
Third trimester	80	65.0
Total	123	100

This shows that women commonly start attending the ANC from the second trimester of pregnancy and attendance increases as the stage of the pregnancy advances.

Table 4.4: Grouping of both subject and control according to their level of Education

	Subjects		Controls	
	Number	**Percent**	**Number**	**Percent**
Non formal Education	70	57.0	92	72.4
Primary	38	31.0	15	11.8
Secondary	15	12.0	16	12.6
Tertiary	_	_	4	3.2
Total	123	100	127	100

More than fifty percent of both groups do not have the formal education. Those with primary education constitute 30.89 percent in the subjects as opposed to 11.81 percent in the control group. Twelve percent of both groups have the secondary school education. Those attending tertiary institution constitute less than 4 percent.

Table 4.5: Grouping of subjects and controls according to their occupation**.**

	Subjects		**Controls**	
	Number	**Percent**	**Number**	**Percent**
Full time House wife	73	59.4	52	41.0
Trader	49	39.8	71	56.0
Civil Servant	1	0.8	4	3.0
Total	123	100	127	100

Full time housewives constitute 59.4 percent of the subjects and 41 percent of the control group. Those doing petty trading like sewing, ground nut, selling knitting etc constitute 39.8 percent and 56 percent in both the subjects and control groups respectively. The civil servants among the two groups combined equal to 3.8 percent.

Table 4.6**:** Age grouping with observed mean values of PEFR among each group

Age Range (Years)	Subjects		Controls	
	Number (%)	Mean PEFR (SEM)	Number (%)	Mean PEFR (SEM)
15-19	38(30.9)	369.0 ± 7.00	21(16.5)	354.8 ± 12.8
20-24	48(39.0)	379.6 ± 4.43	38(29.9)	380.3 ± 7.60
25-29	17(13.8)	377.1 ± 6.57	20(15.8)	375.5 ± 8.48
30-34	14(11.4)	373.6 ± 8.93	29(22.8)	370.9 ± 9.28
35-39	3(2.4)	363.3 ± 14.5	7(5.5)	358.6 ± 4.04
40-44	1(0.8)	390.0 (x)	10(7.9)	355.0 ± 18.2
45-49	1(0.8)	350.0 (x)	2(1.6)	380.0 ± 20.0
50-54	1(0.8)	350.0 (x)	0	0
Total	123 ( )	375.2 ± 3.1	127 ( )	371.2 ± 4.3

(x) Single subject represented the group indicated

Those in their twenties constitute much of the study population. The mean values of PEFR shows an increase with increasing age, reaching a peak in the 20 – 24 years age range in both groups. The values began to decrease with increasing age thereafter. This finding supports the previous works by Ali (1983) and Njoku and Anah (1999). The higher values observed in the age range of 40 – 54years in the subjects and 45 -49 years in the controls are not truly the mean values of the age groups, because they are mere absolute values.

Table 4.7: Pearson's correlation coefficient between PEFR and measured variables in both subjects and controls

	Subjects		Controls	
Measured Variable	**R**	**P**	**R**	**P**
Age	0.031	0.736	0.09	0.316
Height	-0.1	0.271(NS)	0.078	0.361(NS)
Weight	-0.072	0.43(NS)	0.056	0.534(NS)
Chest Circumference	0.175	= 0.053	0.097	0.278(NS)
Thoracic Length	0.09	0.324(NS)	0.238	< 0.05
BMI	-0.02	0.826	0.019	0.836
PR interval	-0.096	0.288	-0.067	0.454
QRS duration	-0.084	0.355	-0.185	< 0.05
QTC	0.113	0.212	0.034	0.708
Electrical Axis	-0.083	0.369	-0.021	0.813
PCV	0.178	<0.05	-0.021	0.819
WBC	0.013	0.89	0.074	0.408
TC	-0.088	0.331	-0.025	0.78
HDL	-0.065	0.46	0.051	0.57
LDL	-0.063	0.491	-0.042	0.64

There is positive correlation between PEFR with age, thoracic length and chest circumference in both groups.

Table 4.8: Linear regression coefficient for predicting PEFR from measured variables in the subject group

Predictors Used	Coefficient for age (yrs)	Height	Weight	BMI	Chest circum-ference	Thoracic Length	Constant	Multiple correlation coefficient	Standard Error of Estimate
Age, Weight	0.36		-0.47				391.67	0.01	25.58
Age, Height	0.32	-67.8					474.33	0.01	88.17
Age, BMI	0.24			-0.38			377.96	0	28.16
Age, chest circumference	0.004				1.29		269.94	0.03	54.19
Age, T.L. BMI	0.21			-0.64		1.65	321.22	0.01	62.02
Weight, chest circumference			-1.37		2.43		249.34	0.08	53.55
Weight, T. L.			-0.62			2.3	320.56	0.02	59.52
Weight, C.C. T.L.			-1.48		2.3	1.44	210.65	0.08	69.65
Age, T.L.	0.17					1.5	314.21	0.01	60.13

Table 4.9: Linear regression coefficient for predicting PEFR from measured variables in the control group

Predictors Used	Coefficient for age (yrs)	Height	Weight	BMI	Chest circum-ference	Thoracic length	Constant	Multiple correlation coefficient	Standard Error of Estimate
Age, Weight	0.5		0.15				350.37	0.01	24.1
Age, Height	0.59	64.85					253.98	0.01	113.8
Age, BMI	0.6			-0.19			359.67	0.01	26.36
Age, chest circumference	0.41				0.56		316.12	0.01	50.78
Age, T.L. BMI	0.21			-2.13		5.41	200.77	0.07	59.33
Weight, chest circumference			-0.014		0.7		315.94	0.01	51.87
Weight, T. L.			-0.69			5.71	186.13	0.07	61.32
Weight, C.C. T.L.			-0.78		0.28	5.58	173.68	0.07	70.89
Age, T.L.	0.002					4.15	210.57	0.06	59.26

From the table 4.8, the formula incorporating age and weight i.e. 0.36AGE – 0.47WT + 391.67 had the lowest standard error of estimate (SEE); (25.58) and, therefore, will be the best prediction formula for PEFR in pregnant women (subjects) in Kura local government area of Kano State. The formula incorporating age and Body mass index (BMI) i.e. 0.24AGE – 0.38BMI + 377.96 follows very closely with SEE value of 28.16. Similarly, from Table 4.9, the formula incorporating age and weight i.e. 0.50AGE + 0.15WT +350.37 have the lowest SEE of 24.10 and, therefore, is the best prediction formula for PEFR in non-pregnant (control) females. This is followed closely by the one incorporating age and Body mass index in which SEE is 26.36 i.e. 0.60AGE – 0.19BMI + 359.67.

Figures 4.1and 4.2, are graphical displays of the observed variation of PEF with age in subject and control groups respectively, along with graph showing predicted variation of PEFR with age using a formula derived from this study and a different formula derived by Njoku and Anah, (1999). Using the keys to the graphs, it would be noted in figure 4.1 and 4.2 that the best predicted linear equations for the two groups produced graphs that ran closely with observed values. The values in this study are significantly lower ($p<0.05$) than the predicted values derived from Njoku's formula.

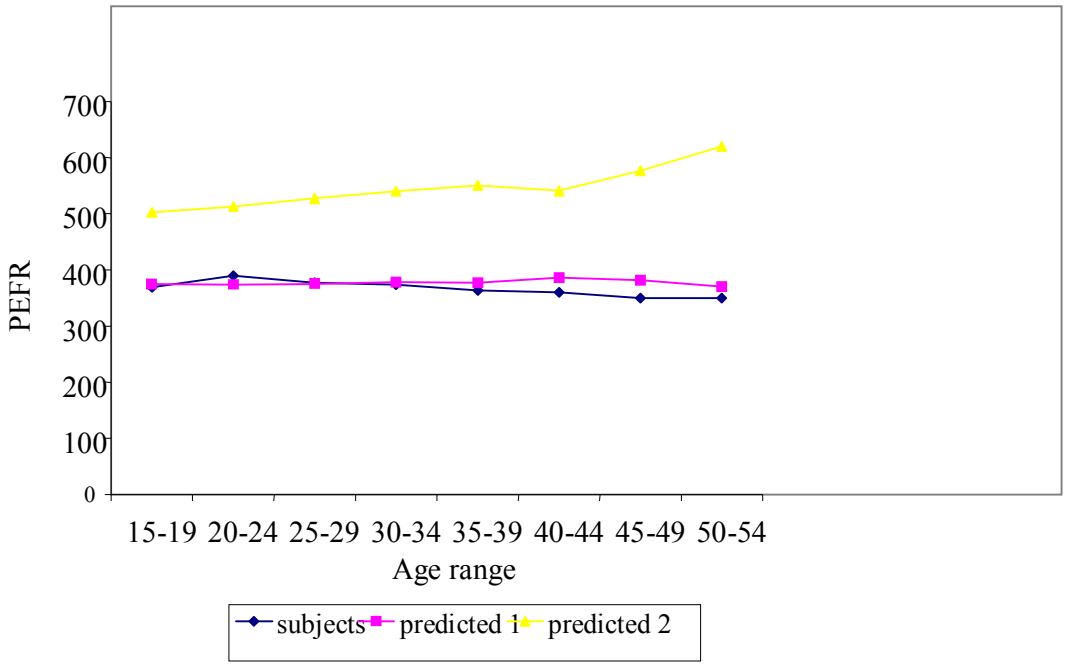


Figure 4.1: PEFR values among the subjects

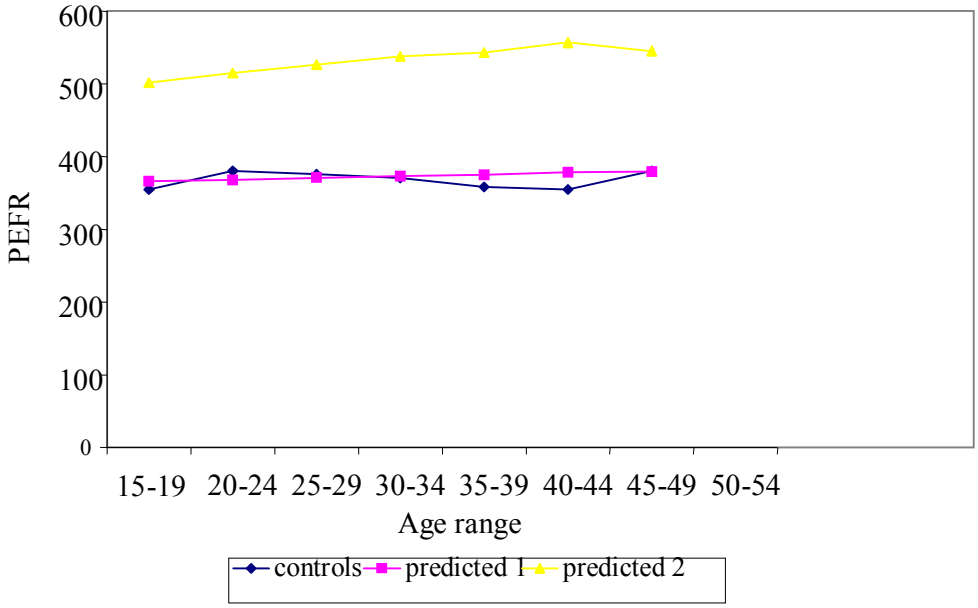


Figure 4.2: PEFR values among the controls

Two sample T – test was used to compare the mean values of the two groups. There is no significant statistical difference between the mean PEFR of the two groups ($P > 0.05$). Also, the result indicates no much difference in the mean height, and weight between the two groups, ($P > 0.05$). Significant statistical difference $P < 0.05$ in the means of chest circumference, thoracic length and Body mass index has been observed between the subjects and control group, with values of chest circumference and BMI higher and value of thoracic length lower in the subject than in the control group. The QRS duration of the two groups shows a lower value ($p<0.05$) in the subjects than in the control group.

There is significant difference ($P< 0.05$) in the PCV values of the two groups. It is very much higher 35.7% in the control than 32.7% in the subject group. The WBC counts of the two groups do not differ significantly. Among the Lipoproteins; TC, LDL show no appreciable difference ($P>0.05$), but the HDL is higher ($P<0.05$) in the subject compared to the control group.

Chi – square test was used to compare some of the categorical variables. The subject group has higher number ($P<0.05$) of those with formal education than the control group. The Fisher exacts between formal and non-formal employment among the two groups is however not significant ($P>0.05$).

Within the subject group, Student t- test was used to compare the means of some variables between the second and third trimesters of the pregnancy. There is no statistical difference ($P>0.05$) in the PEFR, PCV and WBC values between the two trimesters of the pregnancy. However, there is significant difference ($P<0.05$) in the

values of lipoproteins, with Total Cholesterol, HDL and LDL higher in the second trimester than in the third trimester.

CHAPTER 5 DISCUSSION

The study involved Hausa- Fulani females at their reproductive ages, who attended Antenatal and other clinics at Kura General hospital in Kura local government area, Kano state, from August 2005 to December 2005.The cohorts were free from chest illness and with relatively little exposure to any form of air pollution. They enjoyed environmental and nutritional background similar to a typical rural community in the northern part of Nigeria.

More than 60 percent of the females that participated in this project work were less than 30 years of age as of the time of conducting this research.

This study has a smaller sample size when compared with other similar studies in Nigeria Njoku and Anah (1999) and elsewhere Gregg and Nunn (1989). It is limited to females of child bearing age; hence it will have limited application. The age range of the cohorts is similar to those of Ali (1983).

It is observed that considerable variations exist between accepted normal values of ventilatory functions of Caucasian as compared to Nigerians, Cote et al., (1966), Femi-pearse, (1971). Similar cross-cultural variations in Nigerians have been documented by Ali (1983); Njoku and Anah (1999). The result from this study show that the values of peak expiratory flow rate, determined with the Wright's peak flow meter is lower in females residing in the Northern part than those residing in the southern part of Nigeria, and their European counterparts. This agrees with the findings of (Abid Ali, 1983), who reported lower values not only on PEF but also on other Spirometric measurements, like FVC, FEV1 etc.

Similarly, values of PEFR by recent workers Njoku and Anah (1999) in Nigeria are higher than those recorded in this study. Differences in the environment generally and in education may be a contributory factor to this trend as more than 50 percent of both groups in this project work do not have the formal education. Also, subjects used in recent studies were on the average taller than those used in this study and this may be another contributory factor. From this study, there is no statistical difference in the mean PEFR values between the subjects and controls. In addition, the values between the second and third trimesters of the pregnancy are not different. This shows that PEFR is not affected by pregnancy. The values presented here, while lower than those from Caucasian studies, support the work of Brancazio et Al., (1997) who showed that PEFR is not affected by pregnancy and lactation. In this study, age correlated positively in both subjects and controls. It usually forms part of the prediction formula in most studies Njoku and Anah (1999); Ali (1983) whose results have wide application. The negative and insignificant correlation of height and weight with PEFR in the study group is in opposition with findings of other studies Njoku and Anah (1999). This is largely because of genetic reason. The prediction formula for the women in this part of the country incorporated age and weight, while that in the southern part incorporated age and height. It seems likely that below certain height limit, the weight is much more significant in correlation with PEFR than the height. Therefore, BMI may be more important than the use of a single height or weight in using prediction formulae. The chest circumference and thoracic length correlated positively with PEFR in both the subjects and controls, which is in support of the normal assertion, since the lungs are housed in the thorax.

The duration of intervals and segments of the ECG wave forms of the cohorts in this study is similar to those documented on females Caucasians Hiss et al., (1960) and those residing in the southern part of Nigeria Araoye, (1984), age and pregnancy state being negligible. Araoye documented PR interval of 0.14 – 0.20 second; QRS duration of 0.06 – 0.11 second; and QTc duration of 0.371 – 0.444 in female subjects. In this study the PR interval was found to be 0.12 – 0.20 second; QRS duration of 0.04 – 0.12 second and QTc duration of 0.207 – 0.499 second. This shows that the durations and intervals of the wave forms of ECG are universally similar. The QRS axes of two (2.5%) of the pregnant subjects at their third trimester range from -16 to + 90^0. This has support from the findings of previous workers Bonica (1973) that there is a 15^0 left axis deviation of the electrical axis at the advanced stage of the pregnancy. The reason is due to the fact that the enlarging uterus causes a progressive upward displacement of the diaphragm which in turn causes left ward deviation of the electrical axis of the heart. The remaining 78 (97.5%) of the subjects at their third trimester stage have their QRS axes lying between 0 to + 90^0. No left axis deviation was found for the females at second trimester of pregnancy and the control group. Their QRS axes lie between 0 to + 90^0.

In this study there is statistical difference ($p<0.05$) in the PCV values between the subjects and controls. The result indicates that pregnancy is associated with low PCV value, and the value tends to differ with different gestational period. The PCV was found to be low in the second trimester of the pregnancy. This finding is in agreement with Fleming et. al., (1987) who documented that anaemia is present in about 50% of pregnant women in the Savanna of northern Nigeria as in other tropical parts of Africa, and it is most frequent in women of low socioeconomic status, in

primigravidae and during the second trimester of pregnancy. The reason for low PCV in this stage could be due to haemodilution, malarial infestation, iron and folate deficiency. These aetiological factors may interact with one another in a complex manner, affecting both mothers and infants. Fleming et al., (1987) stated that; Malaria, complicated by folate deficiency, is the most important cause of severe anaemia where transmission is hyper endemic. Kura local government area is a typical rural area where mosquito avoidance is difficult because of bushes, sludge and lack of mosquito netting; also the women are less aware of the benefits of malaria prophylaxis during pregnancy. Iron deficiency anaemia could be another reason for anaemia in this group of the subjects due to the nature of their diet and their parity stages. They depend mainly on cereals like sorghum, millet, rice and maize which are poor sources of bioavailable iron. 67.5% of the subjects are Multiparous having two to ten number of children, most of whom the pregnancies succeeded one another at short intervals. Folate deficiency is possibly another factor of causing anaemia in the second trimester of the pregnancy because of recurrent malarial attacks and the cooking habits of women in northern Nigeria. Folates are destroyed by prolonged cooking and reheating to which our soups are subjected to.

During pregnancy, physiological depression of cell mediated immunity occurs, probably to allow tolerance of the foetus Fleming, et al. (1987). From this study there is no significant statistical difference ($p>0.05$) in the WBC counts between the subjects and controls and also between the second and third trimesters of the pregnancy.

There is inverse relationship between the serum lipoproteins and pregnancy from this study. The increase is highest in the second trimester and may be of maternal advantage to burn the excess fat depots for energy and use the carbohydrates and proteins for the growing fetus, Knoop et. al., (1973).

Total cholesterol and lipoproteins are found to be higher in the pregnant than in the non-pregnant group. The observed higher mean values of these lipoproteins and cholesterol in pregnancy may be secondary to increase in the hormonal levels associated with pregnancy, such as oestrogen which when given exogenously tend to increase HDL Gustafson and Svanborg (1972). While no strong claim can be made about the incidence of coronary heart disease (CHD) in the Hausa Community in general and the subjects studied in particular, it is surprising to find that the values of total cholesterol and HDL in second and third trimesters obtained from this study are lower than those of Total cholesterol and HDL obtained by Atiku et, al., (1990), on non- pregnant female medical students of Bayero University Kano and in communities Bang et al., (1971) in which CHD is common. It is possible that the life style and dietary pattern of the subjects may be responsible for the low HDL values obtained in this study. But, whether low HDL values appear to be a feature in this community, in spite of the relatively low incidence of CHD remains to be answered.

CHAPTER 6: SUMMARY AND CONCLUSION

6.1 SUMMARY

Limited knowledge is available on normal values of cardiopulmonary parameters of pregnant women in Northern Nigeria and Kano State in particular. The aim of this project is to determine the normal values of peak expiratory flow rate (PEFR) in healthy pregnant subjects and controls in Kura local government area of Kano State, with view to generate a prediction formula for PEFR inpregnant women in this environment. It is also aimed at assessing the effect of pregnancy on some of the ECG and haematological variables among the cohorts.

Values of peak expiratory flow rate (PEFR), ECG and Haematological variables in 250 females at their reproductive ages, made up of 123 pregnant subjects and 127 non-pregnant control group, living and/or working in Kura local government area and its environs were obtained.

Pearson's correlation coefficient between PEFR and ages as well as ECG, Haematological and other anthropometric measurements were calculated in both groups.

Multiple regression analyses were made to obtain the best prediction formulae using the measured variables in pregnant and non-pregnant groups. Furthermore, graphs were plotted to show the variation of PEFR with age in the subjects and controls with specific weights. Data for plotting the graphs were generated using the prediction formulae generated from this study and other prediction formulae generated from previous studies among Nigerians, whose best prediction formulae incorporated age and height. The graphs illustrated the limitations of applying formulae derived from a different race, tribe and/or generation to an entire different people with dissimilar characteristics. This lends credence to the need to make use of recently generated formulae for local predictions.

CHAPTER 6: CONCLUSION AND RECOMMENDATIONS

6.2 CONCLUSION

1. PEFR, WBC and ECG are not affected much by pregnancy.
2. Pregnancy is associated with low PCV value, especially the second trimester.
3. There is left axis deviation of the heart in some of the pregnant women during the third trimester period.
4. There is inverse relationship between serum lipids and stages of pregnancy.
5. Variations exist in the normal values of PEFR between women in Kura local government area and those in the southern part of Nigeria, hence the difference in the prediction formulae.

6.2 RECOMMENDATIONS

1. Researches should be narrowed down to communities, to allow Scientists to know more about a particular group of people living in an environment.
2. Caution should be exercised on generalizing a formula on an entire population of a nation or state, as they compose of a mixture of heterogeneous societies.
3. As variability exists among different communities, Physicians should relay more on data obtained on black Africans than Europeans, so as to come closer to the exact values on individual subject.
4. Further investigations need to be carried out to ascertain the estimated values of PEFR on both male and female subjects in this environment.

REFERENCES

Abid Ali, (1983): Ventilatory functions in Non- smoking Healthy Nigerian Adults. West Afr. J. of Med. **2**: 1- 8.

Abramowitz, S. Leiner, G. C., Lewis, W. A. and Small, M. J. (1965): Vital capacity in the Negro, Amer Rev Resp Dis. **92**: 287-292.

American Heart Association Guidelines (2005) for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care - Part 8: Stabilization of the Patient With Acute Coronary Syndromes." *Circulation* 2005; **112**: IV-89 - IV-110.

Angus, G. F. and Thurlbeck, W. M. (1972): Number of alveoli in the human lung. J. Appl. Physiol., **32**; 483.

Araba A. B. (1973): A survey of Hematological Variables in 600 Healthy Nigerians. Nig. Med. Journal 6 (1); 49-53.

Araoye, M. A. (1984): The 12- Lead Electrocardiogram (ECG) in Negroes: - 1- Normal values.

Atiku M.K, Gwarzo, M.Y., and Wudil, A. M (1990): Serum lipids, total cholesterol and High density lipoprotein choresterol profile in healthy Hausa subjects. Afr. J Nat & Mat Sci; **4**(2): 52-56.

Aubier M, Murciano D, Milic-Emili J, et al. (1980); Effects of the administration of O_2 on ventilation and blood gases in patients with chronic obstructive pulmonary disease during acute respiratory failure. Am Rev Respir Dis. 122: 747–754.

Avery, M. E. and Mead, J. (1959): Surface properties in relation to atelectesis and hyaline membrane disease. Archs Dis. Cbildb., **97**, 517.

Bachorik, P.S (1982): Clin. Chem. 28 (6): 1375 – 1378.

Bang, H.O., Dyeberg, J and Neilson, A.B (1971): Plasma lipid and lipoprotein pattern in Greenlandic West Coast Eskimos. Lancet, 1: 1143- 1145.

Bates D V. (1989). Respiratory function in disease. 3rd ed. Philadelphia: W. B. Saunders.

Becklake M R. (1986): Concept of normality applied to the measurement of lung function. Am. J Med; **80**: 1158- 64.

Biscoe TJ, Bradley GW, Purves MJ. (1970); The relation between carotid body chemoreceptor discharge, carotid sinus pressure and carotid body venous flow. J Physiol (London) **208:** 99–120.

Bisgard GE, Neubauer JA. (1995); Peripheral and central effects of hypoxia. In: Dempsey JA, Pack AI,eds. Regulation of Breathing. 2nd Edn. New York, Marcel Dekker, pp. 617–668.

Bledsoe SW, Hornbein TF. (1981): Central chemoreceptors and the regulation of their chemical environment. In: Hornbein TF, ed. Regulation of Breathing. New York, Marcel Dekker, 347–406.

Bonica JJ. (1973): Maternal respiratory changes during pregnancy and parturition. Marx GF, ed. Parturition and perinatology. Philadelphia; FA Davis; 2- 19.

Brancazio L. R., Laifer S. A., Schwartzt (1997): Peak Expiratory Flow Rate in normal pregnancy. Obstet & Gynecol. 89; 383 – 386.

Braunwald E. (Editor), (1997); Heart Disease: A text book of Cardiovascular Medicine, Fifth Edition, P. 108, Philadelphia, W.B. Saunders Co.

Brewer Jr. HB (2004); New features of the National Cholesterol Education Program Adult treatment panel III lipid-lowering guide lines. Clin. Cardiol. **26:** 19-24.

Brown, E. S., Johnson, R. P. and Clements, J. A. (1959): Pulmonary surface tension. J. Appl. Physiol., **14**, 717.

Bruce EN, Cherniack NS. (1987); Central chemoreceptors. J Appl Physiol 62: 389–402.

Clinical value of the ECG in noncardiac conditions."*Chest* 2004; **125(4)**: 1561-76.

Cotes, J.E., Rossiter, E.C., Higgines, I.T.T. and Gilson, J.C. (1966) Average normal values for the Forced Expiratory Volume in White Caucasian males. Brit. Med. J. 1 : 1016 – 1019.

Danny (1996): Cardiac Disease. Jn: Report on confidential Inquiries into Maternal Deaths in the United Kingdom 1991-1993. London; HMSO: 109-120.

Dharmadasa, K. & Nadarajah, M. (1968): Electrocardiograms in young Ceylonese. Brit. Heart J. **30**; 765- 767.

Dionyssiou – Asteriou A, Rizos I (1996); Serum Lipoprotein – (a) levels in a Greek population sample without a history of premature myocardial infarction. JAMA **276:** 544-548.

Easton PA, Slykerman LJ, Anthonisen NR. (1986); Ventilatory response to sustained hypoxia in normal adults. J Appl Physiol 61: 906–911.

Elebute EA, Femi- Pearse D (1971): Peak flow rate in Nigerian. Anthropometric determinants and usefulness in assessment of ventilatory function. Thorax; **26**: 597-601.

Euler C von. (1986); Brain stem mechanisms for generation and control of breathing pattern. In: Cherniack NS, Widdicombe JG, eds. Handbook of Physiology; Control of Breathing. Vol. 2, Part 2, Section 3. Bethesda, American Physiological Society, pp. 1–67.

Femi- Pearse D, Elebute E : (1971): Ventilatory function in healthy adult Nig. J Clin. Sci.; 41 : 203.

Fleming A. F. (1987) : Maternal anaemia in northern Nigeria: causes and solutions. World Health Forum Vol. 8

Ganong W. F. (2005): Review of medical physiology. Mc Graw – Hill Companies, Inc. 22nd Edition.

Glazier, J B. et al. (1967): Vertical gradient of alveolar size in lungs of dogs frozen intact. J. App. Physiol., **23**; 694.

Gregg I Nunn A.J. (1989) New regression equation for predicting peak expiratory flow in adults. Brit. Med. J.,298: 1068 – 1074.

Gregg I, Nunn AJ. (1989); Peak expiratory flow in symptomless elderly smokers and ex-smokers. Brit. Med. J., **298:** 1071-2.

Goldman M.J. (1953): RS-T segment elevation in mid-and left precordial leads as a normal variant. Amer. Heart J. **46**; 817-820.

Goldman M J. (1960): Normal variants in electrocardiogram leading to cardiac Invalidism. Am. Heart J; **59**(1): 71-77.

Goldman, M. J. (1976): Principles of clinical electrocardiography. Lange medical publications. 9th Ed.

Grusin, H. (1954): Peculiarities of the African's electrocardiogram and the changes observed in serial studies. Circulation, **9**; 860-867.

Guck. Et al, (1971): Diagnosis of the respiratory distress syndrome by amniocentesis. Am J. Obstet. Gyne. **109**; 440

Gustafson, A. and Svanborg A. (1972). Gonadal steroidal effects on plasma lipoproteins and individual phospholipids. J Clin. Endocrinol. Metab. 35: 203- 207

Guyton AC, Hall JE, (2000): Textbook of medical physiology H. I. E. Saunders company 10th ed.

Hassan M., Harirah Sahar E., Fayez Nasrallah K., Geoge Saade R., and Micheal Belfort A. (2005); Effect of Gestational age and position o Peak Expiratory Flow Rate: A Longitudinal Study. 105: 372 – 375.

Hesham Al- Inany (2002); Iron deficiency anaemina in pregnancy: A short account.

Hiss, R.G., and Lamb, L.E., and Allen, M.F., (1960): Electrocardiographic findings in 67,375 asymptomatic subjects. Normal values. Am.J. Cardiol. 6: 200 – 231.

Kafer E. (1975); Idiopathic scoliosis. Mechanical properties of the respiratory system and the ventilator response to carbon dioxide. J Clin Invest. **55**: 1153–1163.

Knoop, R. P., & Saudek, C.D., Arky, R.A., and O' Sullivan, J.B. (1973): Two phases of adipose tissue metabolism in pregnancy, maternal adaptation for growth. Endocrinology **92**; 984.

Lehman, H. (1949): Blood in Ugandans. Nature;**164**; 954.

Littmann, D. (1946): Persistence of the juvenile pattern in precordial leads of healthy adult Negroes. Amer. Heart J. **32**; 370-382.

Lopes – Vitrella, M. F., Stone, P., Ellis, S. and Colwell, J. A. (1977): Cli. Chem. 23: 82 – 884.

Mc Fall / PB, Dona J.C., Boyle D (1988): Pregnancy complicated by maternal heart disease. A review of 519 women Br J Obstet Gynecol; **95**: 861-7

Melville G. N., Kumar M, Ismail S, Seely C, Wray SR. (1981): Pulmonary functions of Afro- Jamaicans. West Ind Med J; **30**: 119- 123.

Miller G, Cotes J, Hall A, Salvosa C, Ashworth A (1972): Lung function and exercise performance of healthy Caribbean man and woman of African ethnic origin. QJ Exp Physiol, **57**: 325-341.

Miller, J.G., Ashcroft, M.T., Swan, A.V. and Beadnell, H.M.G.S. (1970): Ethnic variation in forced expiratory volume and forced vital capacity of African and Indian adults in Guyana. Amer. Rev. Resp. Dis. **103**: 57-67.

Neegaard, K. Von (1929): Neue Auffassungen uber einen Grundbegriff der Atemmechanik.Die Retraktionskraft der lunge, abhangig von der oberflachenspannung in der Alveoen. Z. ges. Exp. Med., **66**, 373.

Njoku, CH. Anah, C O. (1999): A new formular for predicting Peak Expiratory Flow Rate in Adult Nigerians. Sahel Med. J**. 2**: 39-44.

NUNN J. F. (1971): Applied Respiratory Physiology. 2nd Edition Butter Worths London- Boston.

Odunta, S. A. (1970): Spirometric studies of normal and pre- operative patients in Nigeria. Afr. J. Med. Sc. **1**: 79-84.

Olanrewaju P.A. (2001): Illustrated Textbook of Electrocardiography. Baokum Comm. Press, 34, Fajuyi Road Ile –Ife Nigeria.

Oliver, M I, And Boyd, G. S.(1955): Plasma lipid and serum lipoprotein patterns during pregnancy and puerperium; Cli. Sci.**14**; 15.

Onadeko, K. O. Falase, A. O., and Ayeni, O. (1976): Pulmonary function studies in Nigerian sportmen. Afr. J. Med. Sci. **5**: 291-295.

Ordovos, J J., Pocovi, M. and Grande, F. (1984): Plasma lipids and cholesterol esterification rate during pregnancy. Obst, & Gynaecol. **63** (1); 20-25.

Pattle, R. E. et al., (1962): Inability to form a lung lining film as a cause of the respiratory distress syndrome in newborn. Lancet, **2**, 469

Pattle, R. E. (1955): Properties, function and origin of the alveolar lining fluid. Nature Lond.., **175**, 1125.

Peters, J.P., Heinemann, M., and Man, E. B. (1951): Lipids of serum in pregnancy. Journal of Cli. Invest. **30**:388.

Poon CS. (1989); Effects of inspiratory resistive load on respiratory control in hypercapnia and exercise.J Appl Physiol 66: 2391–2399.

Richter DW. (1982); Generation and maintenance of the respiratory rhythm. J Exp Biol 100: 93–107.

Robert & Crapo (1994): Review art. Pulmonary function testing. New Eng. J of Med. 331 No.**1**: 25-29.

Rossiter CE, Weil H. (1974): Ethnic differences in lung function: evidence for proportional difference. Int. J. Epidemiol. **3**:55-61.

Sabatine MS (2000); Pocket Medicine (Pocket Note book). Lippincott Williams & Wilkins.

Schoenberg JE, Beck GJ, and Bouhuys A (1978): Growth and decay of pulmonary function in healthy blacks and whites. Respire. , **33**; 367-393.

Seriki, O. & Smith, A.J. (1966): The electrocardiogram of young Nigerians. Amer. Heart J. **7**; 153-156.

Somers, K. & Rankin, A.M. (1962): The electrocardiogram in healthy East African (Bantu and Nilotic) men. Brit. Heart J. **24**; 542.

Srikantia, S.G., Padmavati, S. & Gopalan, C. (1964): The electrocardiogram in some Indian population groups. Circulation, **29**; 118-123.

Tayeau, F., Maviel, M. J., Dumon, M.F. et al. (1982): Experimental research on the origin of pregnancy hyperlipoproteinemia. Historical study and examination of 320 sera of pregnant women: Bordeaux Med. 15 (20) 965-970.

Taylor, G. O. (1972): Serum lipids and fatty acid composition in pregnant Nigerian Women. Journal of obst. And Gynae. Of British Commonwealth. **79**: 68-71.

Trowell, H.C., (1960): Non – infective disease in Africa 1st ed. Edward Arnold London.

Walker, A.R.P. & Walker, B.F. (1969): The bearing of race, sex, age and nutritional state on the precordial electrocardiograms of young South African Bantu and Caucasian subjects. Amer. Heart J. **77**; 441-459.

Wasserman KB, Whipp BJ, Casaburi R. (1986); Respiratory control during exercise. In: Cherniack NS, Widdicombe JG, eds. Handbook of Physiology; Control of Breathing. Vol 2, Part 2, Section 3. Bethesda, American Physiology Society, pp. 595–619.

Webb, S. J. S. and Nunn, J. F. (1967): A comparison between the effect of nitrous oxide and nitrogen on arterial PO2. Anaesthesia, **22**; 69.

West J.B. (1990): Respiratory Physiology – the essentials 4th ed. Williams & Wilkins.

Wodcock A. J., Colman M. H., Blackburn CKB (1972): Factors affecting normal values for ventilatory functions. Am Rev Repir Dis, **106**: 692-709.

Zlatkis, A., Zak, B. and Boyle, A.J. (1953): J. Lab. Clin. Med. 41: 486 – 492.

PROFORMA I

A. PERSONAL DATA

1. a) Name ----------

b) Age ----------

c) Address ----------

2. Level of Education

a) Non- formal b) Primary c) Secondary d) Polytechnic e) University

3. Occupation

a) Complete House Wife b) Trader c) Civil Servant

4. Income of the husband per annum ----------

B. ANTENATAL HISTORY

a) I. Pregnant II. Non- Pegnant

b) I. Primiparous II. Multiparous

c) Stage of the pregnancy

i) First trimester ii) Second trimester iii) Third trimester

C. SOCAIL HISTORY

a) Do you smoke? i) Yes ii) No

b) Do you drink alcohol? i) Yes ii) No

D MEDICAL HISTORY

a) Have you ever been admitted in to any hospital in the past?

i) Yes ii) No

b) Are you presently having cough, difficulty in breathing or palpitation?

i) Yes ii) No

c) Are you on any medication now? i) Yes ii) No

d) If yes, for what disease/diseases? ----------

e) Was the drug prescribed by a Doctor, paramedic or under self medication? ----------

PROFORMA II

A. Anthropometric Data

i) Name ----------

ii) Age ----------

iii) Height ----------

iv) Weight ----------

v) Body mass index ----------

vi) Thoracic length ----------

vii) Chest circumference ----------

B. Blood pressure

i) SBP (mmHg) ----------

ii) DBP (mmHg) ----------

iii) Pulse rate (beats/min) ----------

C. PEFR Values

i) First attempt ----------

ii) Second attempt ----------

iii) Third attempt ----------