UNIVERSITY FOR DEVELOPMENT STUDIES

IMPACT OF SOME RISK FACTORS OF PREGNANCY INDUCED HYPERTENSION IN THE UPPER EAST REGION

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(UDS/MBM/0002/17)

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UDS/MBM/0002/17

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SEPTEMBER, 2019

DECLARATION

Student

I hereby declare that this thesis is the result of my own original work and that no part of it has been presented for another degree in this University or elsewhere. Related works by others which served as a source of knowledge have been duly referenced.

Candidate's Signature: ……...…… Date: ……..……

Name: ……...……

Supervisors'

I hereby declare that the preparation and presentation of the thesis was supervised in accordance with the guidelines on supervision of thesis laid down by the University for Development Studies.

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ABSTRACT

Pregnancy Induced hypertension (PIH) is a global concern that affect pregnant women usually after 20 weeks of gestation. It accounts for 5 to 10% of all pregnancies in relation to haemorrhage and infections. Clinicians and researchers are finding it difficult to comprehend the primary cause of this anomaly. The Upper East Region of Ghana has witnessed an upsurge in maternal mortality in 2016 and 2017 due to this precarious situation. Hence the need to investigate on some of the perceived risk factors associated with hypertension in pregnancy. Data on the obstetric history of 50 pregnant women from the Bolgatanga Regional Hospital aided the feasibility of this research. The research analysis was done using three binary classifier models namely; Logistics regression model, Probit model and Artificial Neuron Network model from STATA 12 and IBM-SPSS Statistics 20 software. The results of this research will enable us to comprehend, approach and tackle issues of gestational hypertension that are inherent to the Upper East Region.

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LIST OF ABBREVIATIONS

HDP	Hypertensive Disorders in Pregnancy
SSA	Sub – Saharan Africa
PIH	Pregnancy Induced Hypertension
WHO	World Health Organisation
UNPF	United Nations Populations Fund
GHS	Ghana Health Services
UER	Upper East Region
AMA	Advance Mother's Age
MA	Mothers Age
GW	Gestational Weight
GA	Gestational Age
BMI	Body Mass Index
GDM	Gestational Diabetes Melitus
GWG	Gestational Gain Weight
IOM	Institute of Medicine
HDL-C	High- Density Lipoprotein Cholesterol
LDL- C	Low-Density Lipoprotein Cholesterol

NCEP	National Centres for Environmental Predictions
NICE	The National Institute for Care and Excellence
SUA	Single Umbilical Cord Artery
NRFHT	Non-Reassuring Foetal Heart Tracing
IQ	Intelligent Quotient
PE	Preeclampsia
MCV	Mean Corpuscular Volume
MCH	Mean Corpuscular Haemoglobin
MCHC	Mean Corpuscular Haemoglobin Concentration
RDW	Red Cell Distribution Width
RBC	Red Blood Cell
WBC	White Blood Cell
GL	Glucose Level
PL	Placenta Length
PW	Placenta Weight
UCL	Umbilical Cord Length
UCD	Umbilical Cord Diameter

CL	Cholesterol Level
HB	Haemoglobin
ANN	Artificial Neural Network
ROC	Receiver Operation Characteristics
AUC	Area Under Curve
STATA	Software for Statistics and Data Science
IBM	International Business Machines Corporations
SPSS	Statistical Package for Social Sciences
SD	Standard Deviation
CI	Confidence Interval
Std. Err.	Standard Error
Obs.	Observation
Pr-Value	Probability Value

CHAPTER ONE

INTRODUCTION

1.1 Background of the study

While the thought of having a new born child comes with an extreme joy to expectant mothers, the jolly-ride could be marred by some complications. Hypertensive Disorders in Pregnancies (HDP) remain a major clinical challenge in contemporary obstetric practice due to the associated burden of high maternal and perinatal adversities (Duley, 2009). A research conducted by *Lancet Global Health* indicated that about 73% of all maternal deaths between 2003 and 2009 were due to obstetric causes and deaths due to indirect causes accounted for 27.5 %. Hypertensive disorders alone accounted for 14% of the deaths (The Lancet Global Health, 2014).

Although the exact cause of preeclampsia is unknown, there are many established risk factors such as nulliparity, obesity, pre-existing hypertension, a prior history and family history of preeclampsia, existing autoimmune conditions, and extremes of maternal age (Norwitz and Schorge., 2006). The disorder is responsible for 70 000 maternal deaths and 500 000 infant deaths globally per annum (Khan *et al.*, 2005; Sibai *et al*., 2006).

The fifth goal of the United Nations Millennium Development Goals (UN-MDG) for 2015 is to reduce the maternal mortality ratio (MMR) by three-forth (World Health Organization, 2000). This is because about 830 women die on daily basis and approximately 303,000 women die every year from pregnancy

related causes (Alkema *et al.*, 2016). Evidence shows that developing countries lead the chart on maternal mortality rate. There were 239 maternal deaths per 100,000 live births in the low-income region, compared to 12 maternal deaths per 100,000 live births in developed regions in 2015 (Conde-Agudelo *et al.*, 2005). Sub-Saharan Africa (SSA) has been the most affected with more than half of all the deaths (Alkema *et al*., 2016). Hypertensive disorders of pregnancy, haemorrhage, severe anaemia, sepsis, obstructed labour, and unsafe abortions and its complications are the main direct causes of maternal deaths (World Health Organization, 2005).

Pregnancy-induced hypertension (PIH) is a leading cause of maternal and perinatal mortality and can also lead to long-term health problems like chronic hypertension, kidney failure, or nervous system disorders (Singh *et al*., 2005). Gestational hypertension or pregnancy induced hypertension (PIH) is the development of new hypertension in a pregnant woman after 20 weeks gestation without the presence of protein in the urine or other signs of pre-eclampsia (McGraw-Hill Professional, 2014). Hypertension is defined as having a blood pressure greater than 140/90mmHg (McGraw-Hill Professional, 2014).

Pregnancy induced hypertension can be differentiated from chronic hypertension, which is the high blood pressure that was present before a woman becomes pregnant or that occurs in the first half (before 20 weeks) of pregnancy (The American College of Obstetricians and Gynaecologist, 2013). Pre-eclampsia, which is a type of pregnancy induced hypertension characterized by progressive hypertension pathological oedema, is clinically defined as blood

pressure greater than 140/90mmHg after 20 weeks gestation coexisting with proteinuria (300mg/24 h or greater 1+protein on a dipstick sample of urine collected at random) (Denis *et al*., 1988). Hypertensive disorders are global health problems. It includes pregnancy-induced hypertension (without protenuria), pre-eclampsia (with protenuria) and eclampsia (with convulsion), gestational hypertension and chronic hypertension. Though advances in perinatal care, hypertensive disorders of pregnancy remain a major cause of maternal and foetal morbidity, occurring in 5-10% of all pregnancies (Lindheimer *et al*., 1985).

Worldwide studies showed that pre-eclampsia Pregnancy-induced hypertension complicates 10% of all pregnancies (Palacios and Pena-Rosas., 2010). It is estimated that 9.1% of maternal deaths in Africa are due to hypertensive disorders of pregnancy (Palacios and Pena-Rosas., 2010). Maternal mortality continues to be a great concern with almost (99%) all maternal deaths occurring in developing countries with more than half in sub-Saharan Africa. One in 180 pregnant women die during childbirth when compared to 1 in 4,900 in developed countries. Seventy five percent of maternal deaths occur as a result of complications due to pregnancy and childbirth.

Ghana's maternal mortality ratio declined from 760 per 100,000 live births in 1990 to 319 per 100,000 live births in 2015 (Der *et al*., 2013; World Health Organization, 2016). However, the rate of decline in maternal mortality has been slow and this led to Ghana's inability to achieve the millennium development goal target of 190 per 100,000 live births in 2015.

The maternal mortality ratio remains high and requires spirited effort for Ghana to be able to achieve the sustainable development goal target of 70 per 100,000 live births in 2030. Most maternal deaths occur in the rural settings as compared to urbans. This has largely been attributed to the high prevalence of skilled birth attendance of 74% in urban areas as compared to 43% in the rural areas (Atouye *et al.*, 2015; UNPF, 2015).

Several other reasons have been quoted as major contributory factors to maternal deaths in Ghana. Low antenatal coverage, socio-cultural factors, lack of logistics, equipment, and blood at healthcare facilities has been largely blamed as reasons for high maternal mortality in Ghana (Sophie *et al.,* 2007; Ghana Health Service, 2006).

It was observed that maternal deaths are usually directly related to causes, such as haemorrhage, unsafe abortion, hypertensive disorders, infections, and obstructed labour while indirect causes, include malaria, HIV/AIDS, and anaemia (Sedgh *et al*., 2010; GHS, 2006).

In Ghana, prevalence of pre-eclampsia amongst pregnant women has been shown to be 7.03 % (Obed *et al*., 2006). Although many other interventions have been implemented at national, regional, and community levels to reduce maternal mortality, its high ratio still remains a major concern in Ghana (Sakeah *et al*., 2014; Morhee *et al*., 2006).

The free maternal healthcare policy introduced has helped to reduce maternal mortality in the country; however, lack prevented some women from accessing

the free maternal healthcare (Asamoah *et al*., 2011). These women were unable to access the free maternal healthcare because they could not pay for the cost of transport to the nearest health facility (Ghana Statistical Service *et al*., 2008).

The Upper East Region (UER) of Ghana where poverty is rife cannot be exempted from this predicament. The 2013 annual report revealed that maternal mortality is a major public health issue as the region recorded 34 maternal deaths (Ghana Health Service, 2013).

1.2 Problem statement

Hypertensive Disorders in Pregnancies (HDP) remain a major clinical challenge in the Upper East Region of Ghana. The Region has seen a slower decline in maternal mortality rate albeit several governments interventions instituted aimed at curtailing the situation largely due to lack of transport cost to healthcare centres by maternal mothers and inadequate knowledge on some of the actual risk factors associated with pregnancy complications on the part of health providers. According to a report from the Regional Health Directorate, Upper East Region recorded 36 maternal deaths in 2016 but saw an increase in 2017 of 43 maternal deaths. Hence, the need to investigate on some of the risk factors of pregnancy induced hypertension.

1.3 General Objective

The general objective of this research is to investigate the effects of some risk factors of pregnancy-induced hypertension (PIH).

1.4 Specific Objectives

Precisely, the research will seek to;

- Evaluate the odds of a woman age to pregnancy induced hypertension.
- Evaluate the risk of hypertension at a certain level of parity.
- Evaluate the risk of hypertension at different levels gravidity.
- Examine risk factor(s) with greater impact on hypertension in pregnancy.
- Examine the model that appropriately fits the data.

1.5 Research question(s)

- What are the some of the major causes of pregnancy induced hypertension in Ghana?
- What are the common ones associated with communities in the Upper East Region (UER) of Ghana as observed by health providers?

1.6 Scope of the study

The estimated time frame for gathering information on the obstetric history of mothers and other physical measurements requires minimum six (6) weeks.

The tools needed for the research are tape measure, adult weighing scale, and a pair of scissors. The general materials required are placenta, umbilical cord and blood sample.

Common and appropriate techniques of measurements will be employed to take measurements where necessary.

The research will consider at least 50 maternal mothers for the study.

1.7 Limitations of the study

- Unwillingness of some women and their relatives to grant access to desired information.
- Difficulty in collecting a larger data sample due to cumbersome and sensitive nature of the data.
- It takes a lot of time to collect a complete information on a respondent.

1.8 Significance of the study

This study will provide government and players in the health industry alike most especially maternal health with a basic knowledge on some of the risk factors of hypertension within the Upper East Region. This will enable the government of the day to invest and tackle issues that are contributing or propelling maternal and foetal deaths in the rural and poor regions of the country.

CHAPTER TWO

LITERATURE REVIEW

2.1 Gestational Hypertension and Maternal Age

A woman who is 35 years or older at the time of delivery has been defined as being of "advanced maternal age (AMA)" (Anna *et al*., 2015).

According to the National Institute for Health and Welfare, Finland, the mean age of women giving birth has, for a long time, been around 30 years, and the average age in 2009 was 28 years among primigravids. However, the proportion of parturient aged over 35 in 2009 was 18.7%, and this percentage has been consistently increasing (NIHW, 2010).

Many researches have established the relationship between advance maternal age and extreme perinatal outcomes including the risk of certain pregnancy complications. Precisely, women of advance maternal age have an increased risk of gestational diabetes, placenta previa (Cleary-Goldman, 2005; Jacobsson, 2004, Jolly *et al*., 2000), preeclampsia (Jacobsson, 2004; Ozalp, 2003), miscarriage (Cleary-Goldman, 2005), pregnancy-induced hypertension (Jacobsson, 2004) and the need for Caesarean deliveries (Hsieh *et al*., 2010).

The risk of gestational hypertension has previously been found to be 1·22 times higher in mothers who were 35·0–39·9 years old and 1·63 times higher in mothers who were 40·0–44·9 years old than in mothers who were 25·0–29·9 years old (Timofeev *et al*, 2013).

The trend towards delayed childbearing is a well described phenomenon in high-income countries. Chronic and pregnancy-induced hypertension is associated with advanced maternal age (Lancet, 2015).

Another research revealed that expectant management of women with gestational hypertension could reduce the risk of respiratory distress syndrome in neonates (Kim *et al*., 2015).

Research has shown that low nitric oxide levels and high oxidative stress are signs of ageing and that this has the tendency to adversely affect the relaxation of the endothelium (Taddei *et al*., 2006). As such, paving way for the development of pregnancy-induced hypertension in older mothers, because pregnancy increases cardiac output.

2.2 Parity and Preeclampsia

The lower risk of pre-eclampsia among multiparous women has been attributed to desensitisation after exposure to paternal antigens in the placenta during previous pregnancies (Luo *et al*., 2007; Audrey *et al*., 2003).

The lower risk has also been attributed to smoother trophoblastic invasion after modification of maternal spiral arteries during the first pregnancy (Moore et al., 1983).

2.3 Gravidity and Gestational Hypertension

In human medicine, "gravidity" refers to the number of times a woman has been pregnant, regardless of whether the pregnancies were interrupted or resulted in a live birth (Colin, 2019).

Gravida indicates the number of times a woman is or has been pregnant, regardless of the pregnancy outcome (Cunningham, 2005). Normal labour in a primagravida is significantly different to normal labour in multiparous women, as physiologically the uterus is a less efficient organ, contractions may be poorly coordinated or hypotonic. The average first stage in a primigravida is significantly slower than in a multiparous woman (primarily due to the rate of cervical dilation) (Vahratian *et al*., 2006).

Research has revealed that a woman who has never given birth (nulliparous) and a woman experiencing her first pregnancy are at a higher risk of developing pre-eclampsia (relative risk 2.91 with confidence interval 1.28-6.61) (Duckitt *et al.*, 2005).

2.4 Gestational Weight and Gestational Hypertension

Several studies have shown that hypertensive disorders of pregnancy are more likely to develop in women who have greater gestational weight gain (Heude *et al*., 2012; Visnawathan *et al*., 2008).

Previous studies showed increasing pre-pregnant body mass index (BMI) was associated with increasing risk of gestational hypertension and gestational diabetes mellitus (GDM) (Farah *et al*, 2009; Chu *et al*, 2007). Women who are overweight or obese before pregnancy are subject to increased risk of macrosomia and dystocia (Kaiser *et al*, 2001; Shao *et al*, 2006). Women who are underweight were less likely to have adverse pregnancy outcomes, but more likely to have intrauterine growth restricted infants (Abenhaim *et al*., 2007).

Several studies suggest associations between excessive gestational weight gain (GWG) and risk of hypertension, macrosomia, and caesarean section in pregnant women of different ethnic groups (Jensen *et al*., 2007; Rong *et al*., 2015). Inadequate gestational weight gained increased the risk of preterm birth and growth-restricted infants (Haugen *et al*., 2014 and Lowell *et al*., 2010).

Studies in diverse populations throughout the world have shown that the relationship between body mass index (BMI) and systolic and diastolic blood pressure (BP) is nearly linear (Hall, 2003; Jones *et al*, 2003). A study on risk estimates from the Framingham Heart Study, for instance, suggest that 78% of primary (essential) hypertension in men and 65% in women can be attributed to excess weight gain (Garrinson, 1987). Other clinical studies specify that conservation of a BMI <25 kg/m^2 is effective in primary prevention of hypertension and that weight loss reduces blood pressure in most hypertensive subjects (Jones *et al*., 1999; Stevens *et al*., 2001).

In 2009, the Institute of Medicine (IOM) provided specific recommendations regarding the ideal gestational weight gain according to the BMI categories (Rasmussen, *et al*., 2009). However, some healthcare providers disapproved the report saying that the reference was still too high for the obese women. The obesity sorting in Japan is different as there are more underweight, for that matter the recommendation issued by the Japanese Ministry of Health, Labour and Welfare on gestational weigh gain varies from that developed by Institute of Medicine (Japan society for the study of obesity, 2002).

2.5 Cholesterol Dynamic

Amplified levels of circulating lipids result in their accumulation within endothelial cells. This accumulation decreases the release of prostacyclin, resulting in oxidative stress via endothelial dysfunction (Ghio *et al*., 2011), a key mechanism in the proposed pathophysiology of preeclampsia (Taylor *et al*., 2009).

Not quite long, a meta-analysis was completed on studies evaluating the connection amongst maternal serum triglyceride levels and preeclampsia, and the writers realized that women with preeclampsia had meaningfully higher levels of triglycerides than women with normal blood poressure (Gallos *et al*., 2013). Even though plentiful studies recommend that a dyslipidemic pattern of increased total cholesterol, triglycerides, and low-density lipoprotein cholesterol (LDL-C), laterally with decreased high-density lipoprotein cholesterol (HDL-C) concentrations may be connected with an increased risk of preeclampsia, results are inconsistent (Iftilkhar *et al.*, 2005; Uzun et al., 2005). Numerous of these studies have had small sample sizes, and the gestational age at the time of the lipid quantities has varied, making it tough to compare results across studies. The affiliation between preeclampsia and non-HDL-C, a lipid measurement reflecting atherogenic, triglyceride-rich lipoproteins (roughly 75% of which are LDL-C) (NCEP, 2002), has not often been estimated.

2.6 Umbilical Cord Characteristics

The umbilical cord is the life – line connection between the fetus and the mother through which materials such as nutrients, oxygen, and fluids necessary for intrauterine life are supplied. In view of this, abnormalities associated with

umbilical cord would have adverse effects on the perinatal outcome (Baergen *et al*., 2001).

Even though obstetricians appreciate the major role played by umbilical cord in the direction of the well-being of the fetus, it has been the effort of perinatal pathologists which have contributed meaningfully to the current facts of umbilical cord irregularities and the probable effects of these factors on the consequence of pregnancy (Sepulveda, 1999).

The development of high-resolution ultrasound and colour flow imaging procedures in prenatal care has presented the prospect to assess the morphological features of the umbilical cord and the uncovering of situations which can potentially result in hostile pregnancy outcome (Sepulveda, 1999; Collins, 2002). Current progress in ultrasonography has made it a key constituent of the guiding principle for second trimester sonographic inspection to estimate foetal anatomy and growth, placental location and amniotic fluid volume, and scrutiny of the umbilical cord (Sepulveda *et al*., 2009). Investigations on the morphological and morphometric characteristic of umbilical cord over the years have found positive association with perinatal outcome and foetal weight (Goynumer *et al.*, 2008).

Over the years, the number of umbilical cord vessels has caught the attention of researchers in assessing the morphology of umbilical cord, since single umbilical cord artery (SUA) and velamentous insertion have been established to be associated with poor pregnancy outcome (Persutte and Hobbins, 1995). Other umbilical cord abnormalities such as stillbirths, intrauterine growth restriction, non-reassuring

foetal heart tracing (NRFHT), Low Apgar Score and meconium staining have been identified with adverse perinatal outcomes (Tantbirojn *et al*., 2009).

Quite a few obstetric problems could emanate from other glitches involving the placenta and umbilical cord. Morphological characteristics of umbilical cord such as being thin; having velamentous insertion and abnormal coiling contribute to poor perinatal outcomes ((Eddleman *et al*., 1992; Sepulveda *et al*., 2003; Ghezzi *et al*., 2005; Sebire, 2007). Certain essential modifications in the structure and role of umbilical cord could put a given foetus at risk. Consequently, the ability of one foetus to bear certain umbilical cord abnormality over another may be clarified in terms of disparities in the microstructure, elemental content of umbilical cord vessels, enzymatic content and other biochemical differences in the umbilical cord (Franc *et al*., 1998; Masuda *et al*., 1999). While morphological features such as tensile strength, diameter, umbilical cord circumference, Wharton's jelly content, umbilical cord length and weight could be determined genetically, the umbilical cord development, differentiation, growth and elongation would depend on the sex, nutrient supply and health standing of the foetus (Collins, 2002).

2.7 Umbilical Cord Diameter and Area

The cross – sectional areas of umbilical cord components are vital in assessing foetal weight. A strong association amongst the cross – sectional areas of umbilical cord components and foetal anthropometric parameters has been established (Togni *et al*.,2007). Sonographic resolve of cross-sectional areas of umbilical cord components attested of the following averages and observations; umbilical diameter of 1.5cm and umbilical circumference 3.6cm after birth (Patel *et al*., 1989;

Weissman *et al*., 1994), umbilical cord vein diameter of 8mm and artery diameter of 4 mm at term (Collins, 2002).

Sonographic umbilical cord diameter and area increase as a function of gestational age. The diameter of umbilical artery increases from 1.2 ± 0.4 mm at 16 weeks to 4.2 ± 0.4 mm at term gestation and umbilical vein diameter varies from 2.0 ± 0.6 mm at 16 weeks of gestation to 8.2 ± 0.8 mm at term gestation (Di-Naro *et al*., 2001). A successive growth in umbilical cord diameter and cross-sectional area up to 32 weeks of gestation with a successive decrease in umbilical cord size was detected in a study by Raio *et al*., (1999) in which a significant connection between umbilical cord diameter, cross – sectional area and foetal anthropometric parameters was also observed.

In such alike study, it has been stated that neonates born to women with advanced pre-pregnancy weight, the male infant and heavier infants at birth lean towards to have hefty amount of Wharton's jelly covered around their umbilical cord vessels (Gill and Jarjoura, 1993). A correlation between Wharton's jelly content, umbilical cord diameter and estimated foetal weight in non – macrosomic foetuses of mothers diagnosed of gestational diabetes has been reported (Weissman *et al*., 1997).

2.8 Umbilical Cord Length Indices

It is indeterminate as what features control the length of the human umbilical cord; though, both genetic and environmental factors have been allied with the determination of umbilical cord length. It has been testified that growing of the umbilical cord, placenta and body length may be under comparable control mechanisms some of which are expected to be genetic in origin (Baergen *et al*.,

2001). The "tension theory" proposes that the length of the umbilical cord is presumed to increase with the tensile force applied to it in the uterus, and the utmost tensile force being the foetal movement which necessitates tolerable space within the amniotic cavity. Any intrauterine limitation of its kind would lessen the tensile force resulting in the length of the umbilical cord being short (Lyndon *et al*., 1994). Benirschke (2004) observed that human umbilical cord grows steadily with growing gestation and foetal crown – rump length; and measures roughly 55 cm long at term.

Hostile perinatal outcomes have been observed in awfully short and tremendously long umbilical cords. Short umbilical cords are projected to be less than 40cm whereas long umbilical cords are greater than 70cm long. The umbilical cord length is the only issue documented to show high risk for poor foetal outcome. A strong relation between abnormal umbilical cord and neurological abnormalities and low IQ has been detected (Baergen *et al*., 2001). An average length of 50-60cm is measured normal in full-term newborn which also mirrors intrauterine foetal motility. An abnormally short umbilical cord predisposes the umbilical cord to rupture, haemorrhage, stricture, malpresentation, extended second stage labour, abruption and intrauterine inversion. Whiles too long umbilical cord is known to be associated with entanglement, torsion, knots and thromboses.

It also strongly associated with high rate of asphyxia during delivery, foetal anomalies, non-reassuring foetal status, respiratory distress, foetal growth restriction and delivery interventions. Several researches have revealed that a positive correlation exists between umbilical cord length and parity, pregnancy

weight and foetal sex (Baergen *et al*., 2001; Stefos *et al*., 2003). The umbilical cords of male neonates are found to be longer than females and term vertex foetuses could have long umbilical cords than term breech foetuses (Calvano *et al*., 2000).

2.9 Umbilical Cord Vessel Number and Insertion

The number of umbilical cord vessels is as imperative as the amount of Wharton's jelly and cord length during morphological valuation of the umbilical cord. Usually, an umbilical cord would have two arteries and a vein implanted in Wharton's jelly (Gouden, 2003). Yet the umbilical cord vessel number may vary resulting in certain foetal abnormalities. Umbilical cord vessels totaling two, four or five and fused cords in twins have been observed which associated with known foetal anomaly (Cohen *et al*., 1992; Schimmel *et al*., 1998). Structurally, the walls of umbilical cord arteries and the vein are identical; the intima has a thin layer of endothelial cells, collagen, elastin and a matrix (Pennati, 2001). Koech *et al*., (2008) stated that in preeclamptic cords, there was an increase in thickness of the tunica media and intima in the arteries and higher rate of internal elastic lamina duplication. However, a reduced vessel diameter and wall thickness in both cord artery and the vein in preeclampsia as against normal pregnancies and pregnancies affected by chronic hypertension have been observed (Inan *et al*., 2002). Single umbilical artery (SUA) is the more common congenital abnormality of the umbilical cord, occurring in approximately 0.2 to 1 percent of all human pregnancies (Heifetz, 1984). Again, the prevalence of SUA is known to be between 0.5-2.0% in uncomplicated neonates and 1.5-7% in aborted and aneuploid (9-11%) foetuses. Multiple gestations rank 3-7 times higher risk of SUA (Di-Naro *et al*., 2001).

It is presumed that the roots of growth of SUA may include primary agenesis, secondary atrophy or atresia, and tenacity of the single allantoic artery in the body stalk (Persutte *et al*., 1995). Single umbilical artery strongly relates with stillbirth with an incidence rate of 3-20%, more recurrent in twins, diabetic pregnancies, usually associated with long cords and small placentae (Collins, 2002). Early discovery of SUA therefore calls for a comprehensive sonographic study to identify any of the anomalies that associates to SUA (Hamada *et al*., 2001). Fascinatingly, the organ systems of the foetus which commonly suffer structural abnormalities as a result of SUA, from mild to severe are cardiac, gastrointestinal, central nervous system, genitourinary, respiratory and musculoskeletal systems (Gouden, 2003). Majority of SUA display a major than usual arterial diameter, approaching half or equal the diameter of the umbilical vein. The transverse intraluminal umbilical artery diameter dimension is believed to offer the needed support in the identification of this anomaly (Sherer *et al*., 1997).

In a study, all pregnancies identified with SUA 20 to 36 weeks of gestation had umbilical arterial diameter measuring greater than 4mm whiles all pregnancies with two umbilical arteries had arterial diameter less than 4mm (Persutte *et al*., 1994). In their view, Sepulveda *et al*., (1996) suggested the use of umbilical vein diameter to arterial diameter ratio instead of the bigger arterial diameter in diagnosing SUA. Comparing 55 SUA foetuses with 55 control foetuses with two umbilical arteries, these investigators detected in all but one foetus with SUA that this ratio is ≤ 2; however, none of the controls had a ratio ≤ 2.

The umbilical cord is purposefully made to enable foetal growth until delivery; consequently, the cord needs not to separate which in turn mandates a particular anatomy of its insertion to both the foetus and the placenta. Failure of such an attachment would results in foetal demise (Collins, 2002). The Anatomy of the umbilical cord is such that its point of insertion onto the placenta relies deeply on the implantation of the blastocyst. Umbilical cord usually inserts into the placenta at the center (Centric) or near the centre (Eccentric). However, when the blastocyst fails to attach at the embryonic pole, the connecting stalk may attach at the margin or to the smooth adjacent chorion resulting in marginal or velamentous insertions respectively as pregnancy advances in age.

Centric and eccentric umbilical cord insertions are found in more than 90% of all cord insertions into the placenta, this is tailed by marginal also known as battledore and the least occurring is velamentous insertion. These are commonly used terminologies for the purpose of qualitative judgment of cord insertions (Pathak *et al*., 2010). The centric and eccentric cord insertions are considered normal and have no medical importance. Marginal cord insertion is known to be associated with vessel rupture, preterm labour, intrauterine growth restriction, stillbirth, and neonatal death. The regularity of velamentous insertion increases with maternal risk factors such as maternal smoking habit, advanced age, or diabetes mellitus and multiple births (Heifetz, 1996). The distance of umbilical cord insertion from the placental center is clinically established as a good indicator of maternal insufficiency (Whittle *et al.,* 2006). This can be well explained by the answer to the question, “How might a given placenta size yield different birth weights?” The

genetics of the mother affects the constitutionally appropriate birth weight and more so, on the placental weight. Maternal weight gain, medical disorders, environmental exposure and lifestyle including substance abuse, tobacco use, etc. can alter the foetal and the placental growth. With these maternal factors apart, it has been proven that deviation of placental proportions from round and distance of umbilical cord insertion; modify the functional efficiency of the placenta (Yampolsky *et al*., 2009). The association of the shape of placenta with the placental efficiency lies in the design of the placental vascular tree which happens to be the only provider of foetal nutrient and oxygen. The chorionic plate vessels produce high capacitance blood supply machinery to ensure bulk transportation of blood, fast sufficient from the umbilical cord to the placental villi where nutrient and oxygen are exchanged and retreats to the umbilical cord again. Discrepancies in the vital proportions of the placental disk and the structure of the vascular tree would therefore reduce the level of transportation competence, hence the ability of the placental mass to functionally yield foetal mass (Yampolsky *et al*., 2008). Placentae with non – centrally inserted umbilical cord are characterized by thinly spread vascular coverage, heavy weight, large diameter and are thicker (Yampolsky *et al*., 2009). Pathak *et al*., (2010), qualitatively define the types of umbilical cord insertions as follow:

i. Centric insertion is defined as the umbilical cord inserting within 2 cm of the center of the chorionic plate.
ii. Eccentric insertion is defined as the umbilical cord inserting greater than 2 cm from the center and within the margin of the chorionic plate.

iii. Marginal insertion is when the umbilical cord inserts into the margin of the chorionic plate.

iv. Velamentous insertion is when the umbilical cord inserts outside the chorionic margins into the membranes. These researchers derived indices that describe quantitative association of the umbilical cord insertion into the placenta.

They computed the cord centrality and eccentricity indices mathematically to describe the closeness or farther the cord insertion is from the placental center and the shape of placenta relative to whether it is circular or ovoid respectively.

2.10 Placenta Characteristics

Placenta is important for conservation of pregnancy and for helping normal growth and development of fetus (Udaina *et al*., 2001). It is the most exact record of the infant's prenatal practise (Bernirshke *et al.,* 1981). It forms the morphological record of anatomical state, intrauterine events and intrapartum events of gestation. Pregnancy problems like hypertension are reflected in placenta in an important way both macroscopically and microscopically. Several studies have shown that utero-placental blood flow is reduced in PIH owing to maternal vasospasm (Bewly *et al.,* 1991). This leads to tightening of foetal stem arteries and has been connected with the changes seen in the placenta of preeclamptic women (Stock *et al.,* 1980). Maternal vasospasm leads to foetal hypoxia and accordingly it may lead to foetal suffering and foetal death (Thomson *et al.,* 1969).

2.11 Placental Weight

The placenta has complex metabolic and endocrine activities and is significant for progress and survival of the foetus in utero. Foetal growth is controlled by the comparison between foetal metabolic demand and maternal-placental supply which is strictly related to utero-placental blood flow, placental size and its transfer capabilities. A reduced amount of maternal placental supply than is needed would advise that the foetus must try to familiarize to the condition by the alteration of its body structure and endocrine status, choosing growth of specific organs and using cardiovascular adaptations (Pardi *et al*., 2002). The weight of the placenta can only be measured after delivery, however, the measurements of the delivered placenta show the systematic growth of the placenta right from conception to delivery. The measurement improves the ability to carefully observed variances between individual dimensions in intrauterine experience as well as providing a biologically active technique to spot the physiology of the foetal experience. The growth of the placenta is directly proportional to its functional efficiency as the only foetal source of both nutrients and oxygen (Salafia *et al.*, 2005).

A term placenta measures between 15 to 25 cm in diameter with a thickness of about 3 cm and weighs from 500 to 600g (Sadler, 2004). Placental size measures, including placental area and thickness, affirm placental efficiency and growth. In the first place, they indicate two different dimensions of placental growth: the area reflects lateral expansion of the chorionic disc; whiles thickness specifies vertical arborization of the villous and vascular nutrient exchange (Salafia *et al.*, 2005; Salafia *et al.*, 2008; Barker *et al.*, 2010).

Additionally, they almost replicate changed stages of intrauterine environment adequacy. For example, while placental area development is almost complete by early part of the third trimester, the placental thickness growth mostly happens late in the third trimester. Again, they may associate with the badly-behaved of the foetal cardiovascular system, such as cardiac workload and hemodynamic burden (Salafia *et al.*, 2005).

Also, little is known about the affiliation between measurements, including the size of the placenta, the shape of the chorionic plate (foetal surface), distance of the umbilical cord insertion from the center of the placenta and the deviation in placental shape from the typical normal circular appearance and their association with pregnancy and neonatal outcome (Pathak *et al.*, 2010).

Several umbilical cord insertions into the chorionic plate are described qualitatively as central, eccentric, marginal (Battledore) and velamentous (membranous). Marginal and velamentous insertions are believed to result from instabilities of implantation (Kouyoumdjian, 1980).

Central and eccentric insertions constitute more than 90%; whereas marginal and the least frequent is velamentous forms the remaining 10%. On the conflicting, factor accounting for the eccentric (paracentral) cord insertion is still not clearly defined. Cord insertion at any place between central and marginal insertion is commonly considered to be eccentric, yet none of these terms has been quantitatively described (Pathak *et al.*, 2010). The kind of cord insertion may also be defined as how far the insertion point is situated from the center of the placenta, or how near the umbilical cord insertion is to the chorionic plate margin. The

distance of the umbilical cord insertion from the placental center has been proposed as a clinically useful marker of placental insufficiency (Viero *et al.,* 2004; Whittle *et al.,* 2006).

2.12 White Blood Cell Indices

The erythrocytes, the most copious cells in the bloodstream, suffer greatly from the effects of PE. The disease is associated with important changes in the erythrocyte morphology (Hernandez *et al.,* 2008) with early degeneration and de-structuring of its membrane, leading to lysis in the bloodstream (Lurie *et al.,* 2000; Heilman *et al.,* 2004).

For the good performance of its functions, the erythrocyte must remain intact. The red blood cell's ability to maintain the physical and chemical integrity of its membrane in adverse situations is called stability (Bernadino *et al.,* 2013; Ozan *et al.,* 21997). In this logic, the ability of the biological membrane to resist disintegration in the face of mechanical hostility encouraged by the blood flow itself and the friction with the wall of the blood vessels constitutes the so-called mechanical stability, whereas its ability to remain unbroken due to volume expansion in a hyposmotic environment is called osmotic stability (Shiga *et al.,* 1990).

Though the evaluation of osmotic stability of the erythrocyte membrane is well establishhed in the literature (de Freitas *et al.,* 2014; Mascarenhas *et al.,* 2014), the number of studies on osmotic stability of erythrocyte membrane in preeclampsia is still quite small (Aires *et al.,* 2018; Ozan *et al.,* 1997; Abad *et al.,* 2010).

The mechanical stability of erythrocytes also deserves attention (Gu *et al.*, 2014; Tarasev *et al.*, 2016) particularly for the analysis of erythrocyte membrane behavior in conditions of increased mechanical aggression, which is the key task found by red cells in the blood circulation of women with preeclampsia.

2.13 Haemoglobin Indices

While the etiology of preeclampsia is unidentified, hemodynamic studies recommended that several of the clinical results might be clarified by a comprehensive vasoconstrictive disorder and abnormal endothelial cell function. Vasoconstriction could be attributed to the amplified concentrations of hemoglobin found in preeclampsia equated with normal pregnancy. Free hemoglobin may be resulting from hemolytic placental hemorrhage and in concentrations identified to be existing in preeclampsia. Vasodilatation facilitated by endothelium-derived relaxing factor is repressed. Infusion of oxyhemoglobin into human coronary arteries hampers acetylcholine-induced vasodilatation. An increased free hemoglobin concentration is the cause of vasoconstriction in preeclampsia (Sarel *et al*., 1990). In women who have hypertensive disorders of pregnancy, essentially those with preeclampsia, blood volume does not upsurge, which results in a comparatively higher hemoglobin concentration (Yip *et al*., 2000). Pritchard *et al*., 1984, disclosed that the average hematocrit for women with preeclampsia which was 0.405, likened with a mean of 0.374 for women with normal pregnancy.

Most women with a pregnancy-induced hypertensive disorder are symptomless, which is an imperative part of the defense for regular antenatal visits in pregnancy. Laboratory tests have been used for prediction, diagnosis, and monitoring of the

disease advancement. The judgment of pre-eclampsia is even based on a laboratory test (Dekker *et al.,* 2001). Some studies have connected high maternal serum hemoglobin levels (Hb≥13.2) in pregnancy consequences, such as preeclampsia, preterm birth and small for gestational age (Tarim *et al.*, 2004). Many investigators believe that the circumstances for the progress of preeclampsia are set as early as the first trimester (Vedernikov *et al.,* 1999). In one study in primiparous, the regularity of subsequent hypertension ranged from 7% at Hb values under 10.5g/dl to 42% at Hb concentrations over 14.5g/dl (Murfy *et al.,* 1986).

2.14 Red Blood Cells Indices

There are instances of literatures which on theories about preeclampsia. Firstly, defective trophoblast attack was attributed to cause preeclampsia (Pijnenborg *et al.,* 1983). Another theory about preeclampsia was vicissitudes in immune system of pregnant women triggering increased inflammatory reply. According to the latter theory, increased inflammatory response leads to wrong placentation, increased capillary permeability, microvascular thrombosis, and increased vascular tonus (Pijnenborg *et al.,* 1983; Saito *et al.,* 2007). Mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), mean corpuscular hemoglobin concentration (MCHC), red cell distribution width (RDW), and red blood cell (RBC) are acknowledged as the red blood cell indices. MCV defines the size of the red blood cells. MCH determines the amount of hemoglobin per red blood cell. MCHC indicates the amount of hemoglobin per unit volume (Bessman *et al.*, 1983). Again, RDW is an indicator of red cell size variation called anisocytosis. It mirrors early fluctuations in red blood cells and all these parameters are central for detecting

and studying anemia (Sultana *et al.,* 2011). They are regularly assessed in a fully automated hematologic analyzer, as part of the complete blood count. Similarly, it has been shown that RDW was higher in prehypertension. However, prehypertension was labelled as slightly elevated blood pressure which would likely turn into high blood pressure (hypertension) in non-pregnant people if routine changes like eating healthier and beginning to exercise were not done (Tanindi *et al.,* 2011). Moreover, the association between RDW values and non-cardiac and cardiac mortality in patients with cardiovascular and thrombotic disorders, diabetic ketoacidosis, acute and chronic heart failure, coronary artery disease, and stroke has been explored (Montagnana *et al.,* 2011; Liu *et al.,* 2013). However, in the literature, there have been limited data on the relationship between red blood cell indices, including RDW, and preeclampsia.

2.15 Blood Sugar Level (Glucose)

Insulin resistance has also been conjectured to contribute to the pathophysiology of preeclampsia. Likened to women who have normotensive pregnancies, women who develop preeclampsia are more insulin resistant prior to pregnancy (Valdes *et al*., 2014), in the first and second trimesters (Hauth, *et al.,* 2011), and years after pregnancy (Alsnes, *et al.*, 2014). This consequence is somewhat clarified by the element that many preeclampsia risk factors are also connected with insulin resistance, for example, obesity, advanced maternal age, non-white race, chronic hypertension, diabetes and gestational diabetes (Valdes *et al.,* 2014; Hauth *et al.,* 2011). However, insulin resistance at 22-26 weeks gestation was a noteworthy

independent predictor of preeclampsia after adjustment for these common risk factors, signifying an independent effect (Hauth *et al.,* 2014).

A study conducted by Charley conveyed a bigger risk of late onset diabetes among women with a history of eclampsia (Charley, 1976). This severe form of preeclampsia causes seizures and leads to very high rates of maternal and fetal morbidity and mortality. Current investigations recommend that preeclampsia is also a risk factor for future diabetes (England, 2011; Savitz, 2014). This consequence is obvious even when women who had preeclampsia with gestational diabetes are omitted (England, 2011; Feig, 2013). However, preeclampsia is an uncertain predictor of future diabetes when equated to gestational diabetes. In considering both pregnancy conditions, the risk of growing diabetes is soberly increased in women who had preeclampsia (without gestational diabetes), seriously raised in women who had gestational diabetes (without preeclampsia), and peaks in women who had both preeclampsia and gestational diabetes (England, 2011; Feig, 2013).

A registry study of 226,832 women in Norway exposed that only 0.5% of women without GDM or preeclampsia had received a prescription to treat diabetes within five years of birth, while 2% of women with preeclampsia had received such a prescription, 19% of women with GDM diagnoses were taking diabetes drugs and over half (55%) of women with both conditions had received a diabetes prescription (England, 2011). Similarly, Feig and colleagues informed that the number of women that would need to be tracked for five years to detect one case of diabetes

was 123 for preeclampsia, 68 for gestational diabetes, and 31 for preeclampsia and gestational diabetes (Feig, 2013).

2.16 Body Mass Index

Obesity has been associated with a 2-4-fold bigger risk of preeclampsia in different populations (Cnattingius *et al.*, 1998; Bhattacharya *et al.*, 2007), and thus, it is a leading known attributable risk for this disorder. A population-based study from Dar es Salaam, Tanzania, stated that the prevalence of obesity amongst women of reproductive age amplified more and more from 3.6% in 1995 to 9.1% in 2004 (Villamor *et* al., 2006). The Tanzanian Demographic Health Survey for the years 2004 and 2005 reported a prevalence of 13% and 4%, respectively of overweight and obesity among women of reproductive age (National Bureau of Statistics, 2005).

Meanwhile, since clinical histories of births are deficient or not appropriate for investigation in many African countries, research on preeclampsia in women of African origin have been mostly based on immigrants to high income countries or descendants of immigrants (Nakimuli *et al.*, 2014)]. Other studies have described that women of African beginning are at increased risk of preeclampsia, but it is not clear to which level this is clarified by the existence of specific risk factors for preeclampsia. Similarly, studies on the association amongst preeclampsia and obesity are mainly based on women in resource rich countries (Jayabalan, 2013).

The etiology, epidemiology and cultural significance of overweight and obesity possible vary from population to population. There are also suggestions that the course and outcome of preeclamptic pregnancies fluctuates by race and ethnicity

(Goodwin *et al.,* 2005). These characteristics call for assemblage of high-quality data to study overweight and obesity as risk factors of preeclampsia in native African women.

CHAPTER THREE

METHODOLOGY

3.0 Introduction

This chapter focuses on the analyses of the results obtained from the sampled data. The statistical analyses software STATA (version 12) and IBM (SPSS statistics 20) where the main statistical tools used for the analysis. However, due to the nature of the dependent/outcome variable and for that matter binary, the logistic regression model, the probit regression model as well as the artificial neural network (ANN) all of which are binary classifiers were adopted for the final analysis.

3.1 Data and Source

A case-control study was conducted in the Upper East Regional Hospital, Bolgatanga in the Upper East Region of Ghana. Data on the history of 50 pregnant women during their gestational period was collected for the study.

3.1.1 Variables

- Dependent variables

Pregnancy Induce-Hypertension (PIH) and Normotension

- Independent variables

Mothers age (MA), Parity, Gravidity, Gestation Weight (GW), Glucose level, Placenta length (PL), Placenta weight (PW), Umbilical cord length (UCL), Umbilical cord diameter (UCD), Cholesterol level, Body Mass Index (BMI), High Density – Lipoprotein cholesterol (HDL-C), Low Density Lipoprotein cholesterol

(LDL-C), White Blood Cell (WBC), Red Blood Cell (RBC) and Haemoglobin (Hb).

3.2 Binary Models

The study will use the probit model and logistic model as well as the Artificial Neural Network (ANN) to analyze the data. Most binary outcomes utilize either the Probit model, Logit model, Logistics regression model. Logistics give odd ratios. In logistic regression the odds ratio represents the constant effect of a predictor X, on the likelihood that one outcome will occur. The dependent variables for this study are binary outcomes where pregnancy induced hypertension (PIH) equal to one (1) as the value of interest and zero (0) otherwise.

The model for the logistics regression is given as;

y = (1 with probability p, 0 with probability 1-p) (3.1)

3.2.1 Logistic Model

Logistic fits a logistic regression model of dependent variable on independent variables, where dependent variable is a 0/1 variable. Logistic display estimates as odds ratios. The logistic regression model specifies

$$p = \frac{e^{x\beta}}{1 + e^{x\beta}} \qquad (3.2)$$

The above equation can be transformed as

$$e^{x\beta} = \frac{p}{1 - p} \qquad (3.3)$$

Here, $\frac{p}{1-p}$ measures the probability that $y = 1$ relative to the probability that $y = 0$ and is called odd ratio or relative risk.

3.2.1.1 Assumptions of Logistic Regression

i. Binary logistic regression requires the dependent variable to be binary and ordinal logistic regression requires the dependent variable to be ordinal.

ii. Logistic regression requires the observations to be independent of each other. In other words, the observations should not come from repeated measurements or matched data.

iii. Logistic regression requires there to be little or no multicollinearity among the independent variables. This means that the independent variables should not be too highly correlated with each other.

iv. Logistic regression assumes linearity of independent variables and log odds. Although this analysis does not require the dependent and independent variables to be related linearly, it requires that the independent variables are linearly related to the log odds.

v. Finally, logistic regression typically requires a large sample size. A general guideline is that you need at minimum of 10 cases with the least frequent outcome for each independent variable in your model.

3.2.1.2 Limitations

i. Logistics regression model cannot predict continuous outcomes

ii. Independence of observations is a disadvantage because a lot of scientific and social scientific research relies on research technique involving multiple observations on the same individual.

iii. Logit models are vulnerable to over confidence. That is, the model can appear to have more predictive power than they actually do as a result of sampling bias.

3.3 Probit Model

A more formal mathematics of probit is given as

$$Pr\left(y_{(j\neq 0|x_j)}\right) = \phi(x_{j\beta}), \qquad (3.4)$$

where ϕ is the standard cumulative normal and β is the co-efficient of parameter x_j.

The log-likelihood function for the probit is:

$$\ln L = \sum_{j\epsilon s} \omega_j ln\phi(\phi x_j\, \beta) + \sum_{j\notin s} \omega_j \ln\{1 - \phi(x_j\beta)\} \qquad (3.5)$$

where ϕ is the cumulative normal and ω_j denotes the optional weights. ln L is maximized.

3.3.1 Assumptions

i. It requires observations to be independent

ii. The dependent variable takes only two values.

iii. They should be sufficiently large number of observations.

iv. It assumes that random errors have multivariate normal distribution

3.3.2 Limitations

i. The only limitation of probit models is that they require normal distributions for all unobserved components of utility.

3.4 Artificial Neural Network (ANN).

Neural networks are made of individual perceptrons whose output y_j can be written as

$$y_j = f_j\left(\Sigma_i w_j x_{j,i}\right), \qquad (3.6)$$

where $x_{j,i}$ are the inputs of the perceptron, $\omega_{j,i}$ its weights and f_j a so called "activation function", typically the sigmoid:

$$\sigma_s(x) = \frac{1}{(1 + e^{-sx})}. \qquad (3.7)$$

The perceptrons are organized in such a way that the output of a perceptron located upstream becomes the input of a perceptron downstream, forming *de facto* a net organized in three types of layers:

i. The input layer made of the observed predictors.

ii. The hidden layer(s) containing non-observed perceptrons;

$$z_k = \sigma_S\left(\omega_{0,k} + \sum_{i=1}^{p} \omega_{i,k} x_j\right), \qquad (3.8)$$

computing the nonlinear features from linear combinations of the inputs.

iii. The output layer containing the probability of failure.

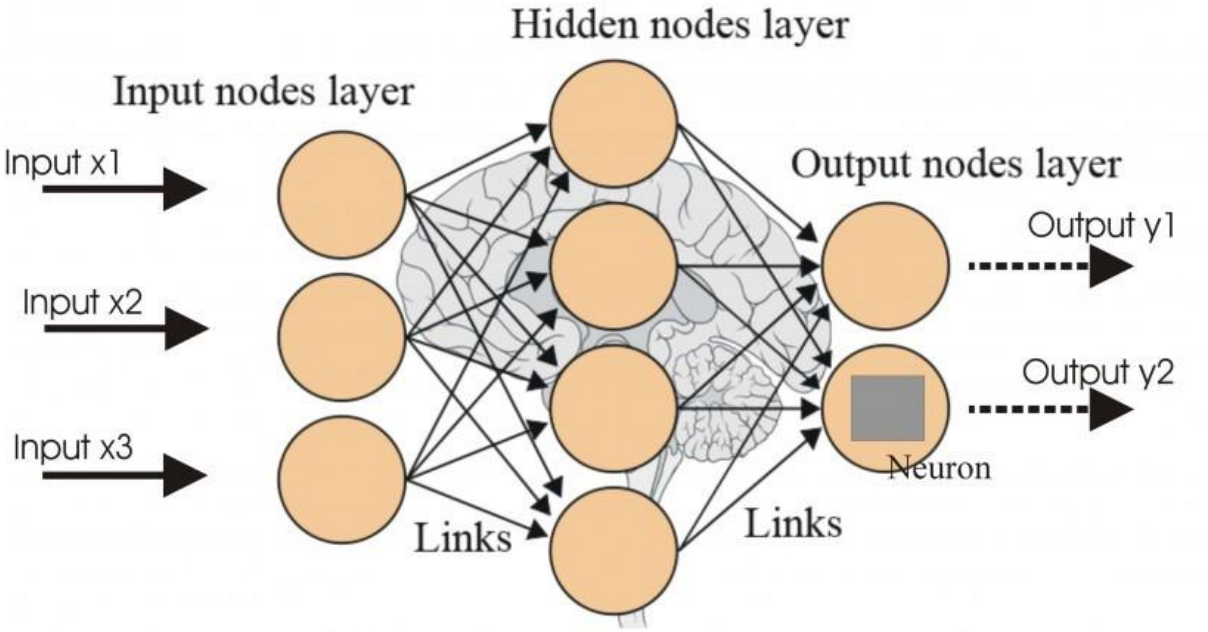

Figure 3.1 Good Audience human brain view of layers of the Artificial Neural Network

3.4.1 Advantages

i. Storing information on the entire network: Information such as in traditional programming is stored on the entire network, not on a database. The disappearance of a few pieces of information in one place does not prevent the network from functioning.

ii. Ability to work with incomplete knowledge: After ANN training, the data may produce output even with incomplete information. The loss of performance here depends on the importance of the missing information.

iii. Having fault tolerance: Corruption of one or more cells of ANN does not prevent it from generating output. This feature makes the networks fault tolerant.

iv. Having a distributed memory: In order for ANN to be able to learn, it is necessary to determine the examples and to teach the network according to the desired output by showing these examples to the network. The network's success is directly proportional to the selected instances, and if the event cannot be shown to the network in all its aspects, the network can produce false output

v. Gradual corruption: A network slows over time and undergoes relative degradation. The network problem does not corrode immediately.

vi. Ability to make machine learning: Artificial Neural Networks learn events and make decisions by commenting on similar events.

vii. Parallel processing capability: Artificial Neural Networks have numerical strength that can perform more than one job at the same time.

3.4.2 Disadvantages of Artificial Neural Networks (ANN)

i. Hardware dependence: Artificial neural networks require processors with parallel processing power, in accordance with their structure. For this reason, the realization of the equipment is dependent.

ii. Unexplained behavior of the network: This is the most important problem of ANN. When ANN produces a probing solution, it does not give a clue as to why and how. This reduces trust in the network.

iii. Determination of proper network structure: There is no specific rule for determining the structure of artificial neural networks. Appropriate network structure is achieved through experience and trial and error.

iv. Difficulty of showing the problem to the network: ANNs can work with numerical information. Problems have to be translated into numerical values before being introduced to ANN. The display mechanism to be determined here will directly influence the performance of the network. This depends on the user's ability.

v. The duration of the network is unknown: The network is reduced to a certain value of the error on the sample means that the training has been completed. This value does not give us optimum results.

3.5 Statistical Test

i. **Receiver Operation Characteristics Curve (ROC)**; is a graphical plot that illustrates the diagnostic ability of a binary classifier system as its discrimination threshold is varied.

ii. **Sensitivity (True Positive Rate)**; measures the proportion of actual positives that are correctly identified as such (e.g., the percentage of sick people who are correctly identified as having the condition).

iii. **Specificity (True Negative Rate)**; measures the proportion of actual negatives that are correctly identified as such (e.g., the percentage of healthy people who are correctly identified as not having the condition).

iv. **Classification Table**; the classification table is another method to evaluate the predictive accuracy of the logistic regression model. In this table the observed values for the dependent outcome and the predicted values (at a user defined cut-off value, for example Probability = 0.50) are cross-classified.

CHAPTER FOUR

4.0 RESULTS AND DISCUSSIONS

4.1 Preliminary Analysis

The preliminary results of the study are presented in Table 4.1. The results entail the maternal indices of 50 women who were enrolled for the study of which 16 explanatory variables were considered including maternal age (MA), gravidity, parity, gestational weight (GW), blood – sugar level (glucose), placenta length (PL) and placenta weight (PW), umbilical cord length (UCL), umbilical cord diameter (UCD), cholesterol level (CL), body mass index (BMI), high density lipoprotein (HDL-C), low density lipoprotein (LDL-C), white blood cells (WBC), red blood cells (RBC) and hemoglobin (HB).

The study revealed that maternal ages for the sampled mothers ranged from 16yrs as the minimum to a maximum age of 41yrs. The average age for maternal mothers was 27.84yrs with a standard deviation (SD) of 6.548. The parity level of maternal women in the study ranged from 0 to 6 births with an average of 1.54 births. The standard deviation (SD) was 1.581births.

However, it was discovered that the number of pregnancies (gravidity) was between 0 to 7 pregnancies and the mean number of pregnancies for the 50 women was 2.56 with a standard deviation of 1.668 pregnancies. The weight of mothers as a result of the pregnancy or the gestational weight (GW) had an average of 70.44kg and a standard deviation of 16.218kg. The gestational weight (GW) for the 50 women extended from 43.7kg to 130kg.

Likewise, the blood-sugar level (glucose) was determined and the results revealed that blood-sugar level ranged from 7.9mg/dL to 35mg/dL. The average level of blood-sugar for the study population was 28.17mg/dl with a standard deviation of 9.417mg/dL. Also, the placenta length of mothers was measured with an inelastic tape measure after delivery and the values recorded. The placenta length ranged from 7.5cm to 35cm with a mean length of 20.09cm for the study population. The standard deviation was found to be 4.524cm.

Placenta weight was determined using a weighing scale and the measurement was recorded in grams. The mean placenta weight for the population under study was 613g with variation from the mean (SD) of 170.766g. However, the placenta weight ranged from 300g to 1000g for the 50 women.

The study revealed that umbilical cord length for mothers had a minimum value of 20cm and a maximum value of 85cm. The average value for umbilical cord length of the investigated population was 50.96cm and had a standard deviation of 11. 719cm. The umbilical cord diameter was determined using a pair of dividers and then transferred onto a standard rule for the values to be taken. The study showed that the average cord diameter for mothers under investigation was 1.23cm and ranged from 0.3cm to 2.2cm. Deviation from the mean was 0.55cm.

The lipid panel which was conducted using the blood samples from the 50 maternal mothers enrolled for the study showed that cholesterol level of mothers ranged from a minimum value 17.3mg/dL to maximum value of 57mg/dl with a mean of 35.52mg/dl. The standard deviation (SD) for the lipid profile was 9.01mg/dl.

The body mass index (BMI) for the pregnant women a minimum value of 7.3kg/m^2 and a maximum of 47.2kg/m^2. The mean BMI for the group under study was pegged at 25.723kg/m^2 with a standard deviation (SD) of 6.802kg/m^2. The results did reveal that high-density cholesterol (HDL-C) had a mean of 1.96mg/dL and standard deviation (SD) of 0.856mg/dL. The maximum high-density cholesterol level was 0mg/dL and a maximum value of 4mg/dL.

Similarly, low-density cholesterol (LDL- C) had a maximum value of 16.63mg/dL and a minimum value of 0.3mg/dL. The mean LDL-C was 6.435 with a standard deviation (SD) of 4.666mg/dL. Nevertheless, blood counts of the maternal group indicated that white blood cells averaged 13.539 with a standard deviation (SD) of 6.236. The maximum value for WBC count was 30.95 and a minimum vale of 4.78. Again, red blood cell count had a maximum value of 5.13 and a minimum value of 2.24. the mean value for red blood cells count was 3.823 with a standard deviation (SD = 0.563).

To conclude, the results specified that hemoglobin (Hb) averaged 10.378 with a standard deviation (SD = 1.456). The minimum Hb level was 5.2 whiles the maximum value was 12.8.

Table 4.1: Descriptive statistics of variables.

Variable	Mean	SD	Minimum	Maximum
MA (yrs.)	27.84	6.547659	16	41
Parity	1.54	1.580622	0	6
Gravidity	2.56	1.667945	0	7
GW (kg)	70.436	16.2183	43.7	130
Glucose (mg/dL)	28.174	9.41672	7.9	52.9
PL (cm)	20.09	4.523961	7.5	35
PW(g)	613	170.7755	300	1000
UCL (cm)	50.96	11.71944	24	85
UCD (cm)	1.232	0.5490028	0.3	2.2
Cholesterol (mg/dL)	35.524	9.01012	17.3	57
BMI (kg/mm2)	25.72326	6.802333	7.28571	47.2
HDL- C(mg/dL)	1.96	0.8561899	0	4
HDL-C(mg/dL)	6.435	4.665768	0.3	16.62
WBC (mm3)	13.5908	6.263665	4.78	30.95
RBC (mm3)	3.823	0.5631915	2.24	5.13
HB (gm/dL)	10.378	1.456343	5.2	12.8

4.2 Area Under ROC Curve

Table 4.2 shows the true mean values of the predictor variables. From the summary results, the ROC area values for all the various variables fall under the 95% confidence interval i.e. the upper and the lower bounds. This further insinuates that we are 95% certain that the true mean of each variable falls within the range of the data or the population under study. Hence, we can confidently say that the data was free of outliers.

However, in this case, no model has been applied, the receiver operating characteristics (ROC) area values and other preliminary test are just for the preliminary analysis of the data.

Table 4.2: ROC area for independent variables

	ROC area	
Variables	**Area**	**[95% Confidence Interval]**
MA (yrs.)	0.6133	0.44636 0.78031
Parity	0.6317	0.47350 0.78983
Gravidity	0.6333	0.47828 0.78838
GW (kg)	0.6633	0.50632 0.82034
Glucose (mg/dL)	0.3758	0.21063 0.54104
PL (cm)	0.475	0.29393 0.65607
PW(g)	0.41	0.23263 0.58737
UCL (cm)	0.5167	0.34793 0.68540
UCD (cm)	0.5033	0.34091 0.66576
Cholesterol (mg/dL)	0.4667	0.29385 0.63949
BMI (kg/mm2)	0.6925	0.53860 0.84640
HDL- C(mg/dL)	0.4792	0.32093 0.63740
HDL-C(mg/dL)	0.4992	0.33529 0.66304
WBC (mm3)	0.3367	0.17768 0.49566
RBC (mm3)	0.3775	0.21315 0.54185
HB (gm/dL)	0.3533	0.17462 0.53205

Figure 4.1 represents the receiver operating characteristics (ROC) area curves for mothers' age and mothers' parity level. In Figure 4.1, the vertical axis is the true positive rate (sensitivity) i.e. the proportion of actual positives that are correctly identified as such. The horizontal axis also indicates the false positive rate

(specificity) i.e. the measure of proportion of actual negatives that are correctly identified as such. Figure 4.1 specifically indicates the ROC area values for mothers age (0.6133) and that of mothers parity level (0.6317) as well as standard error and the confidence intervals. From the Figure, there is 0.633 probability of correctly identifying the proportion of true positives (women with pregnancy induced hypertension) and 0.6317 chance of correctly identifying the proportion of the trues positives (women with pregnancy induced hypertension) taking into account their age and parity levels respectively.

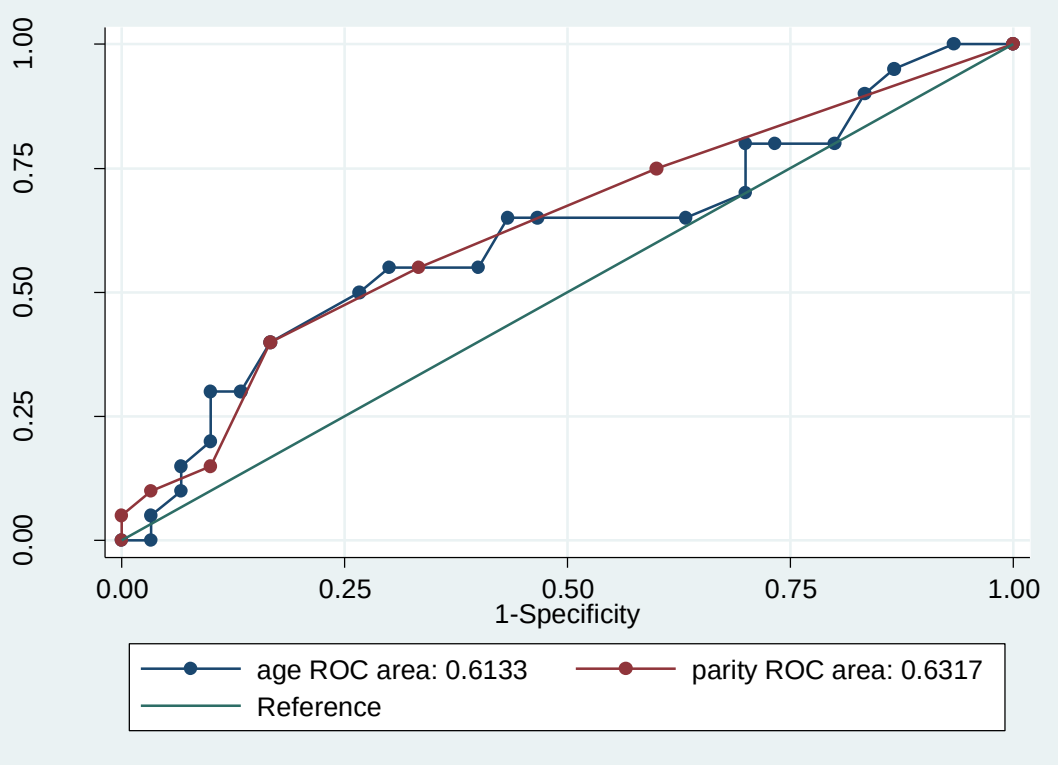

Figure 4.1: STATA Graphical ROC area of Mothers age and Parity

Figure 4.2 represents the receiver operating characteristics (ROC) area curves for mothers gravidity and gestational weight. In Figure 4.2 the vertical axis is the true positive rate (sensitivity) i.e. the proportion of actual positives that are correctly identified as such. The horizontal axis also indicates the false positive rate (specificity) i.e. the measure of proportion of actual negatives that are correctly identified as such. Figure 4.2 specifically indicates the ROC area values for gravidity (0.6333) and that of gestational weight (0.6633) as well as standard error and the confidence intervals. From the table, there is 0.6333 probability of correctly identifying the proportion of true positives (women with pregnancy induced hypertension) and 0.6633 chance of correctly identifying the proportion of the trues positives (women with pregnancy induced hypertension) taking into account their gravidity and gestational weight respectively.

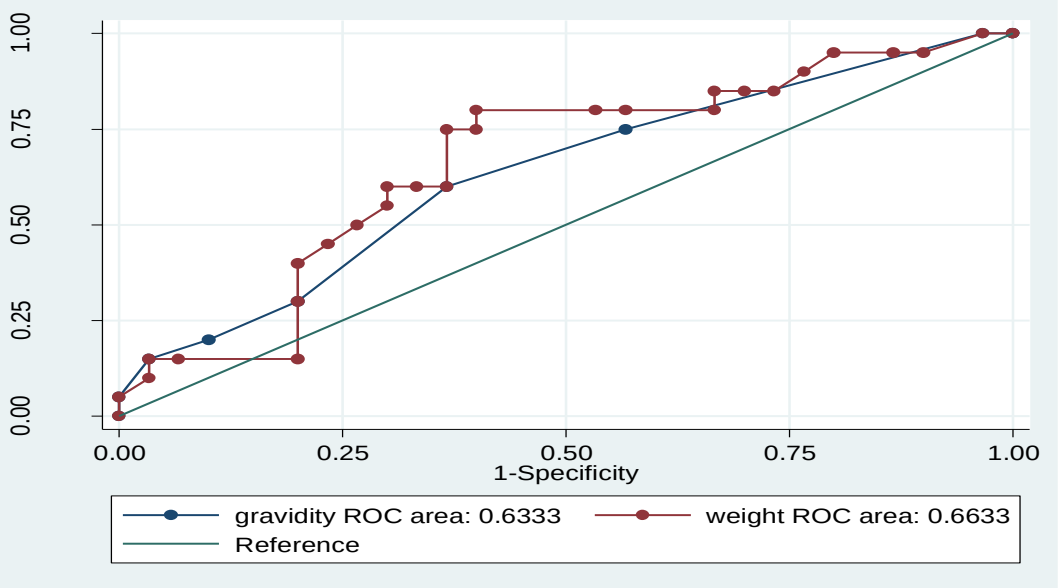

Figure 4.2 Graphical ROC area of gravidity and gestational weight

Figure 4.3 represents the receiver operating characteristics (ROC) area curves for glucose level and placenta length. In Figure 4.3, the vertical axis is the true positive rate (sensitivity) i.e. the proportion of actual positives that are correctly identified as such. The horizontal axis also indicates the false positive rate (specificity) i.e. the measure of proportion of actual negatives that are correctly identified as such. Figure 4.3 specifically indicates the ROC area values for glucose level (0.3758) and that of placenta length (0.475) as well as standard error and the confidence intervals. From the table, there is 0.3758 probability of correctly identifying the proportion of true positives (women with pregnancy induced hypertension) and 0.475 chance of correctly identifying the proportion of the trues positives (women with pregnancy induced hypertension) taking into account their glucose level and placenta length respectively.

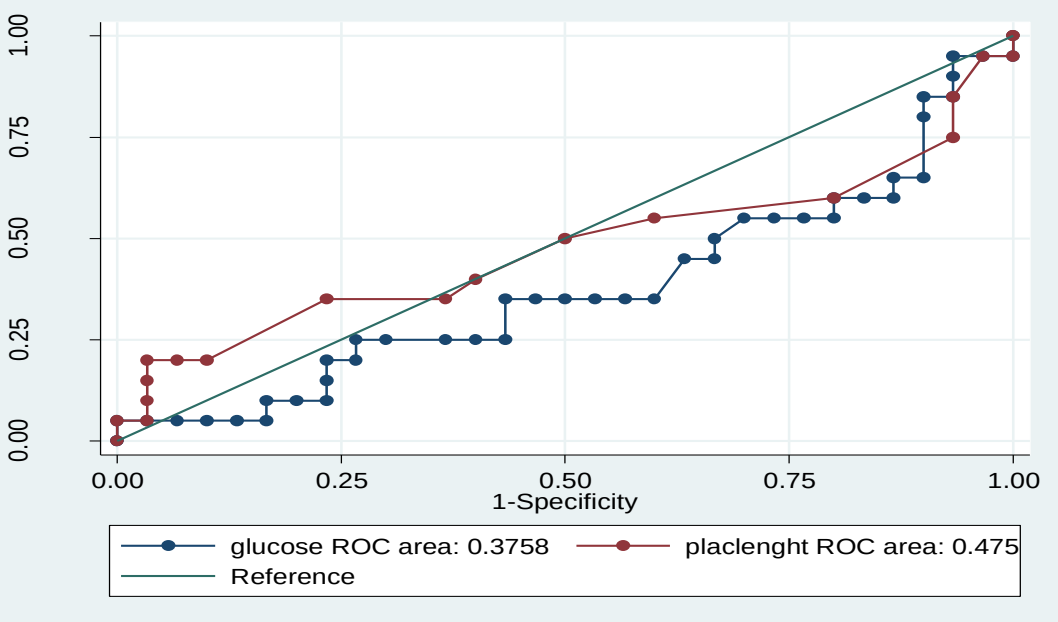

Figure 4.3: Graphical ROC area of glucose and placenta length

Figure 4.4 represents the receiver operating characteristics (ROC) area curves for placenta weight and umbilical cord length. In Figure 4.4 the vertical axis is the trues positive rate (sensitivity) i.e. the proportion of actual positives that are correctly identified as such. The horizontal axis also indicates the false positive rate (specificity) i.e. the measure of proportion of actual negatives that are correctly identified as such. Figure 4.4 specifically indicates the ROC area values for placenta weight (0.41) and that of umbilical cord length (0.5167) as well as standard error and the confidence intervals. From the table, there is 0.41 probability of correctly identifying the proportion of true positives (women with pregnancy induced hypertension) and 0.5167 chance of correctly identifying the proportion of the true positives (women with pregnancy induced hypertension) taking into account their placenta weight and umbilical cord length respectively.

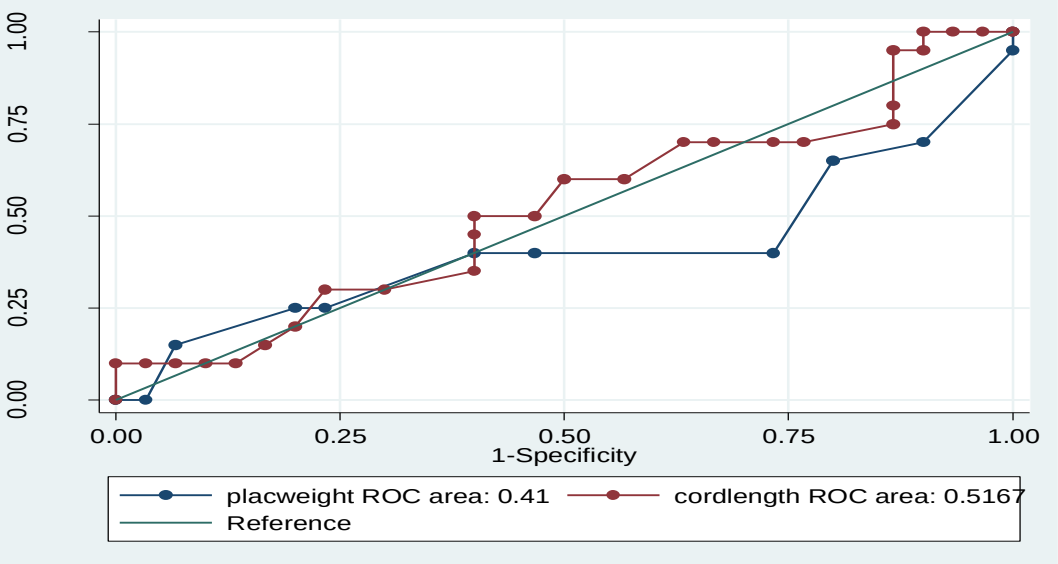

Figure 4.4: Graphical ROC area placenta weight and umbilical cord length

Figure 4.5 represents the receiver operating characteristics (ROC) area curves for umbilical cord diameter and cholesterol. In Figure 4.5, the vertical axis is the trues positive rate (sensitivity) i.e. the proportion of actual positives that are correctly identified as such. The horizontal axis also indicates the false positive rate (specificity) i.e. the measure of proportion of actual negatives that are correctly identified as such. Figure 4.5 specifically indicates the ROC area values for umbilical cord diameter (0.5033) and that of cholesterol (0.4667) as well as standard error and the confidence intervals. From the table, there is 0.5033 probability of correctly identifying the proportion of true positives (women with pregnancy induced hypertension) and 0.4667 chance of correctly identifying the proportion of the trues positives (women with pregnancy induced hypertension) taking into account their umbilical cord diameter and cholesterol level respectively.

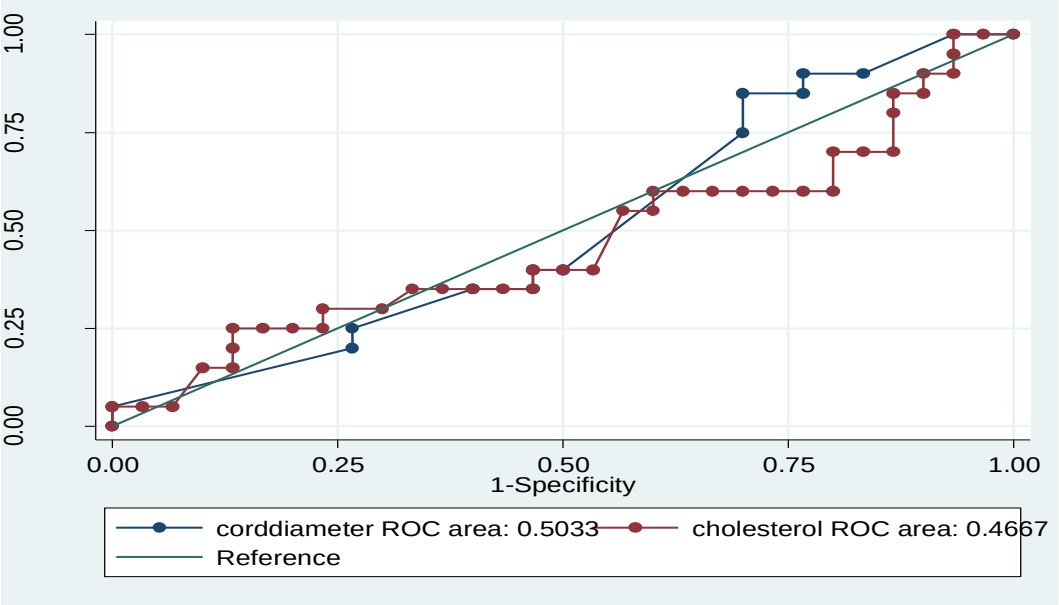

Figure 4.5: Graphical ROC area of umbilical cord diameter and cholesterol

Figure 4.6 represents the receiver operating characteristics (ROC) area curves for body mass index and high-density lipoprotein. In Figure 4.6, the vertical axis is the trues positive rate (sensitivity) i.e. the proportion of actual positives that are correctly identified as such. The horizontal axis also indicates the false positive rate (specificity) i.e. the measure of proportion of actual negatives that are correctly identified as such. Figure 4.6 specifically indicates the ROC area values for body mass index (0.6925) and that of high-density lipoprotein (0.4792) as well as standard error and the confidence intervals. From the table, there is 0.6925 probability of correctly identifying the proportion of true positives (women with pregnancy induced hypertension) and 0.4792 chance of correctly identifying the proportion of the trues positives (women with pregnancy induced hypertension) taking into account their body mass index and high-density lipoprotein respectively.

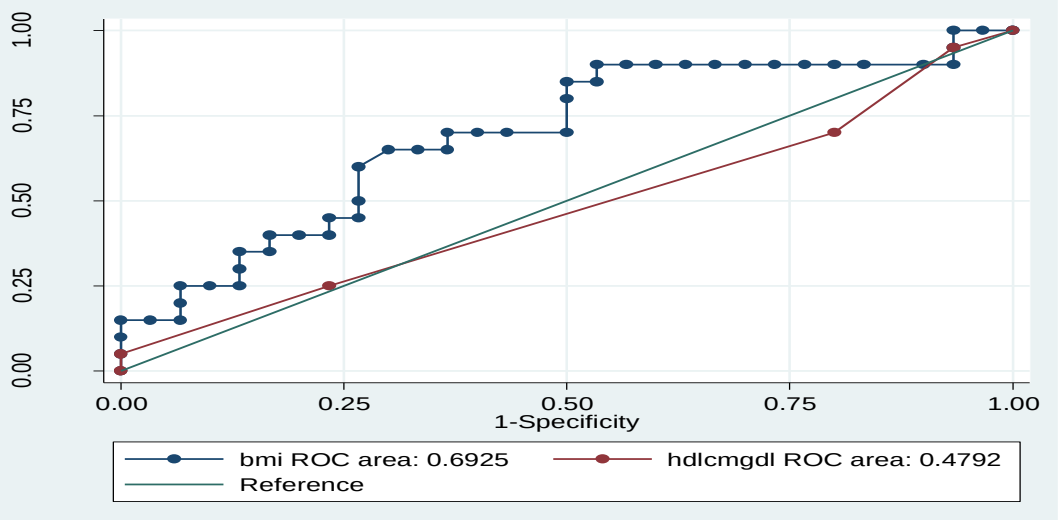

Figure 4.6: Graphical ROC area body mass index and high-density lipoprotein

Figure 4.7 represents the receiver operating characteristics (ROC) area curves for low-density lipoprotein and white blood cells. In Figure 4.7, the vertical axis is the true positive rate (sensitivity) i.e. the proportion of actual positives that are correctly identified as such. The horizontal axis also indicates the false positive rate (specificity) i.e. the measure of proportion of actual negatives that are correctly identified as such. Figure 4.7 specifically indicates the ROC area values for low-density lipoprotein (0.4992) and that of white blood cells (0.3367) as well as standard error and the confidence intervals. From the table, there is 0.4992 probability of correctly identifying the proportion of true positives (women with pregnancy induced hypertension) and 0.3367 chance of correctly identifying the proportion of the trues positives (women with pregnancy induced hypertension) taking into account mothers low-density lipoprotein cholesterol and white blood cells respectively.

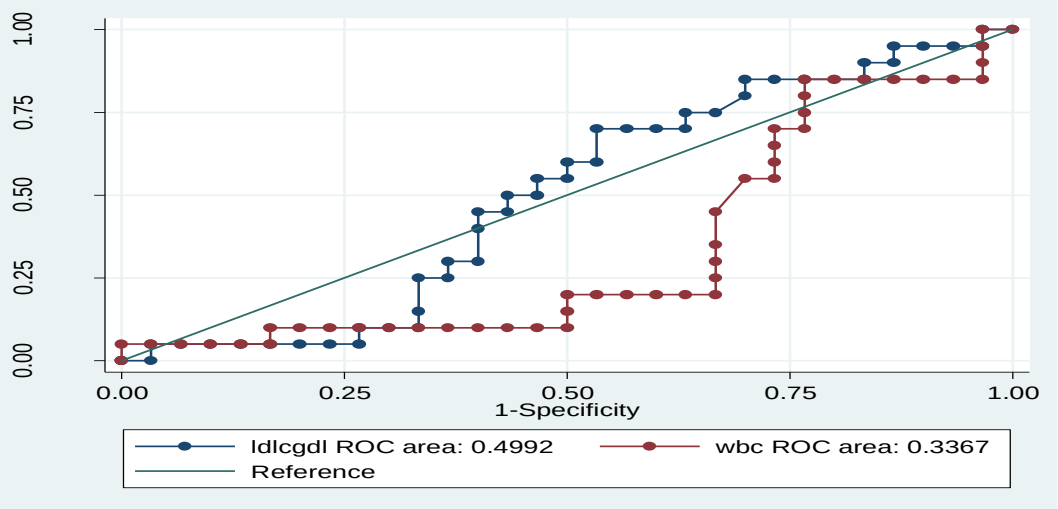

Figure 4.7: Graphical ROC area of low-density lipoprotein and white blood cells

Figure 4.8 represents the receiver operating characteristics (ROC) area curves for mothers red blood cells and haemoglobin. In figure 4.8, the vertical axis is the true positive rate (sensitivity) i.e. the proportion of actual positives that are correctly identified as such. The horizontal axis also indicates the false positive rate (specificity) i.e. the measure of proportion of actual negatives that are correctly identified as such. Figure 4.8 specifically indicates the ROC area values for mothers red blood cells (0.3775) and that of mothers haemoglobin (0.3533) as well as standard error and the confidence intervals. From the table, there is 0.3775 probability of correctly identifying the proportion of true positives (women with pregnancy induced hypertension) and 0.3533 chance of correctly identifying the proportion of the trues positives (women with pregnancy induced hypertension) taking into account their red blood cells and hemoglobin respectively.

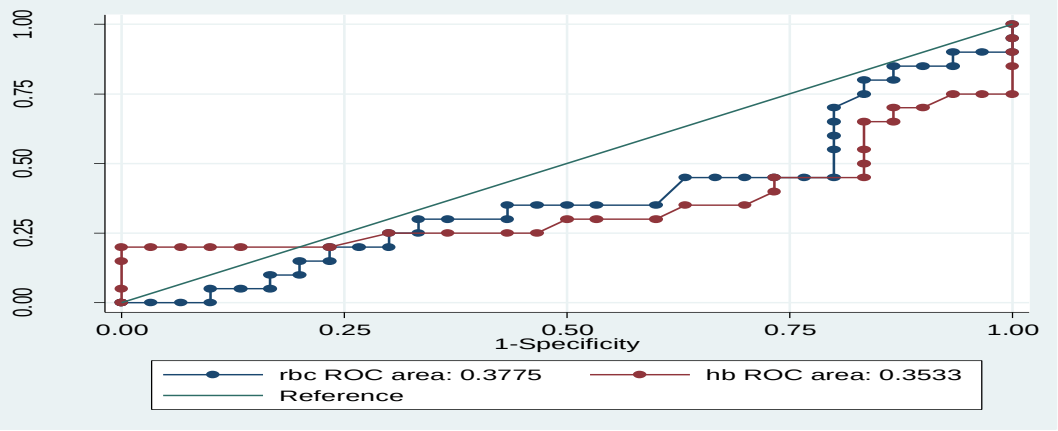

Figure 4.8: Graphical ROC area of red blood cells and hemoglobin

4.2 Parity Distribution

In Table 4.3, the distribution of the 50 women investigated at various parity level are expressed percentagewise. It is evident from the table that nulliparous women or women who have never given birth retain the bigger portion of the women with 34%. Women who gave birth once were the second highest representation with 24% of the experimented population. The results also indicated that parity 2 is composed 16% of the tested population while women with parity 3 constitutes 14% of the sample as well. However, multiparous women of parity 4, 5 and 6 were the least represented with 6%, 4% and 2% respectively.

Table 4.3: Percentage distribution of parity

Parity	Frequency	Percentage
0	17	34
1	12	24
2	8	16
3	7	14
4	3	6
5	2	4
6	1	2
Total	50	100

Figure 4.9 is a bar graph representing the distribution of parity in percentage terms and the frequency at each parity level.

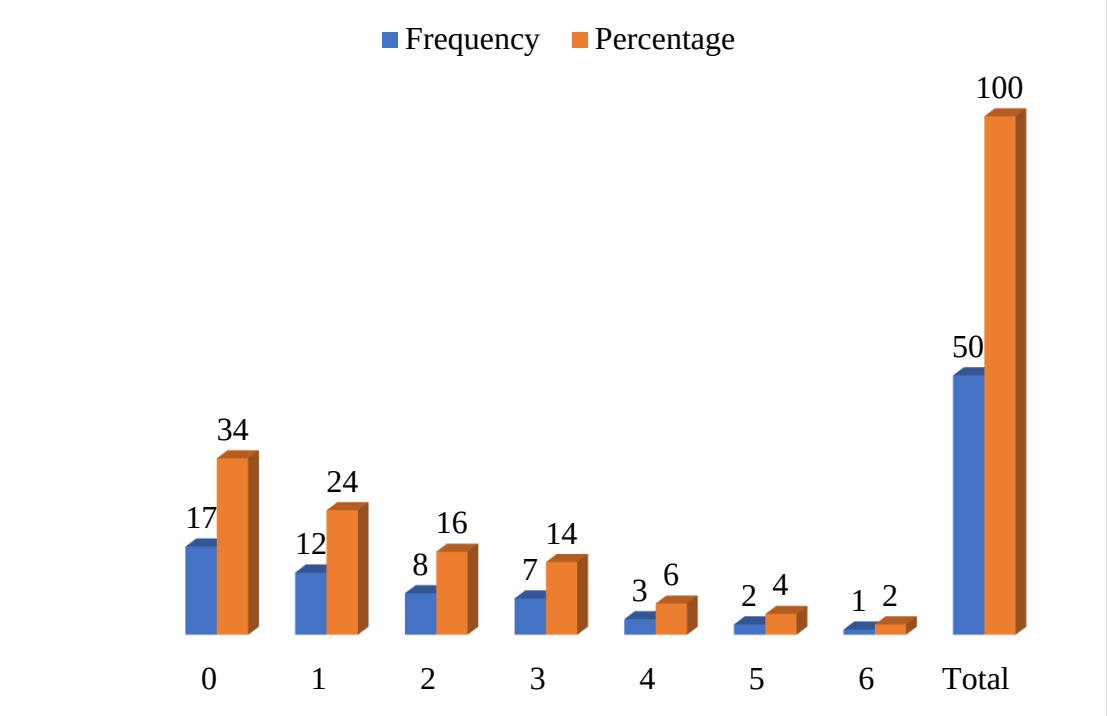

Figure 4.9: Bar graph of percentage distribution of parity

4.3 Parity to Blood Pressure Distribution

Table 4.4 shows the composition of the two levels of the blood pressure i.e. the normal blood pressure (Normotension) and the high blood pressure (pregnancy induced hypertension) in women during gestational period at various level of parity. It was revealed that out of 17 women who have never given birth (nulliparous), 12 were found to have normal blood pressure and 5 with high blood pressure.

The results also show that 8 women had normal blood pressure and 4 had high blood pressure amongst women who had given birth twice (parity 2). However, for women who had already given birth trice (parity 3), 5 were found to have had their blood pressures within the normal range and 2 had their blood pressures above the normal range.

The study also revealed that multiparous women of para 4, 5 and 6 have the smallest representation in terms of the number of pregnant women who tested positive for hypertension. Para 4, 5, and 6 each had only 1 pregnant woman with hypertension.

Table 4.4: Distribution of parity level to blood pressure status

	Parity							
Group	0	1	2	3	4	5	6	Total
Normotension	12	8	5	2	2	1	0	30
PIH	5	4	3	5	1	1	1	20
Total	17	12	8	7	3	2	1	50

Figure 4.10 is bar graph of the distribution of parity levels to blood pressure. The graph indicates the number of women with high blood pressure during gestational period and as well shows the number of women who have normal blood pressure.

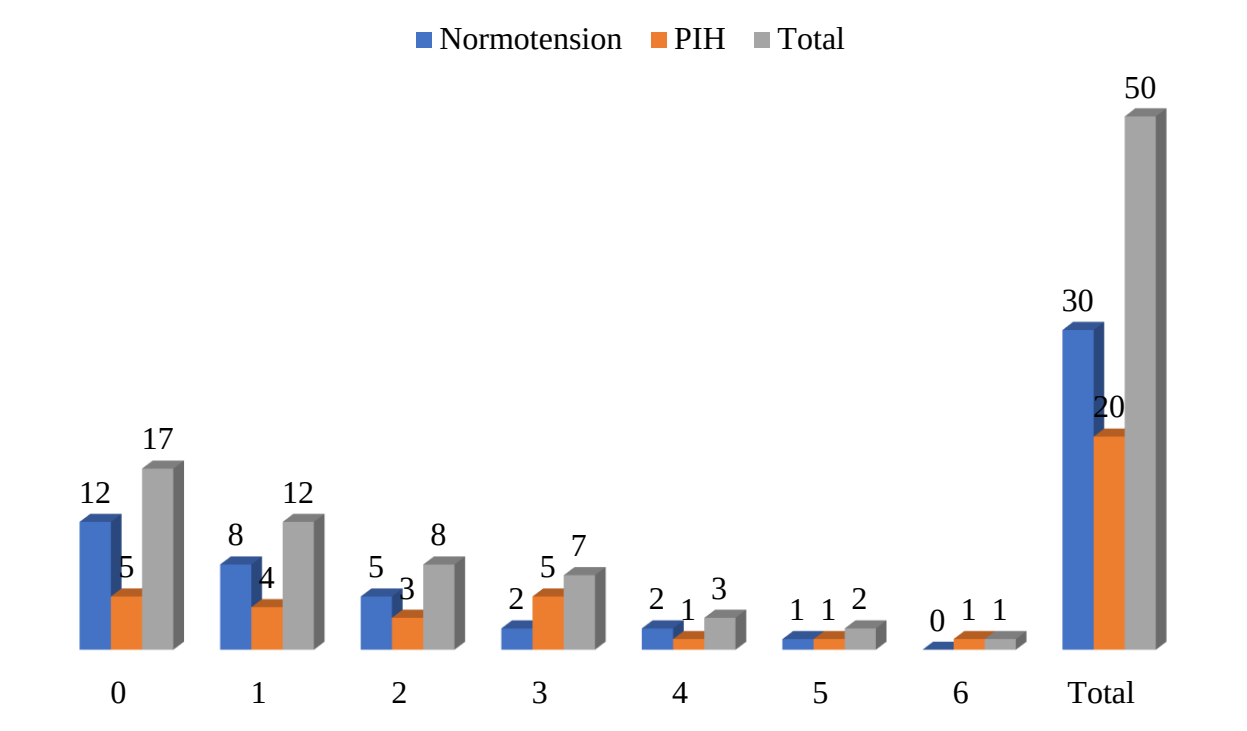

Figure 4.10: Bar chart of the distribution of parity to blood pressure level

4.4 Gravidity Distribution

Table 4.5 show the distribution of the 50 women being investigated at various gravida level expressed as percentage. From the results, women who have never gotten pregnant before or women experiencing their first pregnancy (gravida 0) as well as women of gravida 7 make up only two 2% of the sampled population. However, women of gravida 1 or women who had experienced pregnancy only once constituted larger portion of the population with 17 women representing 34%.

Women of gravida 2, 3, and 4 make up 18%, 22% and 10% of the sampled population respectively. The study shows that women of gravida 5 and 6 represent 6% each of the 50 women sampled.

Table 4.5: Percentage distribution of Gravida

Gravidity	Frequency	Percentage
0	1	2
1	17	34
2	9	18
3	11	22
4	5	10
5	3	6
6	3	6
7	1	2
Total	50	100

Figure 4.11 is a bar graph representing the distribution of gravidity in percentage terms and the frequency of each gravida level.

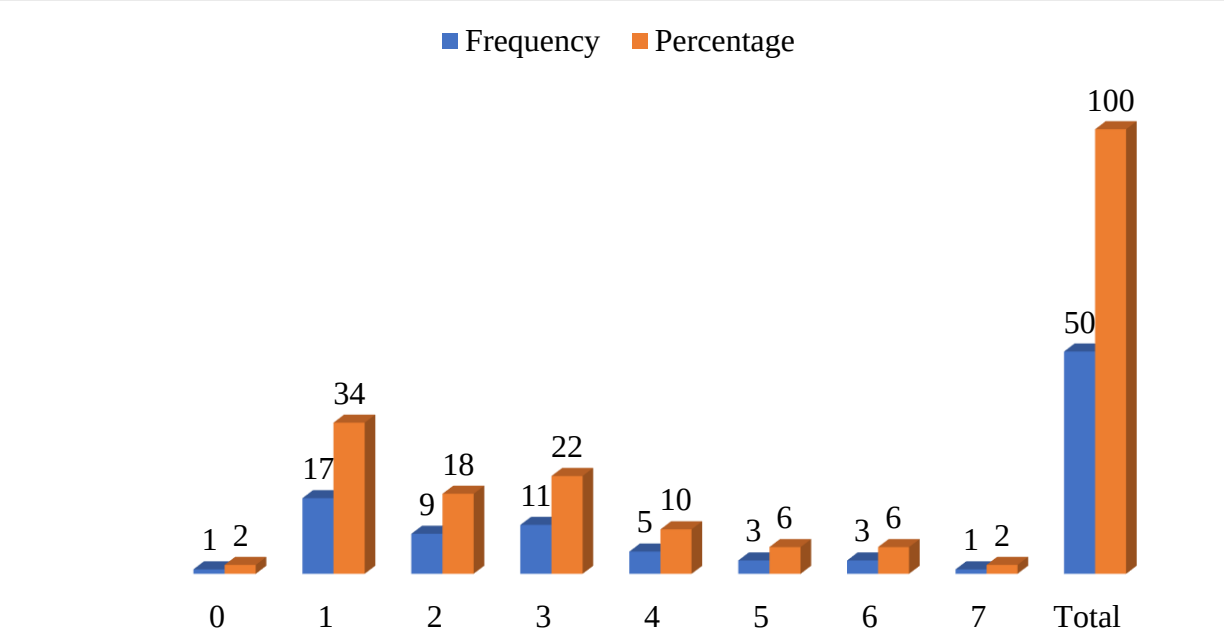

Figure 4.11: Bar graph of percentage distribution of gravidity

4.5 Gravidity to Blood Pressure Distribution

The table below summarizes the distribution of gravida levels of the 50 sampled women to their blood pressure status during the gestational period. Women who are experiencing their first pregnancy or who have never been pregnant before (gravida 0) constituted only 1 person with a normal blood pressure.

However, out of 17 women who ever experienced pregnancy just once (gravida 1), 5 of them had high blood pressure and 12 had normal blood pressure. The research also discovered that women of gravida 2 had 6 people with normal blood pressure whiles 3 had their blood pressures above the normal range.

Nonetheless, 6 pregnant women were found to be hypertensive and 5 appeared normotensive from the gravida 3 group. Women with gravida 4, and 6 had 2 people each to be hypertensive whereas 3 and 2 women respectively had normal blood pressure. Similarly, 1 woman each from gravida 5 and 7 had high blood pressure but then, gravida 5 had 2 women within the normal blood pressure range and none from gravida 7 had normal blood pressure.

Table 4.6: Distribution of gravida level to blood pressure status

	Gravidity								
Group	0	1	2	3	4	5	6	7	Total
Normotension	1	12	6	5	3	2	1	0	30
PIH	0	5	3	6	2	1	2	1	20
Total	1	17	9	11	5	3	3	1	50

Figure 4.12 is bar graph of the distribution of gravida levels to blood pressure. The graph indicates the number of women with high blood pressure during gestational period and as well shows the number of women who have normal blood pressure.

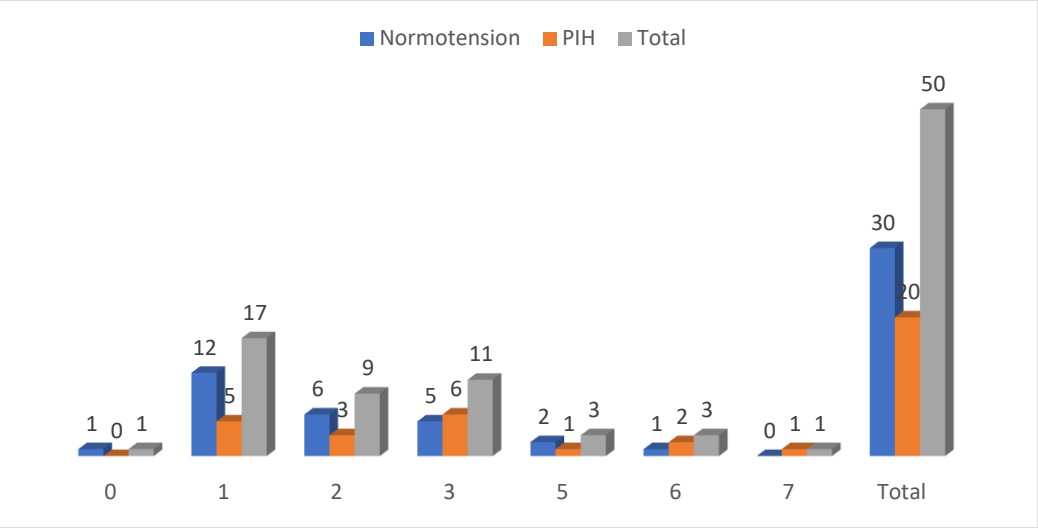

Figure 4.12: Graph of distribution of gravidity to blood pressure level

4.6 Model Analysis

4.6.1 Introduction

This section of the data analysis was conducted to examine the efficacy of various models in terms of their probability values and other statistical measures relevant to the study. The models adopted were three which include the Logistic regression model, Probit regression model and Artificial Neural Network because of their ability to analyze binary dependent variables.

4.6.2 Logistic Regression Model

Table 4.7 shows the results for fifty (50) pregnant women who were sampled for the study from logistic regression model applied using the statistical analysis software (STATA version 12) to analyze the data with probability-value pinned at 0.05 significance.

There was no statistically significant difference between pregnancy induced hypertension and mothers age with a probability value of 0.576. On the contrary, several other studies indicated that there was a positive correlation between advance maternal age and pregnancy induced hypertension (Cleary-Goldman, 2005; Jacobsson, 2004, Jolly *et al*, 2000). Similarly, there was no statistically significant difference between pregnancy induced hypertension and parity level (probability = 0.655); as well as between pregnancy induced hypertension and gravidity level (probability = 0.965). Luo *et al.*, (2007), and Audrey *et al.*, (2003), confirmed this in a research that, increasing number of births (multiparity) lowers the risk of a mother to gestational hypertension just as a lower risk of PIH when she has been through more than one pregnancy (multigravida) (Duckitt *et al.*, 2005).

Again, there was no statistically significant difference between pregnancy induced hypertension and placenta weight (p = 0.085). However, there was a certain trend towards significance between pregnancy induced hypertension and gestational weight (probability = 0.079). But then, a research conducted by Heude *et al.*, (2012), and Visnawathan *et al.*, (2008) indicated that women who gained much weight in their gestational period are at a higher risk of preeclampsia and women who are obese are also at a higher risk of hypertension in pregnancy (Kaiser *et al*, 2001; Shao *et al*, 2006).

Likewise, not much of significance could be said between induced hypertension and glucose level (probability = 0.569). Conversely to our revelation, it has also been articulated in a research steered by Valdes *et al.*, (2014) and Hauth *et al.*, (2011) that many risk factors of preeclampsia are connected to insulin resistance

and thus glucose levels have impact of PIH. Placenta length (probability = 0.565) was not statistically significant; placenta weight (probability = 0.085) was also nearing statistical significance.

The results also revealed that umbilical cord length (probability = 0.126) and umbilical cord diameter (probability = 0.708) did not have any statistically significant difference between them and pregnancy induced hypertension. Equally, there was no statistically significant difference between pregnancy induced hypertension and cholesterol level (probability = 0.423) but however, writers have realized that women with preeclampsia have higher triglycerides (cholesterol) than those with normal blood pressure (Gallos *et al.*, 2013).

However, the results revealed that body mass index (probability = 0.0254) was statically significant which has been attested to in a study by Cnattingius *et al.*, (1998) and Bhattacharya *et al.*, (2007) which suggested that obesity is a greater risk factor of preeclampsia. Low-density cholesterol (probability = 0.027) and white blood cells (probability = 0.01) indicated statistically significant difference between them and pregnancy induced hypertension. Other investigations confirmed that changes in the morphology of the leukocytes (white blood cells) also impact preeclampsia (Hernandez *et al.*, 2008) and it was as well discovered in an investigation that low-density cholesterol impacts preeclampsia (Iftilkhar *et al.*, 2005; Uzun *et al.*, 2005).

The study did reveal that there was no statistically significant difference between pregnancy induced hypertension and high-density cholesterol (probability = 0.534). This is affirmed in a study which stipulates that decreased high-density lipoprotein

cholesterol (HDL-C) concentrations may relate to an increased risk of preeclampsia, (Iftilkhar *et al.,* 2005; Uzun *et al.,* 2005). Red blood cell (probability = 0.105) was not statistically significant as well as haemoglobin level (probability = 0.432) as far as PIH is concern. This contradicts Yip *et al.,* (2000) which acknowledged in a study that women with preeclampsia have higher concentration of haemoglobin and another research that suggested that RDW is an indicator of red cell size variation called anisocytosis was higher in slightly elevated blood pressure which would likely turn to high blood pressure (Tanindi *et al.,* 2011).

The results also revealed, with one more additional year, a woman has 0.94 odds of conceivably pregnancy induced hypertension (PIH). A woman also has 1.75 odds of possibly pregnancy induced hypertension with one more birth just as she has an odd of 0.95 of perhaps PIH with an extra pregnancy.

Again, an extra kilogram increase in gestational weight probably has 0.81 odds of gestational hypertension and so does a woman have an odd of 0.96 of perchance PIH with a unit increase in milligram per deciliter of her blood sugar level (glucose). Nevertheless, with an extra centimeter stretch in placenta length, a woman has 0.94 odds possibility of becoming gestationally hypertensive.

More so, with additional grams in placenta weight, a woman has 0.99 odds of conceivably pregnancy induced hypertension (PIH) and a woman also has 1.1 odds of possibly pregnancy induced hypertension with a unit centimeter increase in umbilical cord diameter. Yet again, there were odds of 1.06, 0.6, 0.64, and 0.70 feasibly PIH with milligram per deciliter unit increase in cholesterol level, HDL-C, LDL-C and hemoglobin respectively. An added kilogram per meter square increase

in body mass index (BMI), there was probably 2.20 odds of PIH. There were also odds of 0.72 and 0.11 possibly pregnancy induced hypertension with a unit increase in cubic millimeter of WBC and RBC respectively.

Table 4.7: Logistic regression model analysis

Variables	Odds Ratio	Std. Err	Z-value	Probability
MA (yrs.)	0.9434523	0.0981971	-0.56	0.576
Parity	1.744633	2.171842	0.45	0.655
Gravidity	0.9520233	1.056554	-0.04	0.965
GW (kg)	0.8151048	0.0950128	-1.75	0.079
Glucose (mg/dL)	0.9634118	0.0629799	-0.57	0.569
PL (cm)	0.9430547	0.0961951	-0.57	0.565
PW(g)	0.9935519	0.0037293	-1.72	0.085
UCL (cm)	1.096422	0.0659842	1.53	0.126
UCD (cm)	0.6959067	0.6730098	-0.37	0.708
Cholesterol (mg/dL)	1.061241	0.0786462	0.8	0.423
BMI (kg/mm2)	2.208173	0.7805745	2.24	0.025*
HDL- C(mg/dL)	0.6083758	0.4633883	-0.65	0.514
LDL-C(mg/dL)	0.6449999	0.1277503	-2.21	0.027*
WBC (mm3)	0.7194296	0.0915285	-2.59	0.01*
RBC (mm3)	0.1095684	0.1492555	-1.62	0.105
HB (gm/dL)	0.6995863	0.3178984	-0.79	0.432

4.6.3. Area Under ROC Curve for Logistic Regression Model

Figure 4.13 shows the probability of accurately predicting the true positive (sensitivity) and negative rate (specificity). From the results below, the model revealed that there is 0.905 chance of predicting accurately the true positive rate of a woman having gestational hypertension and the same chance of 0.905 predicting accurately true negative rate of a woman with normal tension.

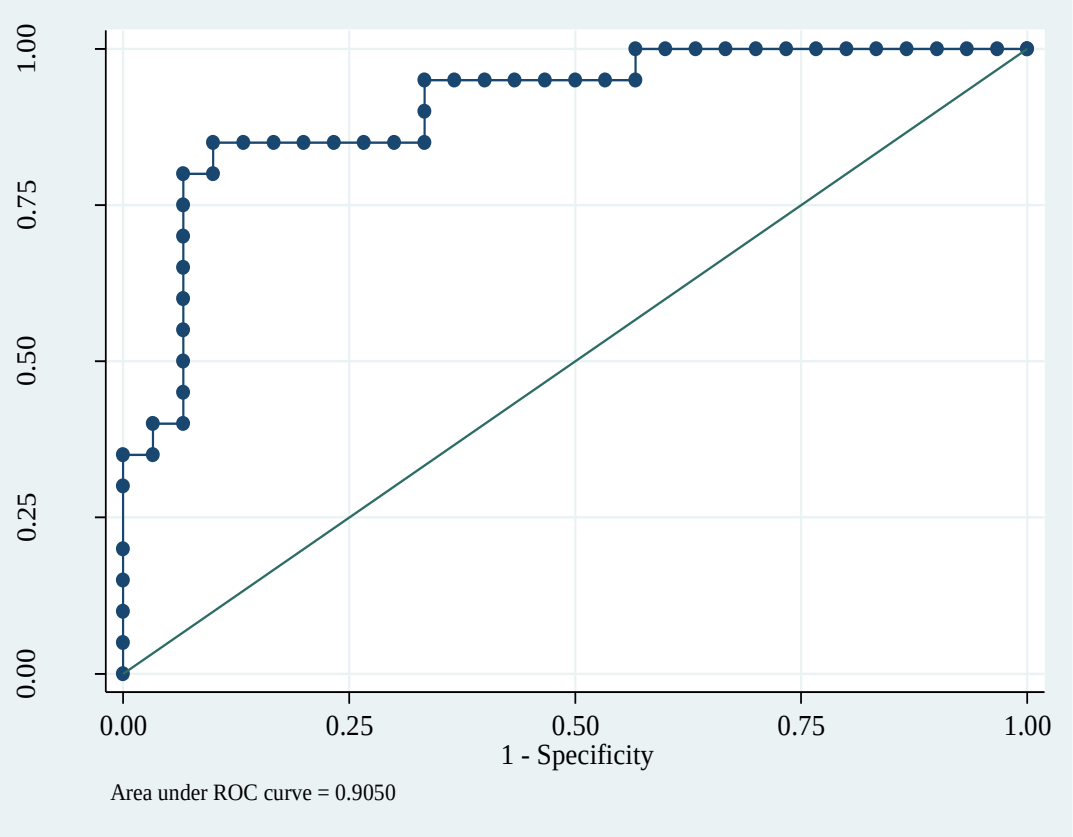

Figure 4.13: Area under ROC curve for logistics regression

4.7 Probit Regression Model

Table 4.8 shows the results attained from the probit regression model of 50 pregnant women selected for the study. It could be seen from the results that there was no

statistically significant difference between pregnancy induced hypertension and mothers age (probability = 0.56) as well as between parity (probability =0.66) and pregnancy induced hypertension. Divergently, several studies indicated that there was a positive relation between advance maternal age and pregnancy induced hypertension (Cleary-Goldman, 2005; Jacobsson, 2004, Jolly *et al.*, 2000) and also, it was revealed in a research that increasing level of parity lowers the risk of PE (Luo *et al.*, 2007, and Audrey *et al.*, 2003) as it is in our case.

Not much of any statistically significant difference could been seen between gravidity (probability = 0.97) and pregnancy induced hypertension. On the other hand, gestational weight (probability = 0.03) was statistically significant whiles placenta weight (probability = 0.07) exhibited trend towards statistically significant difference between with gestational hypertension. Opposingly, research conducted by Heude *et al.,* (2012), and Visnawathan *et al.,* (2008) indicated that women who gained much weight in their gestational period are at a higher risk of preeclampsia and women who are obese are also at a higher risk of hypertension in pregnancy (Kaiser *et al.,* 2001; Shao *et al.*, 2006).

The glucose level (probability = 0.58) of mothers did not show statistically significant different with gestational hypertension and neither was there any statistically significant difference between gestational hypertension and placenta length (probabolity = 0.56). It has also been articulated in a research steered by Valdes *et al.*, (2014) and Hauth *et al.*, (2011) that many risk factors of preeclampsia are connected to insulin resistance and thus glucose levels have impact of PIH.

There was equally no statistically significant difference between (umbilical cord length (UCL), probability = 0.09; umbilical cord diameter (UCD), probability = 0.71; cholesterol level, probability = 0.42) and pregnancy induced hypertension. However, there were significant difference between gestational hypertension and body mass index with probability = 0.006*, as well as low density cholesterol (probability = 0.01*). A study by Cnattingius *et al.*, (1998) and Bhattacharya *et al*., (2007) confirmed that obesity and for that matter higher BMI is a greater risk factor of preeclampsia. Again, it was as well discovered in an investigation that low-density cholesterol impacts preeclampsia (Iftilkhar *et al.*, 2005; Uzun *et al.*, 2005).

Apparently, the results also showed significant difference between white blood cells (probability = 0.01*) and pregnancy induced hypertension although there was a trend toward significance between red blood cells (probability = 0.1) and gestational hypertension. An investigation confirmed that changes in the morphology of the leukocytes (white blood cells) also impact preeclampsia (Hernandez *et al.*, 2008). Contrary to our finding on the RBC, an indicator of red cell size variation called anisocytosis was higher in slightly elevated blood pressure which would likely turn to high blood pressure (Tanindi *et al.*, 2011).

In conclusion, there were no significant difference between high density cholesterol (HDL-C), probability = 0.5; hemoglobin (HB), (probability = 0.43) and pregnancy induced hypertension. Yip *et al.*, (2000), rather approved in a study that women with preeclampsia have higher concentration of haemoglobin. Iftilkhar *et al.*, (2005); Uzun *et al.*, (2005) confirmed that decreased high-density lipoprotein

cholesterol (HDL-C) concentrations may relate to an increased risk of preeclampsia.

That notwithstanding, the results as well indicated that, an additional year to the age of a mother will reduce her chance of pregnancy induced hypertension slightly by 0.70 percentage points. Again, one more birth and a unit increase in umbilical cord length with increase the chance of gestational hypertension by 7.00 and 1.00 percent respectively.

Similarly, it was realized that One (1) more pregnancy will reduce the chance of PIH by a percentage point of 0.6 likewise a kilogram change increase in gestational weight; a unit increase in milligram per deciliter of glucose level; 1-centimeter increase in placenta length; a unit gram increase in placenta weight will reduce the chances of pregnancy induced hypertension by (2.6%, 0.5%, 0.7%, and 0.00%) respectively.

Though a unit kilogram per millimeter square increase in body mass index will increase the chance of PIH by 10.00 percent and an extra increase in cholesterol level will also increase the chance of hypertension in pregnancy by 42%, but then a unit increase in the quantity of the remaining variables such as UCD, HDL, WBC, RBC and HB with reduce mother's chance of gestational hypertension by 4.6, 6.3, 4.2, 28, and 4.6 percentage points respectively.

Table 4.8: Probit regression model analysis

	Delta-method			
Variable	**Margin**	**Std. Err.**	**Z**	**P- value**
MA (yrs.)	-0.0074155	0.0127285	-0.58	0.56
Parity	0.0708999	0.1624424	0.44	0.663
Gravidity	-0.0062634	0.1417537	-0.04	0.965
GW (kg)	-0.0260441	0.0117101	-2.22	0.026*
Glucose (mg/dL)	-0.0047485	0.008522	-0.56	0.577
PL (cm)	-0.0074692	0.0128416	-0.58	0.561
PW(g)	-0.0008241	0.0004542	-1.81	0.07
UCL (cm)	0.0117268	0.0068657	1.71	0.088
UCD (cm)	-0.0461851	0.1244164	-0.37	0.71
Cholesterol (mg/dL)	0.0075721	0.0093637	0.81	0.419
BMI (kg/mm2)	0.1009164	0.0368764	2.74	0.006*
HDL- C(mg/dL)	-0.0633096	0.0912281	-0.69	0.488
LDL-C(mg/dL)	-0.0558625	0.0225175	-2.48	0.013*
WBC (mm3)	-0.0419501	0.0163143	-2.57	0.01*
RBC (mm3)	-0.2816924	0.1773342	-1.59	0.112
HB (gm/dL)	-0.0455132	0.0586217	-0.78	0.438

4.8 Area Under ROC Curve for Logistic Regression Model

Figure 4.14 shows the probability of accurately predicting the true positive (sensitivity) and negative rate (specificity). From the results below, the model

revealed that there is 0.901 chance of predicting accurately the true positive rate of a woman having gestational hypertension and the same chance of 0.901 predicting accurately true negative rate of a woman with normal tension.

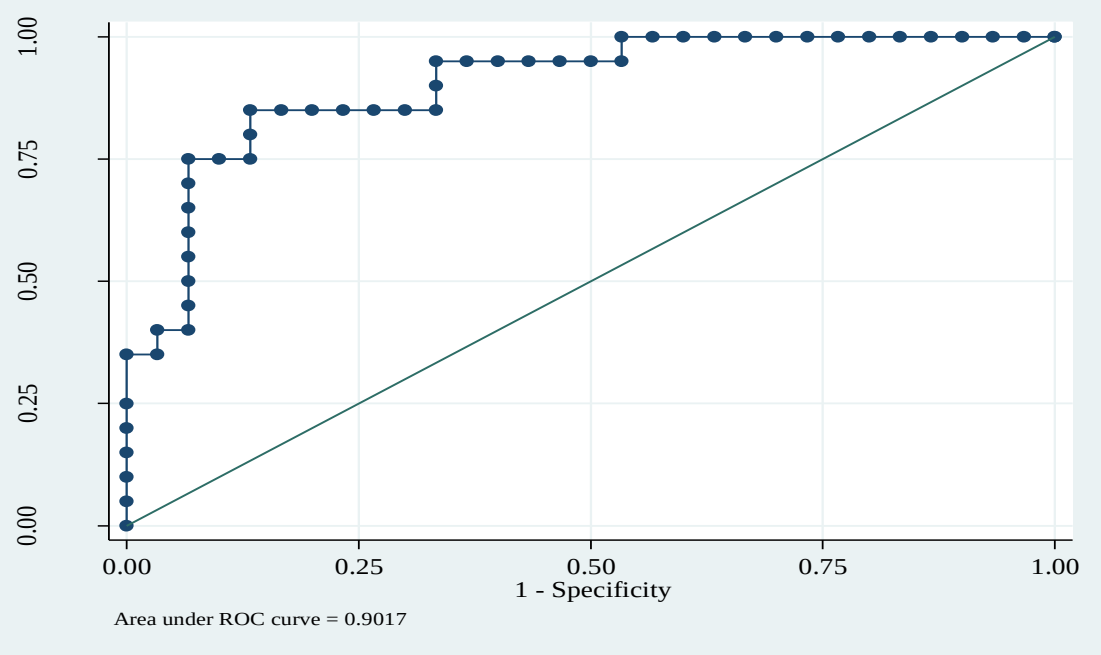

Figure 4.14: Area under ROC curve of Probit model

4.8 Artificial Neural Network Model

4.8.0 Introduction

Table 4.9 displays the output results attained from neural network model of 50 sampled women and sixteen (16) different maternal indices. The sigmoid activation function was used for both input and output layers. Was considered due to its ability to force a neural network to sum to 1 so that they can represent a probability distribution across discrete mutual exclusive alternatives.

4.8.1 Case Processing

The case processing summary table shows that, 38 cases were assigned to the training sample, 3 cases were also assigned to the testing sample and 3 cases were assigned to the holdout sample. Forty-four valid cases were included in the analysis whiles 6 cases were excluded due to various reason identified during the data processing.

Table 4.9: Case Processing Summary

Sample	N	Percent
Training	38	86.40%
Testing	3	6.80%
Holdout	3	6.80%
Valid	44	100.00%
Excluded	6	
Total	50	

4.8.2 Independent Variable Importance

The independent variable importance unlike the probability value does not compare to any value(s) in making inference. The normalized importance values are derived from the ratio of importance values of the variables to the variable with the smallest value expressed as a percentage. Hence, the percentages indicate the level of influence the model places a variable on the dependent variable.

Table 4.10 revealed that parity (74%), gravidity (63%), haemoglobin (100%), RBC (68%), and WBC (64%) have greater impact on pregnancy induced hypertension than the rest of the variables. However, this can not be completely through as there are several researches which proved otherwise of some of the variables. Advanced maternal age (24.6%), glucose level, BMI (33.3%), cholesterol level (32.2%) and gestational weight (23.9%) though appeared to have the least impact, but other

studies insinuate that these are well known, and established risk factors of pregnancy induced hypertension. A study by Cnattingius *et al*., (1998) and Bhattacharya *et al*., (2007) confirmed that obesity and for that matter higher BMI is a greater risk factor of preeclampsia. Again, it was as well discovered in an investigation that low-density cholesterol impacts preeclampsia (Iftilkhar *et al*., 2005; Uzun *et al*., 2005). Better still, several other studies indicated that there was a positive corelation between advance maternal age and pregnancy induced hypertension (Cleary-Goldman, 2005; Jacobsson, 2004, Jolly *et al*, 2000).

Table 4.10: Independent Variable Importance

Variable	Importance	Normalized Importance
MA (yrs.)	0.040	24.6%
Parity	0.120	74.3%
Gravidity	0.103	63.7%
GW (kg)	0.039	23.9%
Glucose (mg/dL)	0.052	32.2%
PL (cm)	0.043	26.7%
PW(g)	0.026	16.3%
UCL (cm)	0.034	20.8%
UCD (cm)	0.011	7.1%
Cholesterol (mg/dL)	0.025	15.2%
BMI (kg/mm2)	0.054	33.3%
HDL- C(mg/dL)	0.027	16.9%
HDL-C(mg/dL)	0.048	29.9%
WBC (mm3)	0.104	64.5%
RBC (mm3)	0.111	68.8%
HB (gm/dL)	0.162	100.0%

Figure 4.15 is a bar chart of the variable importance usually referred to as the Importance Chart in neural network is simply a bar chart of values of the importance table sorted in descending values of impact rated by the model. The bar chart clearly indicates the model rates hemoglobin (HB) to have higher impact on

PIH than the rest of the variables umbilical cord diameter has the lowest impact on PIH according to the ANN model by means of the sigmoid activation function.

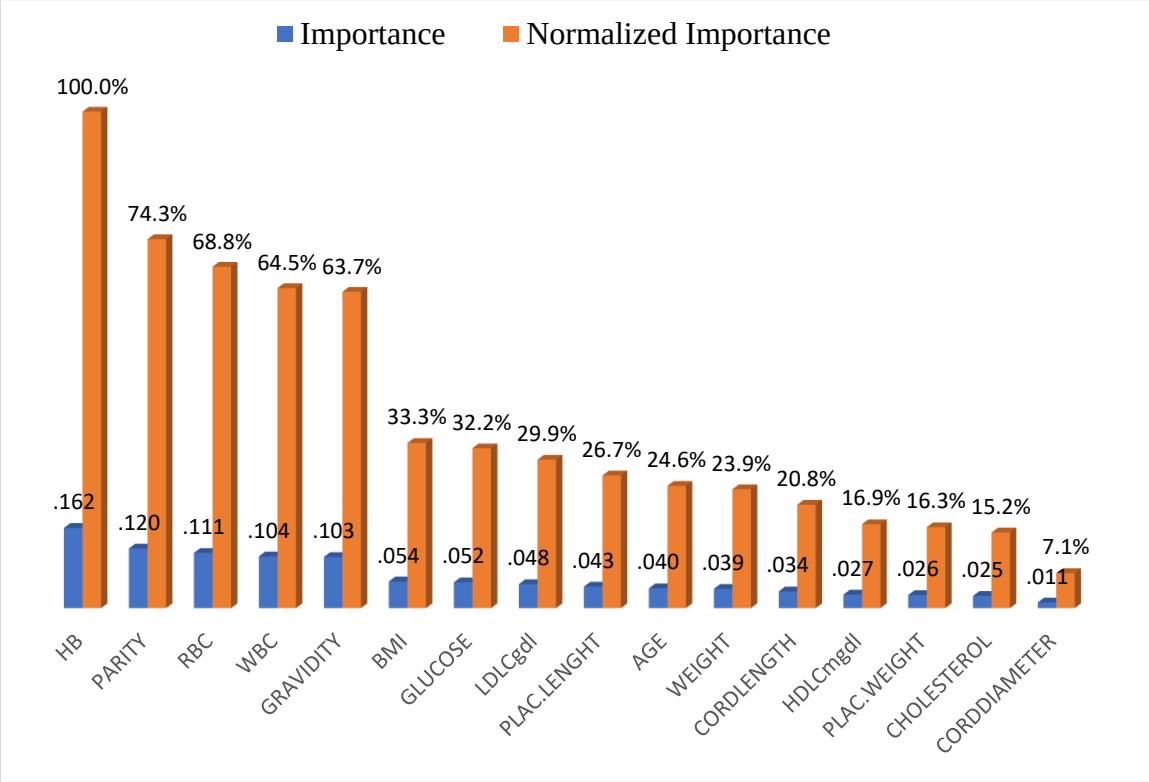

Figure 4.15: Bar graph of importance

4.9 Area Under ROC Curve for ANN Model

Table 4.11 shows the probability of accurately predicting the true positive (sensitivity) and negative rate (specificity). From the results below, the model revealed that there is 0.897 chance of predicting accurately the true positive rate of a woman having gestational hypertension and the same chance of 0.897 predicting accurately true negative rate of a woman with normal blood pressure.

Table 4.11: AUC for Artificial Neural Network

Area Under the Curve	
Group	**Area**
0	0.897
1	0.897

4.10 Structure of Activation Function

Figure 4.16 is the structure of the sigmoid activation function indicating all sixteen (16) independent variables which constitutes the input layer; the bias term making up the hidden layer: and the output layers which contains the probability of either malignant (pregnancy induced hypertension) or the benign (normal blood pressure).

In this case as indicated in the methodology, the pregnancy induced hypertension (PIH) is coded one (1) and the normal blood pressure (normotension) is coded zero (0).

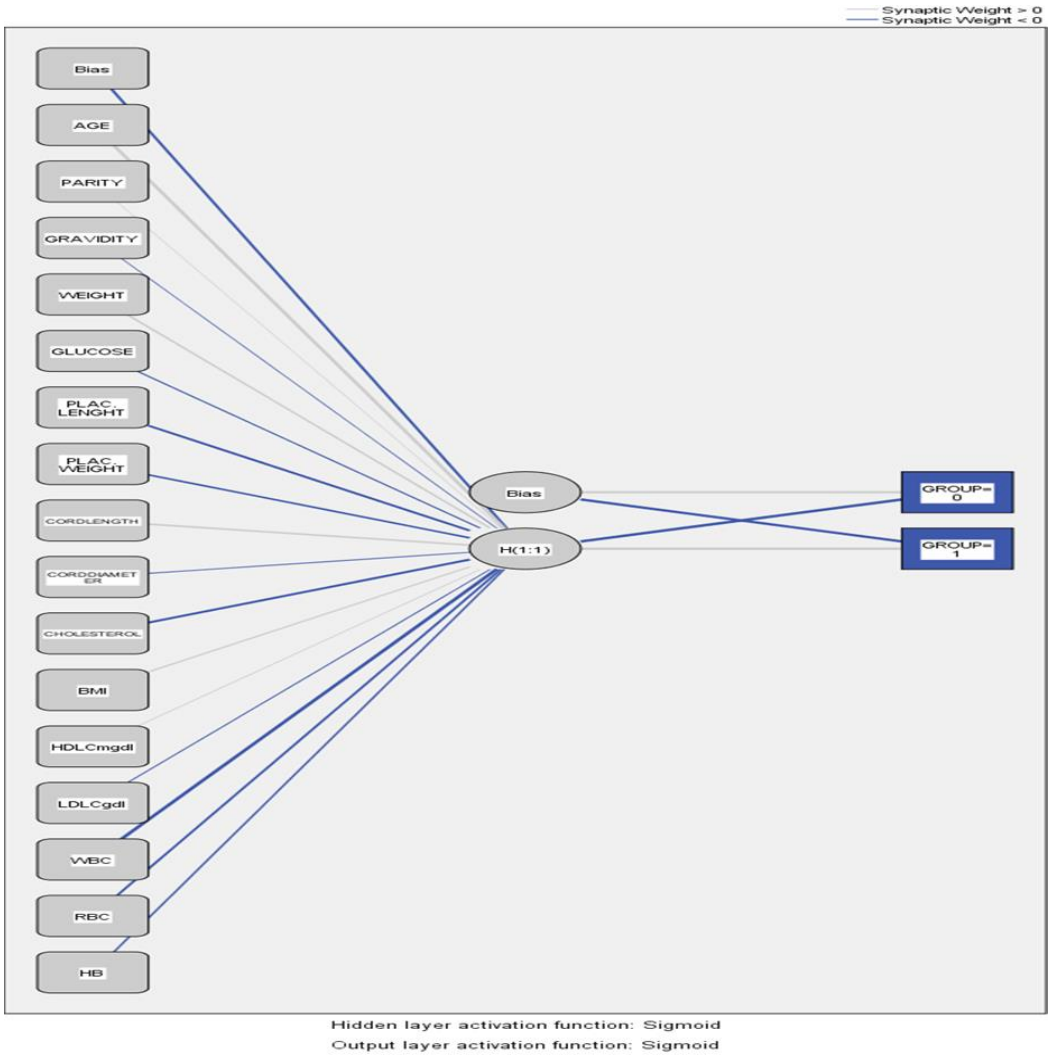

Figure 4.16: STATA 12 software graphical output for Layers of the neural network using sigmoid activation function

4.11 Cumulative Gain Chart

Figure 4.17 is the cumulative gains chart gives a photographic assistance in determining model performance. Gain and Lift charts are used to evaluate performance of classification model. It measures how much better one can expect to do with the predictive model comparing without a model. The figure in question shows the percentage of the overall number of cases in a given group "gained" by aiming a percentage of the total number of cases.

From our cumulative gain chart, the first point on the curve for pregnancy induced hypertension (PIH) is at (10%, 25%), which means that if you score a dataset with the network and sort all of the cases by predicted pseudo-probability of PIH, the top 10% will contain approximately 25% of all of the cases that actually take the group PIH. Similarly, the top 20% contains approximately 45% of women with PIH, the top 30% contains roughly 75% of all the cases of pregnancy induced hypertension.

Meanwhile, the cumulative chart also indicated that the top 40% contains approximately 85% of all the cases that actually take the class PIH if the data were scored by the network and all cases sorted by predicted pseudo-probability of PIH. In the same way, the top 50% contains roughly 85% of all the cases belonging to the group PIH. Moreover, the top 60%, 70%, 80%, 90%, 100% all contains about 100% of all cases that take the category pregnancy induced hypertension.

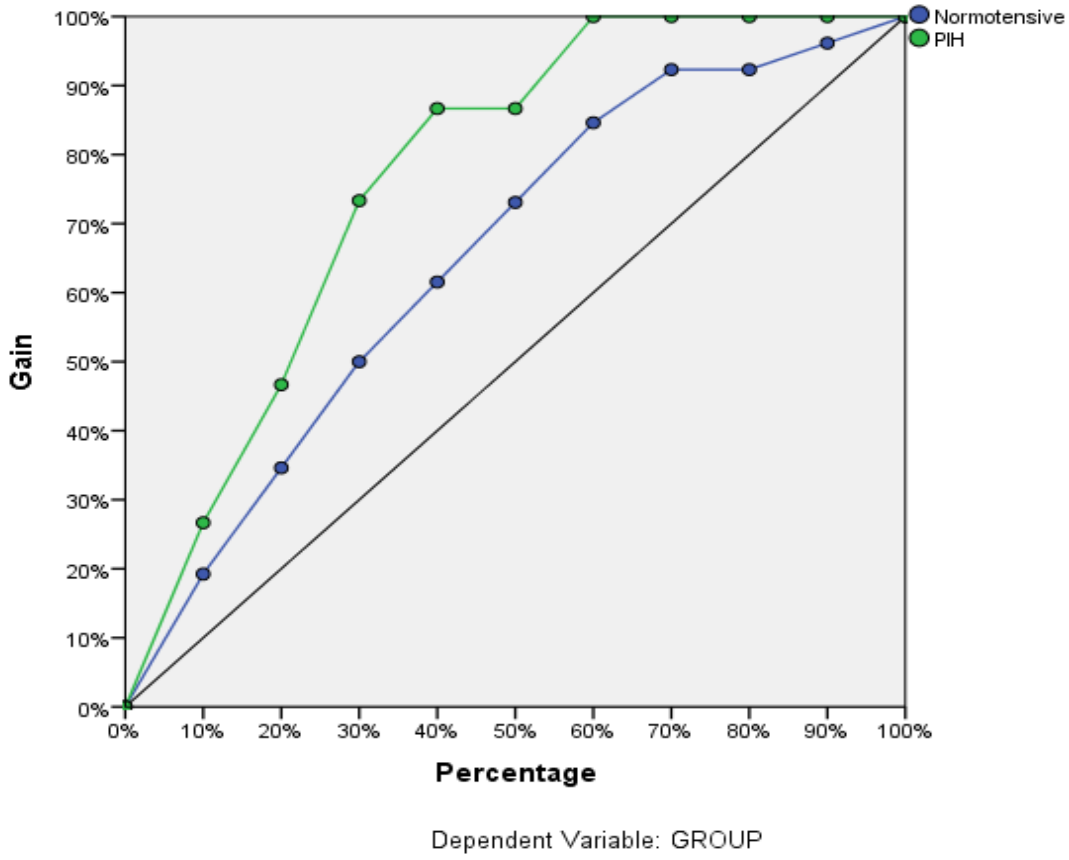

Figure 4.17: SPSS IBM software graphical output of cumulative gains chart

4.12 Cumulative Lift Chart

Figure 4.18 is the cumulative lift which is derived from the cumulative gains chart in which the y axis corresponds to the ratio of the cumulative gains for each curve to the baseline usually consisting of ten deciles. It is computed by taken the ratio of gain percentage to the random expectation percentage at a given decile level. The random expectation at the Xth decile is x percentage.

The lift chart also gives another alternative way to looking at the information in the cumulative gains chart. The Cumulative Lift of 2.8 for top two deciles indicates that in selecting 20% of the records based on the model, one can expect 2.8 times

the total number of cases found by randomly selecting 20% of cases without a model. Again, for top four deciles indicates that in selecting 40% of the records based on the model, we can expect that roughly 2.2 times the number of cases found by selecting 40% of the cases without a model.

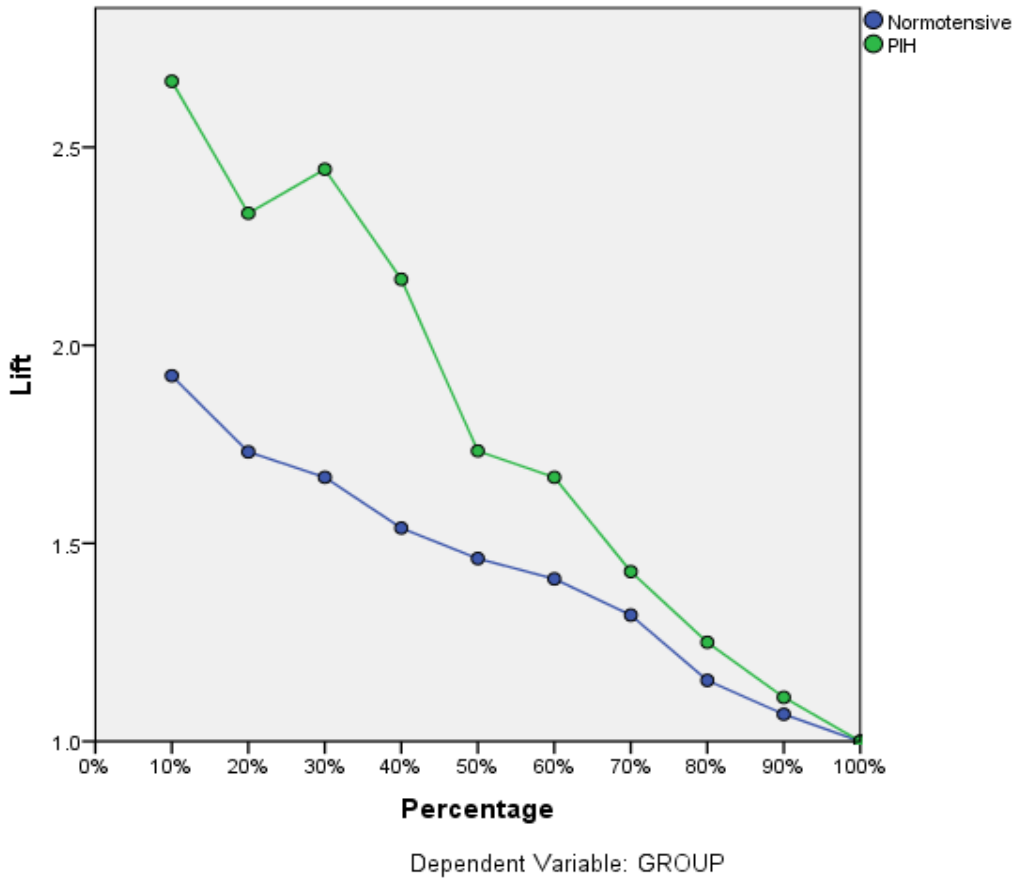

Figure 4.18: SPSS IBM software graphical output of cumulative lift chart

4.13 Model Comparison

Table 4.12 shows the results of postestimation test conducted on the population under investigation using Probit model, Logit model and the artificial neural

network model (ANN). The model diagnostics was done with a primary aim to compare models and adopt the one that gives the best results for further analysis.

The results from the postestimation test shows that 86% of the overall population were correctly classified under the Probit regression model for the data analysis. The logistic regression model also indicated a percentage of 86% correctly classified. However, 63.2% was realized as the overall cases that were correctly classified when the artificial neural network was applied in the data scrutiny. This further suggests that in terms of correct classification of true positive rate, Logistic regression model is most appropriate in analyzing the data.

Again, logistic model indicated that there is the probability of 0.905 of accurately predicting the true positive rate (sensitivity) of having hypertension in pregnancy from area under the ROC curve (AUC) whiles the probit regression model indicate a probability of 0.902 accuracy in predicting the true positive rate (sensitivity). But then, the artificial neural network seems to have a better predictive probability value of 0.897 for the same pointer.

In view of the above analysis, the Logistic regression model will be most appropriate amongst the three models to adopt for data analysis.

Table 4.12: Post estimate comparison of models

Classification test	Probit model	Logistic model	Artificial Neural Network model
Correctly classified	86%	86%	63.20%
AUC	0.902	0.905	0.897

CHAPTER FIVE

SUMMARY, CONCLUSION AND RECOMMENDATION

5.0 Introduction

This chapter presents the summary, conclusions and recommendations of the study. However, it has been sub divided into summary, conclusions and recommendations.

5.1 Summary

The study investigated the impact of some risk factors of pregnancy induced hypertension in the Upper East Region using a primary data from the Bolgatanga Regional hospital, upper east region.

The data were taken from pregnant women during their gestational period, after 20 weeks of pregnancy who visited the maternal health unit of the hospital. A total of 50 pregnant women were recruited for the study and their obstetric history were recorded for onward analysis of which sixteen (16) indicators were considered.

In order to achieve the objectives of the study, three (3) different binary classifier models which include the Logistic regression model, the Probit model and Artificial Neural Network model were applied in analyzing the data. The dependent variable had two outcomes namely pregnancy induced hypertension (PIH) coded 1; and normal blood pressure (normotension) coded 0.

The preliminary results indicated that, mothers ages were between a minimum of 16yrs to a maximum of 41 of age. and the mean age for mothers being 27.84yes. Parity was between 0 birth to 6 births amongst the study sample. Number of

pregnancies (gravidity) averaged 2.56 pregnancies with a minimum of 0 and a maximum of 7 pregnancies.

The primary results also revealed that pregnant mothers had gestational weight which ranged between 43.70kg to 130kg and average gestational weight was 70.44kg for the entire sampled population. Again, blood sugar level (glucose) for maternal mothers was as low as 7.9mg/dL and as high as 52.9mg/dL with a mean of 28.17mg/dL.

Nonetheless, the results show that the minimum cholesterol level was 17.30mg/dL and a maximum of 47.2mg/dL. Also, the study population averaged 25.72kgmm2 for body mass index with a maximum of 47.20kgmm2 and a minimum of 7.285kgmm.

However, further investigation on the distribution of parity within the sampled data reveal that, women who have never given birth (parity 0) constituted 34% of the population under study followed by women who gave birth ones (parity 1) with a 24%. The percentage kept decreasing as the number of births increases with least representation of 2% of women occurring at parity 6 although parity 2,3,4, and 5 constituted 40% of the entire data.

Similarly, the number of pregnancies among the study group was investigated and that women who never got pregnant or experiencing pregnancy for the first time (gravida 0) and those with multigravida 7 only contributed 2% of the data whilst women who ever got pregnant just once (gravida 1) had the larger percentage of 34%. But then, gravida 3 had the second highest representation of 22% of the

sampled population and then came gravida 2 with 18%. Gravida 4, 5 and 6 together constituted 22% of the study population.

The summary statistics on the distribution of parity to blood pressure shown that, of 17 pregnant mothers who have never given birth (parity 0), 5 of them were found to be hypertensive and 5 women from parity 3 were also found to be hypertensive. Four (4) women from parity 1 and 3 women from parity 2 had gestational hypertension whiles a parity 4, 5 and 6 altogether had 3 women having gestational hypertension from a total of 6 maternal mothers.

In the same way, women in their first pregnancy (gravida 0) did not have high blood pressure whereas those with gravida 1, 2 and 3 had 5, 3 and 6 pregnant women respectively had gestational hypertension. Also, women with gravida 4 and 6 had 2 women each found to be hypertensive though women with gravida 5 and 7 had one (1) person each with pregnancy induced hypertension.

Further analysis from the three models revealed that logistic regression had the highest percentage of correct classification of the data with a value of 86% and as well had the highest true positive rate of 0.905. From the logistic regression results, body mass index (BMI), low-density lipoprotein cholesterol (LDL-C) and white blood cells (WBC) showed to be statistically significant with respective probability values of 0.025, 0.027 and 0.01.

However, the probit model results indicated that gestational weight (0.026), Body Mass Index (0.006), Low-density Lipoprotein Cholesterol (0.013) and White Blood Cell (0.01) were statistically significant.

5.2 Conclusion

The current study indicated that though body mass index (BMI) has a greater impact on hypertension in one model, gestational hypertension, low- density lipoprotein cholesterol and white blood cells equally contribute significantly to the development of hypertension in pregnancy.

5.3 Recommendation

It is imperative for the Government of Ghana and other Non-Governmental Organizations to invest in strengthening the healthcare delivery system especially in rural Ghana by making available basic logistics, medical, and laboratory equipment, as well as improving upon maternal health education, and consistently organizing capacity building training programs for healthcare workers.

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