UNIVERSITY OF GHANA, LEGON

STUDIES ON THE MORPHOLOGICAL VARIATIONS OF THE HUMAN UMBILICAL CORD

BY

JOHN AHENKORAH

UNIVERSITY OF GHANA, LEGON

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JOHN AHENKORAH

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DECLARATION

I hereby declare that this thesis is the result of work done by myself. Except for literature cited which served as sources of information, the work is in no way a reproduction in part or in whole of any work ever presented for the award of a degree in any university.

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JOHN AHENKORAH

(STUDENT)

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PROF. F. N. L. ENGMANN	PROF. F..K.ADDAI	PROF. A.LAWSON
(Supervisor)	(Supervisor)	(Supervisor)

DEDICATION

This thesis is dedicated to the Almighty God and to the following:

Christiana, Angela and Christabel Ahenkorah.

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ABSTRACT

Four hundred and twenty-four consecutive umbilical cords of babies delivered at the Korle-Bu Teaching Hospital were studied along side some birth variables. This research was to first of all, find whether there were any possible correlations between any pair of variables. Secondly, whether any variant of the cord could have any clinical significance. The umbilical cord measurements were made using a tape measure and the results statistically analysed using the Statistical Package for the Social Sciences (SPSS) VERSION 10.0.

The range of umbilical cord length was between 23.0 cm to 88.0 cm. The mean cord length was 54.52 ± 10.82 cm. There was a significant positive but low correlation between cord length and foetal weight ($r = 0.228$, $p < 0.01$), cord length and placenta weight ($r = 0.250$, $p < 0.01$), cord length and placenta thickness ($r = 0.118$, $p < 0.05$), cord length and foetal head circumference ($r = 0.121$, $p < 0.05$), and cord length with baby's full length ($r = 0.234$, $p < 0.01$). Placenta weight and Birth weight had a moderate significant positive correlation ($r = 0.539$, $p < 0.05$). The correlation between maternal age, parity and placenta widest diameter with cord length was non significant ($p > 0.05$).

The cords were mostly accentrically located on the placentas (87.3%) of the total 424. Non-furcate cords were not significantly different from furcate cords in their mean placenta weight, birth weight, cord length,

placenta thickness and placenta widest diameter ($p > 0.05$).

The dangers of furcate cords, possible factors influencing the length of the cord, as well as pregnancy outcome of short and long cords were discussed.

CHAPTER ONE

INTRODUCTION

1.1 DESCRIPTION OF UMBILICAL CORD

The human umbilical cord is a complex and fascinating structure found between the foetus's umbilicus and the foetal surface of the placenta during pregnancy. It has been studied for many years by early investigators (Gardiner, 1922; Reynold, 1952; Walker and Pye, 1960; Malpas, 1964) all showing an interest in the length of the umbilical cord.

Recent studies on the cord have been directed into other aspects apart from the length, recording useful information on the subject (Sloper *et al.*, 1979; Ogita *et al.*, 1989; Durand *et al.*, 1996; Atalla *et al.*, 1998; Raio *et al.*, 1999; Sornes, 2000; Calvano *et al.*, 2000; Pennati, 2001).

Knowledge about the umbilical cord is important because the vessels in the cord are essential parts of the foetal circulation. The umbilical cord has also been found to be a marker for intrapartum complications (Rayburn *et al*, 1981; Malpas, 1964). Miller *et al.* (1981) and Moessinger *et al.*, (1982) have reported that, delay in the completion of second stage of labour could sometimes be attributed to short umbilical cords. Long cords on the other hand, have been reported to cause entanglement around foetal parts (Horwitz *et al.*, 1964) as well as knotting, which could be fatal to the developing foetus (Naeye, 1987; McLennan *et al.*, 1988).

The length of the umbilical cord is widely believed to be one predisposing factor in breech presentation of the foetus at birth (Soernes & Bakke, 1986).

It has been reported that the umbilical cord's amniotic surface and the endothelium of the vein, are two major sources of prostaglandin E2 during term labour (Harold *et al.*, 1988; McCoshen *et al.*, 1989).

An important role of prostagladin E2, just like oxytocin, has been found to be the induction of labour by causing uterine contractions. Prostaglandin E2 can also be inserted vaginally to soften the cervix in women with pre-eclampsia, cardiac or renal diseases. In cases of intrauterine foetal death, prostaglandins alone or with oxytocin seem to ensure effective delivery (Foegh, 1989; Hayashi, 1990).

Umbilical cord blood is used in scientific research studies, as in prenatal diagnoses to find chromosomal and biochemical abnormalities and determine foetal well being (Nicolaides, 1986; Attala *et al.*, 1998).

In a study to determine the water content of the human umbilical cord, Sloper *et al.*, (1979) reported that the mean water content of the umbilical cord fell with increasing gestation and that the mean umbilical cord water content was higher for preterm cords than in term cords. They also found that the foetal end of the cord had significantly higher water content than the placental end.

Very little work has been done in this area of cord length measurement and the correlation that might exist between the cord with foetal and maternal factors. In Africa, apart from Agboola, (1978) and Nnatu, (1991) who have reported on the length of the umbilical cord, little or no attention has been paid to the subject of umbilical cord length especially in Ghana.

To the best of my knowledge, scanty studies on the morphological variations of the human umbilical cord has been done with respect to its length and various variables measured after delivery i.e. placenta weight, baby weight, placenta thickness, placenta widest diameter, baby full length, and baby head circumference.

The objective of the research is to find out whether there are any possible correlations between cord length and the various variables of birth.

1.2 STRUCTURE OF THE UMBILICAL CORD.

The gross anatomy of the human umbilical cord is generally simple. It is normally made up of a vein and two arteries surrounded by a mucoid connective tissue. Wharton's jelly, that gives protection to the vessels (McKay *et al.*, 1955). The vein brings nutrient rich and oxygenated blood from the mother to the developing foetus whereas the arteries return deoxygenated and waste products to the mother.

The Wharton's jelly has been reported to be composed of open-chain polysaccharides, hyaluronic acid, carbohydrate, distributed in a fine network of microfibrils and small amounts of collagen (Graumann, 1964; Yamada and Shimizu, 1976). The surface of the cord is covered by amnion and according to Benirschke and Kaufmann (1990), the amnion of the cord attaches firmly to the Wharton's jelly in contrast to the amnion that covers the chorionic surface of the placenta, which can be dislodged easily. The tensile and biomechanical properties of the cord have been reported by Ghosh *et al.*,1984 and Pennati, 2001, to enable the incompressible cord to resist torsion.

Histologically, it has been reported that the epithelium of the cord shows changes from an unkeratinized, stratified squamous type, which provides the transition from the abdominal wall to the cord's surface, to a stratified columnar farther away and finally into a simple columnar epithelium (Hoyes, 1969; Sinha, 1971; Hempel, 1972).

Reynold (1952) reported that the human umbilical arteries are histologically unique due to the presence of nodes of Hoboken (crescentric folds of the intima) not seen in other vessels of the body. Some investigators have reported other vessel differences like the unusually rich organelles in the endothelial cells of the arteries and the vein (Parry and Abramovich, 1972). Boyd and Hamilton (1970) and Nikolov and Schiebler (1973) also noted that the arteries have no elastic lamina.

Most investigators generally accept that the umbilical cord has no functional neural investment (Lauweryns *et al.*, 1969; Benirschke *et al.*, 1990). in contrast to formerly accepted views of nerve supply to the cord (Kernbach,1963; Fox and Jacobson ,1969; Pearson and Sauter, 1969).

1.3 DEVELOPMENT OF THE HUMAN UMBILICAL CORD

The umbilical cord develops from the connecting stalk during embryonic life. The connecting stalk is formed from the uncavitated extra-embryonic mesoderm, which joins the caudal end of the embryonic disc and the primary yolk sac to the internal surface of the chorion (Hamilton *et al.*, 1976) (see Fig 1).
The connecting stalk is the only point where extra-embryonic mesoderm traverses the chorionic cavity such that by the second week of embryonic development, the embyro is attached to its trophoblastic shell by the narrow connecting stalk.

During further embryonic development, the embryonic disc undergoes head and tail fold and the attachment of the mesodermal connecting stalk to its

caudal end becomes ventrally situated with the formation of a primitive umbilical ring. The connecting stalk, the allantois (a diverticulum of the yolk sac), vitelline duct (joining the yolk sac and the midgut of the embryo), and the canal connecting the intra embryonic and extra embryonic cavities all pass through the primitive umbilical ring at the ventral side.(see Fig 2).

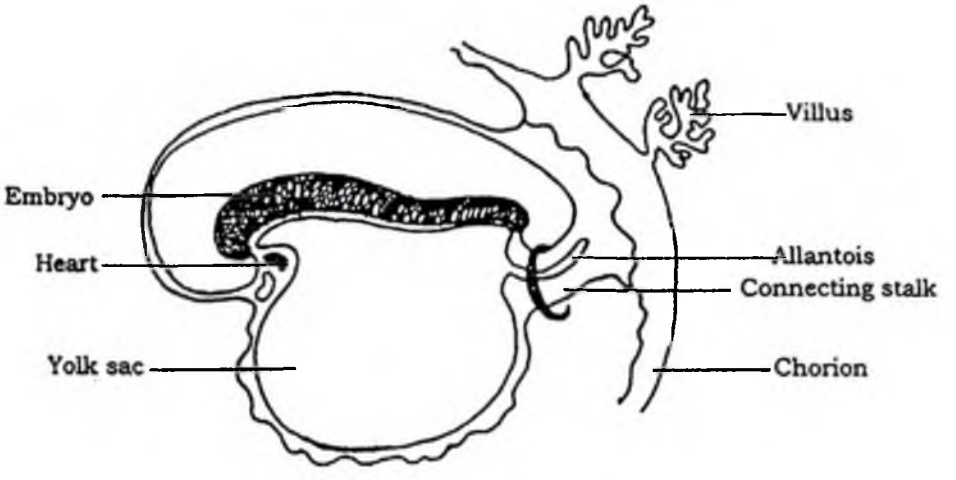

Fig. 1. A 19 day old embryo showing the connecting stalk at its caudal end

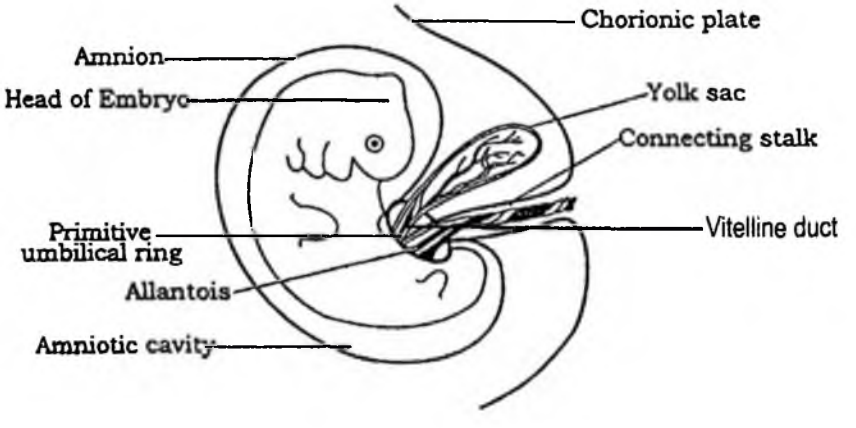

Fig. 2. A 5 week old embryo showing the primitive umbilical ring

As growth of the embryo continues, the enlarging amniotic cavity expands more and more into the extra-embryonic coelom until the remnant of the yolk sac and the elongated vitello-intestinal duct are pushed right up against the connecting stalk. The amnion begin to envelop the connecting stalk, allantois and the vitello-intestinal duct, crowding them to form a primitive umbilical cord.

The proximal part of the primitive cord contains an intestinal loop in its cavity (the extra embryonic space in the cord) and the remnant of the allantois. The extruding intestinal loop forms a physiological umbilical hernia, as a result of the rapidly developing intestinal loops in a temporarily small abdominal cavity. When the loops are withdrawn into the abdominal cavity of the embryo, the allantois and the vitelline duct become obliterated and all that remains in the cord are the umbilical blood vessels. The umbilical cord thus forms a channel for blood vessels between the foetus and the placenta (Sadler, 2000).

For development to continue, the formation of a vascular connection between the uterus of the mother and the embryo is to enable the embryo to obtain nutrients and oxygen. As early as the third week, the trophoblast becomes characterised by primary villi from the chorionic plate (the extra-embryonic mesoderm lining the inside of the cytotrophoblast). The primary villi consist of a cytotrophoblast core covered by a syncytial layer. Mesodermal cells then penetrate the cytotrophoblast core of primary villi, which grow in the direction of the decidua basalis (the part of the endometrium that covers the developing villi). The structure now formed is called a secondary villus.

By the end of the third week, mesodermal cells in the core of the secondary villus begin to differentiate into small blood vessels and blood cells, forming a villus capillary system. The villus is now known as a tertiary villus or definitive placental villus. Capillaries in the villi make contact with capillaries developing at the same time in the mesoderm of the chorionic plate and in the umbilical cord. These vessels, in turn, establish contact with the intra embryonic circulatory system, connecting the placenta and the embryo via the umbilical cord. Branches of the umbilical vessels give rise to chorionic vessels, which radiate to and from the umbilical cord and are clearly seen through the transparent amnion on the foetal side of the placenta.

1.4 PLACENTA

The placenta is an organ formed only in pregnant mammals. It is the only organ formed by cells from two separate individuals (the foetus and the mother-i.e a foetomaternal organ). The foetal part, the chorionic frondosum (bushy chorion) originates from the trophoblast, as villi at the embryonic pole. The maternal part, desidua basalis is formed from the uterine endometrium. The placenta is discoid in shape with a diameter of about 15-20cm. It is approximately 3cm thick and weighs about 500-600g (Sadler, 2000).

In a freshly delivered placenta, about 15-20 slightly bulging areas, the cotyledons or lobes covered by a thin layer of decidua basalis may be seen at the maternal surface. The foetal part is made up of the chorionic plate and chorionic blood vessels, which can be seen through the transparent amnion, with the

umbilical cord inserted or positioned on this side of the placenta. At various stages during intrauterine life, the placenta performs a wide range of functions, which include gaseous exchange, nutrient interchange, excretion, and hormone production.

1.5 FOETAL BLOOD CIRCULATION

A major feature of foetal blood circulation is that the foetal lungs, gut and liver are non-functional, with oxygen and nutrients being obtained from the placenta (Berne *et al.*, 1993). The circulation of blood through a foetus is therefore different from that of a newborn infant. Respiration, the procurement of nutrients, and the elimination of metabolic wastes occur through the maternal blood instead of through the organs of the foetus.

Maternal blood from the spiral arteries pierce the decidual plate and enter the intervillous spaces under pressure due to the narrow lumen of the spiral arteries. Oxygenated and nutrient-rich blood moves through the villous tree and then along the chorionic villi (Sadler, 2000).

Blood then flows through the umbilical vein toward the inferior surface of the liver. At this point, the umbilical vein gives small branches to supply the liver and then joins the inferior vena cava [IVC] through the *DUCTUS VENOSUS* (a venous shunt within the liver). The umbilical vein is reported to be the only vessel of the foetus that carries fully oxygenated blood (Berne *et al.*, 1993).

The IVC empties its blood contents into the right atrium of the foetal heart. From here, most of the blood is directed through the foramen ovale and

foramen secundum into the left atrium. Blood in the left atrium mixes with a small quantity of blood returning from the pulmonary circulation, then passes into the left ventricle, from where it is pumped into the aorta and through the body of the foetus.

Since the lungs of the foetus are not functional, only a small portion of blood continues through the pulmonary circulation. Most of the blood in the pulmonary trunk passes through the *DUCTUS ARTERIOSUS* into the aorta. Blood is returned to the placenta by the two umbilical arteries that arise from the internal iliac arteries (Berne *et al.*, 1993).

1.6 CIRCULATORY CHANGES AT BIRTH.

Changes in the vascular system at birth are caused by cessation of the placenta blood flow and the beginning of respiration (Sadler, 2000).

There has been much interest in the mechanisms of the closure of the umbilical vessels after birth. Yao *et al.,* (1977) observed umbilical cords after delivery and reported irregular constrictions in the walls of the umbilical arteries, closing them. Functionally, the arteries close a few minutes after birth. Closure of the umbilical vein and the ductus venosus occurs shortly *after* that of the umbilical arteries. This mechanism by which the arteries close first after delivery is important. because hemorrhage is prevented at the umbilicus.

The distal part of the umbilical arteries becomes obliterated forming the left and right medial umbilical ligaments, while the proximal portions remain open as the superior vesical arteries of the internal iliac arteries.

After obliteration, the umbilical vein forms the *ligamentum teres hepatis* in the lower margin of the falciform ligament. The foramen ovale becomes the fossa ovalis and ductus arteriosus becomes the *ligamentum arteriosum.*

The *ductus venosus,* which is between the *ligamentum teres* and IVC, is also obliterated, and forms the *ligamentum venosum* (Sadler, 2000).

1.7 MORPHOLOGY OF THE HUMAN UMBILICAL CORD.

Morphologically, the human umbilical cord shows general variability in its gross features. These include its length, and location on the placenta, either centrally, accentric or marginal. There could be separation of the vessels before insertion on the placenta, described as furcate, (Herberz, 1938).

The absence of one of the umbilical arteries, according to (Parilla *et al.*, 1995; Lemtis, 1966 and 1968; Monie *et al.*, 1973) may have adverse effects on the foetus including causing cardiac diseases.

The cord is usually spiralled, thought to be the result of some foetal activity , and the lack of it reflects inactivity and possible central nervous system disturbances (Benirschke and Kaufmann, 1990).

According to Lacro *et al.*, (1987), helices of the cord may be seen by ultrasonograhic examination as early as the first trimester of pregnancy.

A counter-clockwise (left) helix has been reported to occur more frequently than right helices, and occasionally a mixture of left and right helices occurs (Blackburn *et al.*, 1988). Chaurasia *et al.* (1979) however, reported that the incidence of right helical patterns was higher in full term cords than the left helical ones and that some cords did not show any helical pattern whereas others exhibited a mixture.

The vessels in the cord may separate out before reaching the foetal surface of the placenta as mentioned earlier and this gives rise to a furcate umbilical cord on the placenta (Hamilton *et al.*, 1976). Ottow (1923) and Herberz (1938) described it as *insertio funiculi furcata*. In this state, the umbilical vessels lose their protective covering of the Whartons jelly and branch some distance before reaching the placental surface (Fox, 1978). Clinically, these exposed vessels are highly susceptible to damage. Kesseler (1960) has described foetal haemorrhage as a result of this anomaly.

According to Kalousek, lesions of the cord have been found to include knot formation, rupture, torsion and strictures, but these have been found to be extremely rare in spontaneously aborted foetuses, They reported cases of vascular lesions in the cord, including:

Edema of the cord: The cord assumes a swollen appearance due to found a collection of fluid in the Wharton's jelly. Coulter *et a*l. (1975) associated this condition with respiratory distress syndrome in newborns, whereas Rolschau (1978), found that edema of the cord had no influence on foetal development and well-being.

Cord hematomas : They are usually single and consist of an extravasation of blood into the Wharton's jelly. Cord hematomas may be caused by the rupture of either an umbilical artery or vein leading to hemorrhage into the Wharton's jelly. Cord hematomas are reportedly rare findings in gestation under 20 weeks, except for traumatic lesions produced accidentally by a needle during amniocentesis or cordocentesis (Kalousek *et al.*, 1990). Thrombosis of cord vessels has been reported as a complication of cord compression, torsion, stricture or hematoma in third-trimester pregnancies (Benirschke and Kaufmann, 1990).

Anyaegbunam & Sherer (1997) in a review of literature on the umbilical cord, cited some congenital abnormalities which have been reported, such as persistent right umbilical vein (Bell *et al.*, 1986), cysts, pseudocysts, umbilical vessel aneurysm and fused umbilical arteries.

1.8 UMBILICAL CORD LENGTH

Not least of the several intriguing features of the human umbilical cord is its length, which has been found to vary considerably (Walker and Pye, 1960).

Researchers have accumulated some information on umbilical cord length, citing different ranges.

Gardiner (1922), reported that most usual length range of the human umbilical cord was 50-56 cm and that a minimum length of about 35.5 cm was necessary for normal delivery. Agboola (1978) reported a mean cord length of 57.8 cm at term whereas Nnatsu (1991) quoted 58.39±12.02 cm. Walker and Pye (1960) also

found that normal gestation and delivery could occur with a cord of length between 17.8-121.9 cms, with a mean length of 54.1cm.

Naeye (1985) reported that the human umbilical cord progressively lengthens from a mean of 32 cm at 20weeks of gestation to 60 cm at term.

Durand *et al.*(1996) stated that the umbilical cord could vary in length from 20 cm to more than one meter with an average of 50 cm in length.

According to Walker and Pye, (1960) observations on other mammals are scanty but there is evidence that humans share with other primates some similarities in cord length. They reported that a chimpanzee (Pan troglodytes) was found with a cord length around 49cm, and an orangutang (Pongo pygmaeus) with a cord of 60cm long.

The major factor that regulates the length of the cord is still not clearly understood. Moessinger *et al.*, (1982) propounded a theory that cord length is influenced by tension applied to the cord by foetal movement, with greater foetal movement, resulting in longer the umbilical cords. Miller *et al.* (1981) also suggested that cord growth occurs in response to tensile forces placed upon it in relation to intrauterine space availability and movements of the foetus, particularly in the period prior to 30 weeks of gestation. They reported that when the foetus is constrained, as occurs for instance with amnionic adhesions and ectopic pregnancies, the cord is normally short.

Short umbilical cord may therefore be helpful in the evaluation of a sick or dysmorphic newborn infant, since it might suggest diminished foetal movement from either early intrauterine constraint or foetal limb dysfunction.

In a follow up study based on the theory that foetal movement influence cord length, Moessinger *et al.*, (1986) observed that infants with Down's syndrome had significantly shorter cords compared with matched standards (mean cord length of 45.1cm compared with 57.3cm). It was not clear however, whether those infants had short cords on the bases of decreased foetal activity, genetic make up, or both. It might be interesting to see if those with short cords (presumed to have been the most hypoactive *in utero*) could be predicted to exhibit some neuro-developmental outcome.

Walker and Pye (1960) measured 177 umbilical cords and found no evidence of growth in cord length after the 28 or 30th week of gestation. In other words, the umbilical cord attains its full length at the beginning of the eighth month of pregnancy. Naeye (1985) however, found that growth of the umbilical cord slowed after 28 weeks of gestation but did not stop before term.

Malpas (1964) found that the length of the umbilical cord at term varies widely. He observed that studies involving statistical analysis of length of the cord were surprisingly few in number. Purola ,1968 found the same situation with studies concerning the correlation that may exist between cord length on the one hand, and various foetal and maternal variables on the other

Malpas (1964) suggested that since the length of the cord can be arranged in a continuous series, a comparison between cord length and other indices of foetal growth becomes a legitimate and a possible line of inquiry.

Obstetrics has recognised the mechanical consequences of an abnormally short or abnormally long cord. A length of at least 32cm has been claimed

necessary to prevent traction on the cord during vaginal delivery. Short cords as mentioned earlier, have been found to occasionally delay completion of the second stage of labour or cause placenta abruptios, inversion of the uterus, and cord rupture (Gardiner, 1922; Rayburn *et al.*, 1981; Miller *et al.*, 1981 and 1982; Moessinger *et al.*, 1982).

Naeye (1985), from retrospective analysis of measured umbilical cord lengths of 35,779 neonates, reported that a short cord (<40cm) was found to be a good predictor of subsequent psychomotor impairment or low intelligent quotient (I.Q) values in later childhood. He concluded that since short umbilical cords originate in developmental conditions long before labour and delivery, recorded measurements of cord length could be important when the obstetric management of labour and delivery or the clinical management of neonates are claimed to be the cause of later mental and motor impairments.

Sornes and Bakke (1989) reported that there seem to be a widely held notion that the uterine cavity is larger during a woman's later pregnancies than her first pregnancy. Although the direct evidence for this assertion is scarce, this theory of a larger intra-uterine space in later pregnancies, which could influence cord length, was supported by their work. They reported that babies born to primiparous (first time) mothers had comparatively short mean cord lengths than tertiparous (third time) mothers. Since cord length at term has been reported as one measure of foetal intrauterine mobility, and indirectly of the space in which the foetus moves, it therefore presupposes that if the uterine cavity is larger the cord length should be longer (Miller *et al.*, 1981).

Interestingly, Sornes and Bakke, (1989) further reported a significant positive correlation, in that a first baby tended to have a shorter cord than the second baby of the same mother.

According to Walker and Pye, (1960), and Wu *et al.* (1996) male children tend to have slightly longer cords than their female counterparts. Wu *et al.* (1996) also reported longer cord lengths in vertex presentation than in breech.

Abnormally long cords have been reported to have higher frequencies of cord prolapse, true knots and coiling of the cord around foetal parts, all of which may represent hazards to the foetus, in the form of foetal distress, asphyxia through cord compression in labour or intrautrine death. (Greenhill, (1962); Moessinger *et al.*, 1982; Miller *et al.*, 1983; Katz *et al.*, 1999; Sornes, 2000).

For example, if the presenting part of the foetus was high in the pelvic brim and the membranes rupture, the umbilical cord may be swept down with the gush of liquor. This may result in cord compression, which can lead to foetal hypoxia as the presenting part descends into the pelvis (Brant *et al.*, 1966; Hickman, 1985).

Wessel *et al.*(1992) implicated umbilical cord complications in the cause of intrauterine foetal death and Kadyrov *et al.*, (1991) reported disturbance in materno-foetal circulation as a result of cord compression.

1.9 KNOTTING OF THE CORD

The mechanism of the formation of True knots (TK) remains obscure, since the process cannot be observed. Early researchers suggested that they are formed during labour (Greenhill, 1962) or perhaps in early foetal life (Javert *et al.*, 1952). According to Blickstein *et al.*, (1987) True knots are formed when during an uneventful pregnancy (probably in a multiparous gravida with a spacious uterus), cord length become sufficient to form a loop, whose diameter may be large enough to allow the foetus to pass through it, thereby forming a knot.

Knotting of the cord may be lethal and it is believed to account for 1 in 10 stillbirths (Mclennan, 1988). Sornes (2000) observed that neonates with a knotted cord are more often larger-for-gestational age compared to other babies, and have longer umbilical cords. He further reported that there is a ten (10) times higher chance of intrauterine foetal death with knotted cords.

A cord entangled around the neck, body, or limb of a foetus is an uncommon but known cause of some stillbirths. Often such a cord leaves easily identified grooves on the skin (Naeye, 1987). Benirschke (1994) reported that in about 1 in 50 deliveries, the cord is loosely looped around the neck without increased foetal risk.

1.10 POSITION OF CORD ON PLACENTA.

The position of the umbilical cord on the placenta shows variability. According to Fox, (1978) it may vary from centric (when the cord is situated at the center of the placenta) to a marginal or peripheral attachment. Uyanwah-Akpom and Fox (1977) reported that it is more commonly situated eccentrically i.e. a little distance away from the center but not touching the margin of the placenta. Occasionally, the cord may be inserted in the membranes outside the placental tissue and this has been described as velamentous, which has the highest risk of vessel damage, leading to foetal bleeding (Uyanwah-Akpom and Fox, 1977).

A peripheral insertion of the cord results in a Battledore placenta (Hamilton *et al.*, 1976) and this has been found to predispose to cord prolapse. Some reports have linked peripheral cord insertion with a variety of anomalies in the course of pregnancy and neonatal development e.g. premature labour (Brody and Frenkel, 1953), abortions (Hathout, 1964), malformed infants (Monie, 1965), neonatal asphyxia (Raaflaub, 1959), foetal growth failure and stillbirth (Salafia and Vintzileos, 1990). Other investigators, however, could not demonstrate any such association with foetal abnormalities (Uyanwah-Akpom and Fox, 1977; Robinson *et al.*, 1983).

The factors that control the insertion of the cord on the placenta at a particular position are far from being fully understood (Uyanwah-Akpom and Fox, 1977). According to Benirschke and Driscoll, (1967) the cord is originally inserted at the center of the placenta, but as the placenta expands, the cord

becomes peripherally sited as a result of central atrophy and unidirectional lateral growth of the chorionic frondosum.

Monie (1965) and Mclennan (1968) have on the other hand, maintained that peripheral cord insertion is a consequence of an oblique implantaion of the blastocyst. Whichever of these concepts is accepted, Uyanwah-Akpom and Fox, (1977) suggest that the site or the position of the umbilical cord on the placenta is determined at an early stage of pregnancy.

CHAPTER TWO

MATERIALS AND METHODS

2.1 STUDY SUBJECTS AND ELIGIBILTY CRITERIA.

This cross-sectional study of neonates delivered by mothers took place at the Labour Ward of Korle-Bu Teaching hospital in Accra.

2.2 SAMPLE SIZE DETERMINATION.

A minimum sample size was estimated based on the following equation and assumptions, (Lwanger *et al.*, 1991):

$N = (z^2 p q)/d^2$

Where N = the number of neonates required in the survey;
Z = the normal deviate of 1.96, (assuming an alpha of 0.05);
P = 0.05, assuming the presence of abnormal cord lengths
q = (1- p)
d = the precision of the estimate (i.e. how close to the population mean the estimate should be , d = 0.05).

This gave an approximate computed minimum figure of 384 neonates. However, 424 neonates were eventually studied. Consecutive deliveries that fulfilled the following criteria were selected.

- Singleton birth
- Pregnancies with known gestational age within 38±2weeks

determined by ultrasound scan or estimated from the first day of the last menstrual period.

Excluded were:

- Mothers with known chronic or gestational diseases eg.hypertention, diabetes, pre-eclampsia, eclampsia and sickle cell (ss).
- Multiple pregnancies and abortions.

Information about the mother was obtained from admission records and was cross-checked from their antenatal/maternity folders.

2.3 OBSERVATION AND RECORDING.

The presenting part of the foetus during the second stage of labour was recorded as cephalic or breech. The mode of delivery was recorded as spontaneous vaginal delivery (SVD), Caesarean section (C/S), or vacuum extraction (VAC). The sex of the baby was recorded as (M) for males and (F) for females. Whether the baby was dead (D) or alive (A) on delivery was recorded.

2.3.1 CORD MEASUREMENTS

Umbilical cord stumps attached to the umbilicus of the babies were measured using a tape measure immediately after delivery. The babies were weighed in kilograms (kg) using a Waymaster weighing balance to the nearest 0.05kg and recorded as birth weight (BWt). Head circumference (HC) was measured in centimetres (cm) using a tape measure. The baby's full length (FL) or

crown-heel length was recorded using a Schaffer baby full-length measure in centimetres (cm).

When the placentas were delivered at the third stage of labour, they were carefully examined to find out whether the amniotic membranes and lobes were complete or incomplete. All traumatic incomplete placentas resulting from manual extractions were excluded.

The umbilical cord segment, from the point of insertion on the placenta to the cut end was measured with a tape measure within 10-20mins after allowing blood trapped in the cord to flow out. The measured length of cord attached to the umbilicus was then added to the length of the cord from the placenta to the cut end, to give an estimated total cord length, which was recorded as (UCL/cm) (Naeye, 1985, Walker and Pye, 1960).

2.3.2 DETERMINATION OF THE POSITION OF CORD ON PLACENTA

The position of the cord on the placenta was recorded as Centric (C), when the cord is situated right in the center of a placenta whose widest and shortest diameters do not differ in more than 2 cm in measurement (i.e. a near circular placenta) see *plate 1*.

The cord insertion was recorded as Accentric (ACC), [when it was situated anywhere between the center and the margin of the placenta] see *plate 2*, and Peripheral/marginal [PER] when any part of the cord touches the margin of the placenta (*plate 3*).

2.3.3 STATUS OF CORD VESSELS.

The status of the cord vessels on the placenta was categorised as FURCATE [F] when the umbilical vessels separated or branched from each other about 2.0 cm before insertion into the foetal surface of the placenta (*plate 4*). On the other hand, when the umbilical vessels continue as a compact triple helix within a sheath of Wharton's jelly until they reach the surface of the placenta, it is recorded as NON-FURCATE [NF] (*plate 5*) (Addai *et al.*, 1992).

2.3.4 PLACENTA MEASUREMENT

After these assessments, the membranes attached to the placenta were trimmed off the margin and the maternal surface washed free from blood clots after excising the umbilical cord completely. The maternal surface (*plate 6*) was carefully examined and an attempt was made to count the number of formed cotyledons (lobules), which were recorded (NOC). Those placentas with poorly delineated sulci and extra placenta lobes were not counted.

On the foetal surface (*plate 7*), the widest placenta diameter (PDIA) was measured with a tape measure in centimetres (*plate 7*). The placenta was weighed and recorded as the trimmed placenta weight (PWT) to the nearest 50 g.

The thickness of the placenta (PT) (*plate 9*) was measured at the point of cord insertion with a plastic meter rule in centimetres, from the chorionic plate to the

basal plate at the cut surface and recorded. This was done, by making a deep and sharp incision with a scalpel blade, separating the placenta into two parts, passing through the point of insertion.

2.3.5 MATERNAL INFORMATION

Parity, gestational age and age of mothers were extracted from the mother's antenatal cards.

2.4 ANALYSIS OF DATA

Data collected were logged into Dbase statistical package for statistical analysis using the Statistical Package for the Social Science (SPSS) version 10.0.

PLATE 1

A CENTRIC UMBILICAL CORD POSITION ON A PLACENTA.

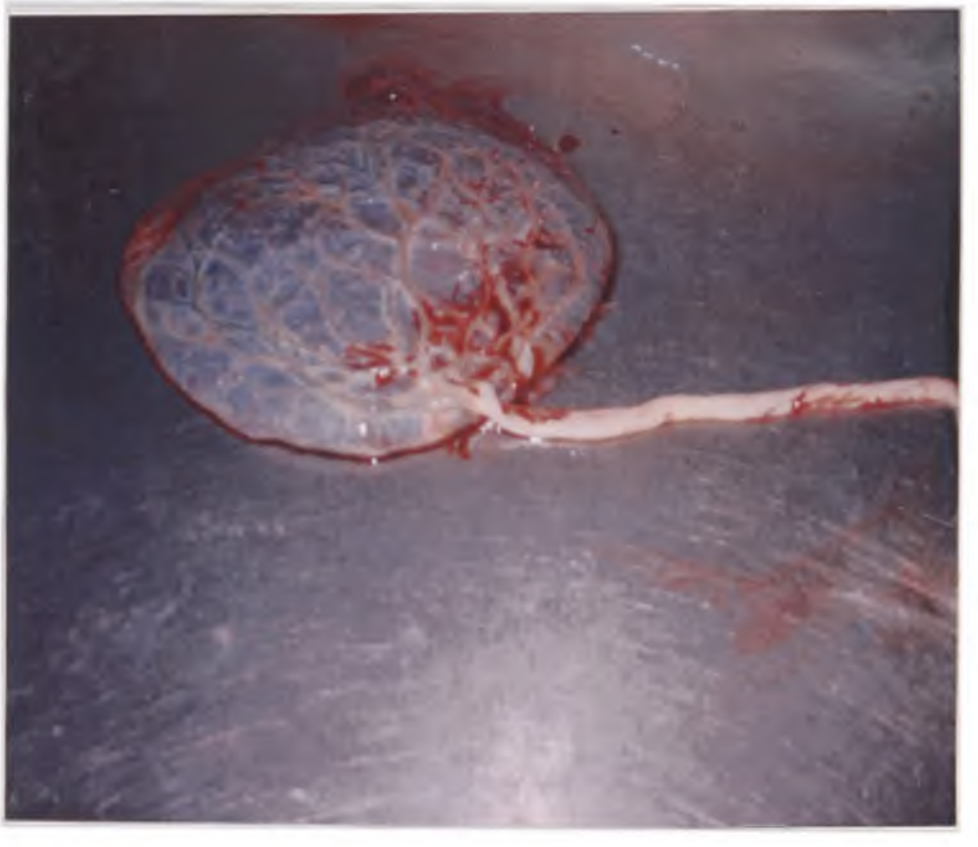

PLATE 3

A PERIPHERAL OR MARGINAL UMBILICAL CORD POSITION ON A PLACENTA

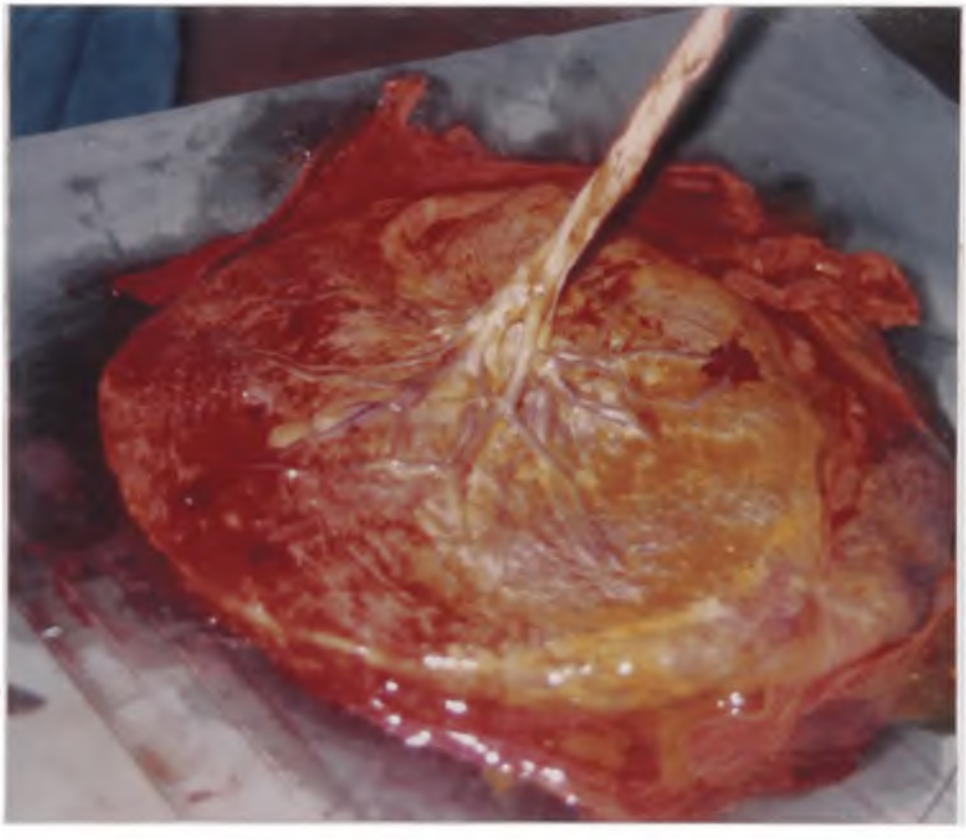

PLATE 4

A FURCATE UMBILICAL CORD

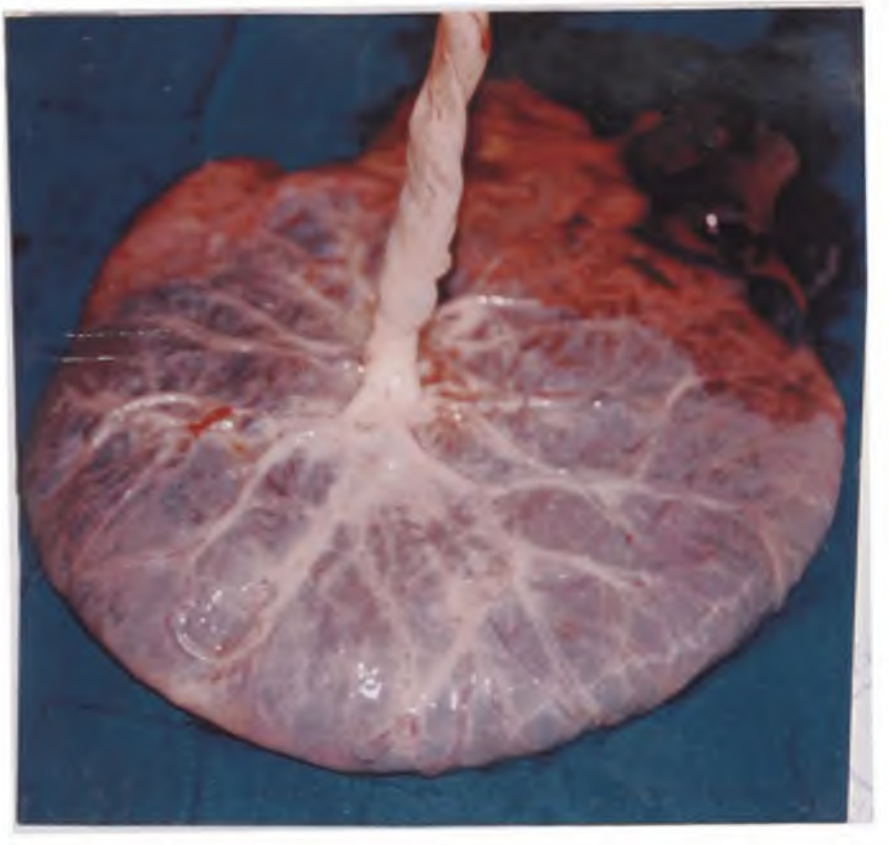

PLATE 5

A NON-FURCATE UMBILICAL CORD

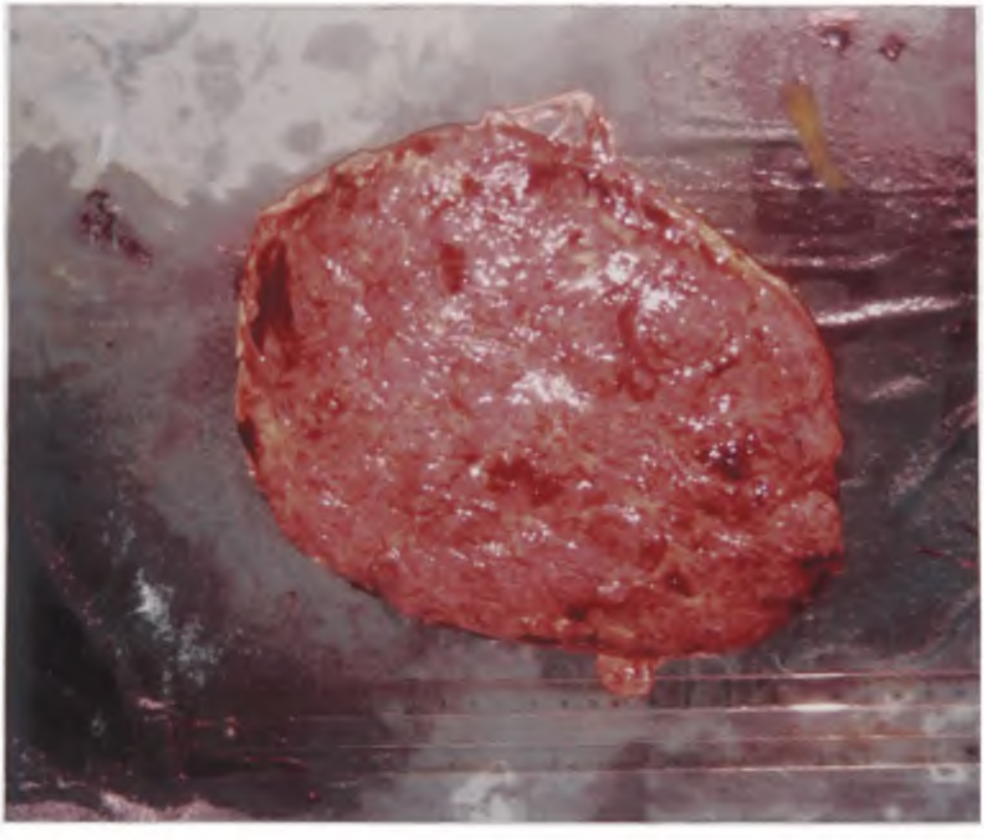

PLATE 6

MATERNAL SURFACE OF A FRESHLY DELIVERED PLACENTA

PLATE 7

FOETAL SURFACE OF A PLACENTA (UMBILICAL CORD AND MEMBRANES HAVE BEEN EXCISED)

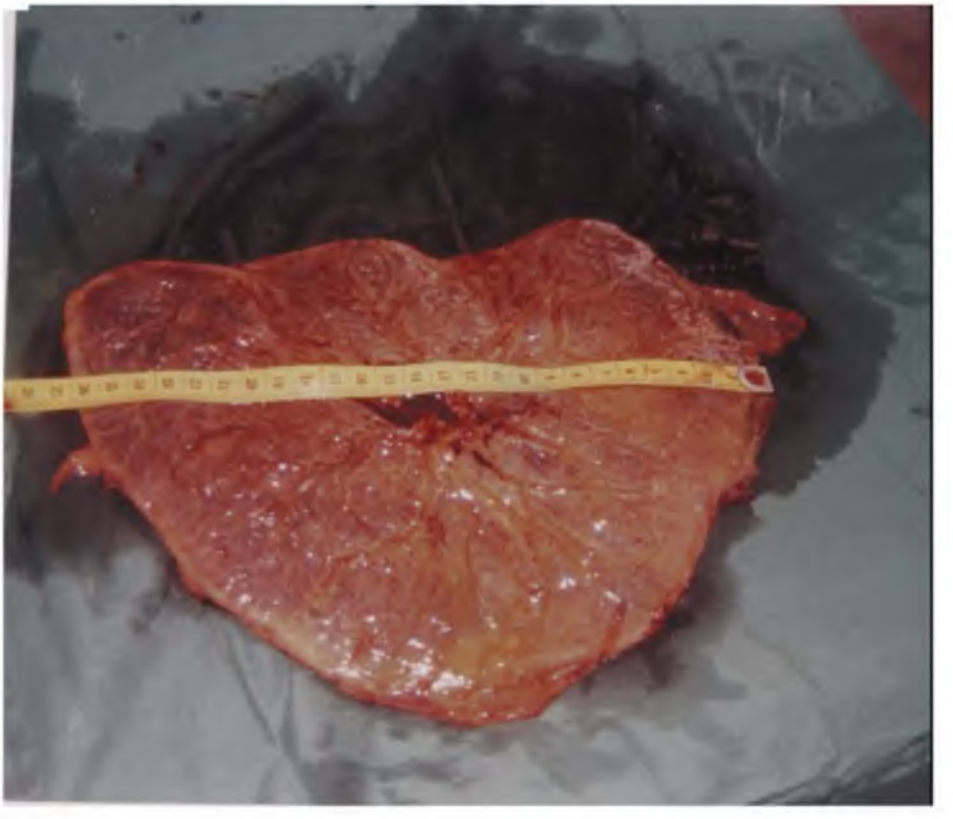

PLATE 8

ESTIMATION OF THE WIDEST PLACENTA DIAMETER

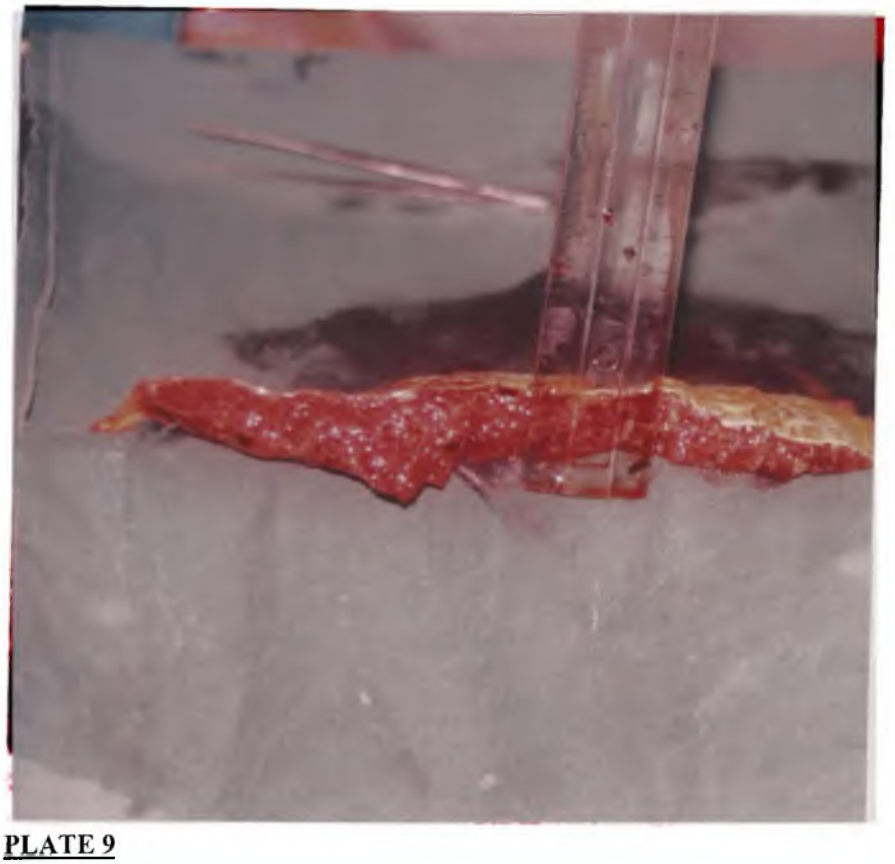

PLATE 9

ESTIMATION OF THE THICKNESS OF A PLACENTA

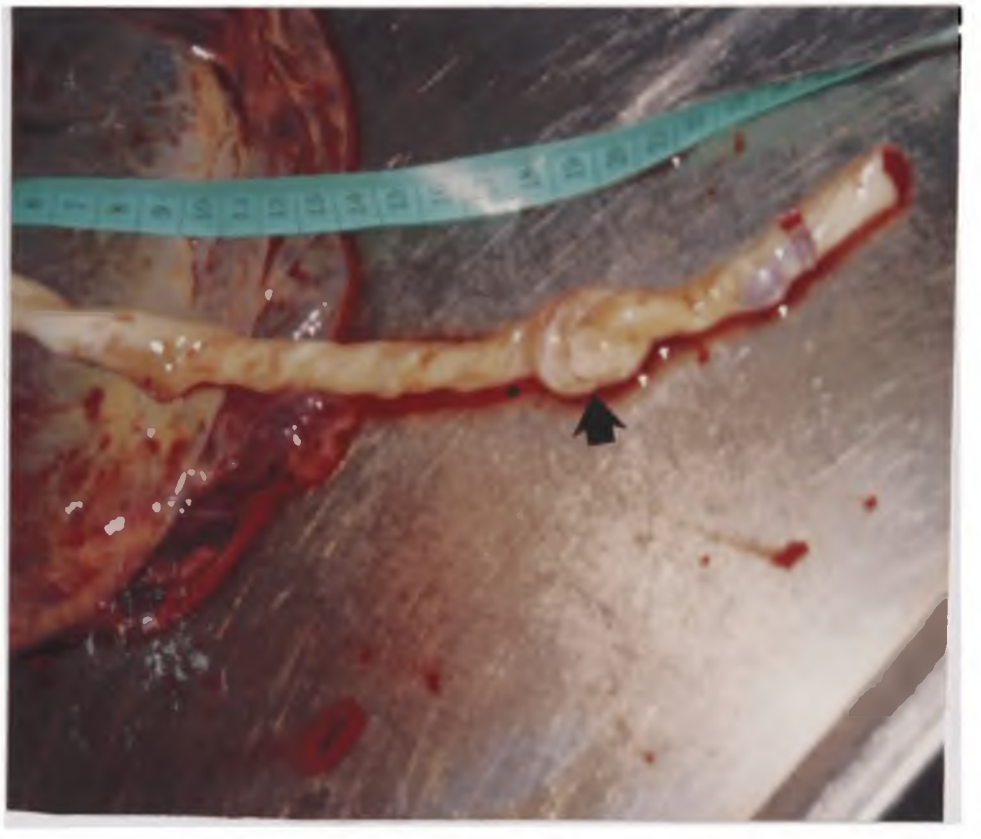

PLATE 10

A TRUE KNOT OF THE UMBILICAL CORD OF A LIVE BABY (ARROWED)

CHAPTER THREE

RESULTS

3.1 CORD LENGTH

The distribution of the cord lengths measured for the 424 neonates is shown a histogram (Fig. 1).

The class boundary with the highest frequency was between 50.5 to 55.5 and that was 86 . The class boundary with the lowest frequency occurred between 25.5 to 30.5 and that was 2.

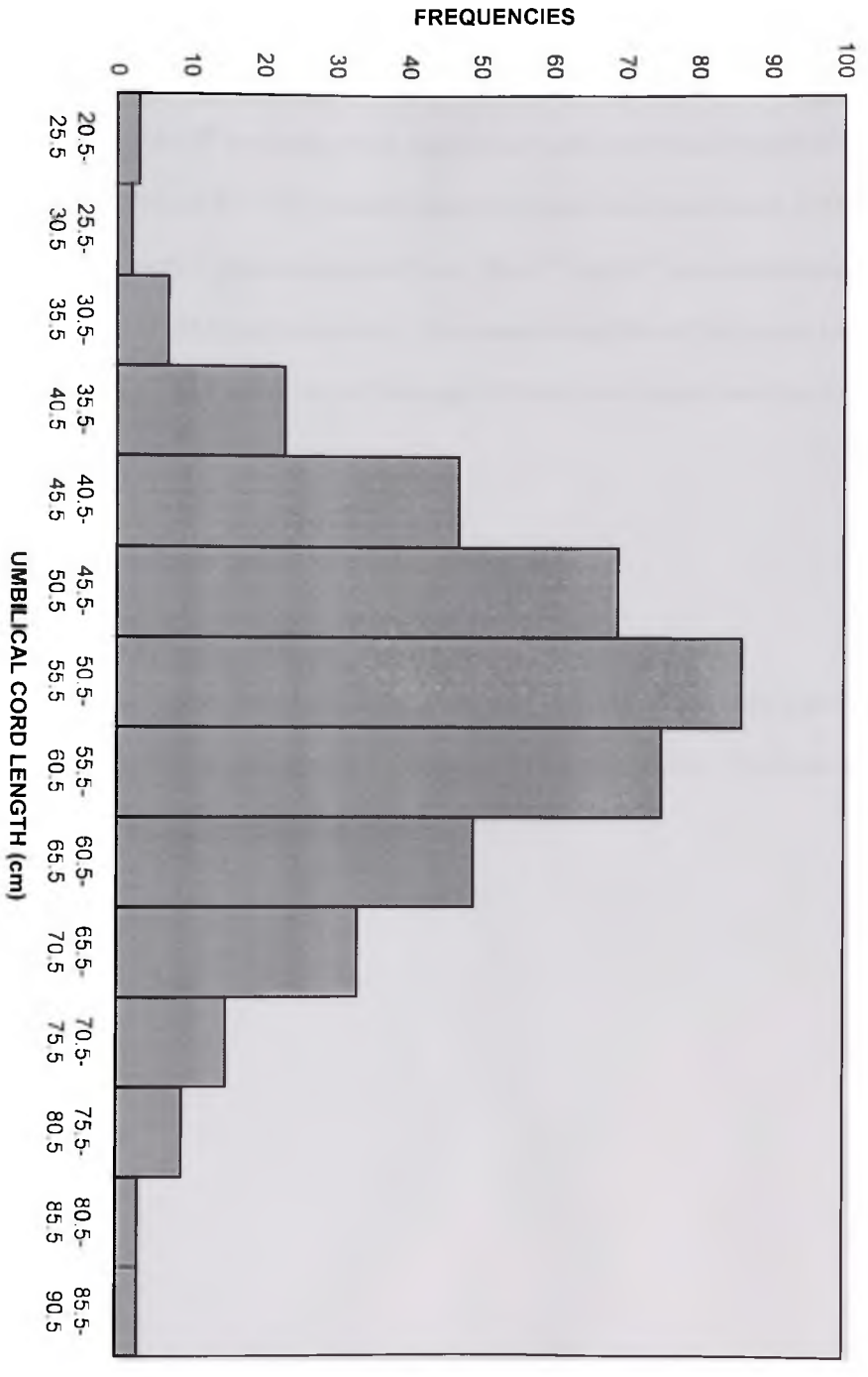

FIG.3: HISTOGRAM OF THE DISTRIBUTION OF CORD LENGTHS OF 424 NEONATES

From the histogram in Fig 3, it can be seen that umbilical cord length has a normal distribution.

The 10^{th} percentile of the sample had cord length less or equal to 41.0 cm, Whiles the 75th percentile had cord length less than or equal to 61.0 cm and the 90^{th} percentile was 68.5 cm. The 25^{th} and 50^{th} percentiles were 48.0 cm and 54.0 cm respectively. This means about 80% of the sample had cord lengths between 40 to 70cm and 10% had cord lengths less than 41cm.

The mean cord length in the series was 54.521±10.82cm with a minimum and maximum cord length of 23 cm and 88 cm respectively. The mean results (±1SD) are shown in Table.1

TABLE 1. Mean measured results of variables (with min and max values) (±1SD) (N= 424 neonates).

VARIABLES	minimum	maximum	Mean	S.D
Umbilical cord length (cm)	23.00	88.00	54.52	10.82
Placental weight (g)	250.00	950.00	579.01	130.04
Placental Diameter (cm)	17.00	30.00	21.09	1.96
Placental Thickness (cm)	1.00	3.50	2.27	0.40
Baby weight (kg)	1.50	4.70	3.20	0.47
Head Circumference (cm)	28 00	38.00	33.58	1.69
Full length of Baby (cm)	38.00	55.00	48.70	2.32
Gestation length (weeks)	36.00	41.00	37.81	1.11
Maternal Age (years)	15.00	46.00	27.94	5.71

By sex, a total of 214 males with mean cord length of 55.3±10.591 (SEM = 0.724 occurred in the series). There were 210 females with a mean cord length of 53.7±11.001 (SEM = 0.759).

A t-test calculated gave t =1.579 p = 0.115 (p>0.05), 95% CI= -0.405 to3.717. Male babies have slightly longer cords than their female counterparts but this difference was not statistically significant p>0.05. There was no statistically significant difference between the sexes in their mean placenta weights. However, there were statistically significant differences in mean birth weight, p<0.05 head circumference, p<0.05 and full length p<0.05 in favour of the males (Table 2).

TABLE 2. **Comparison of means of variables between 214 male and 210 female neonates.**

Variables	Sex	Mean	t-Test	Sig. (2-tailed)		S/NS
Length of cord (cm)	M F	55.30 53.70	1.579	0.115	p>0.05	NS
Placenta Wt (g)	M F	577.57 580.48	-0.230	0.818	p>0.05	NS
Head circumference (cm)	M F	33.78 33.37	2.482	0.013	P<0.05	S
Full Length (cm)	M F	49.07 48.32	3.339	0.001	P<0.05	S
Birth weight (kg)	M F	3.25 3.15	2.113	0.035	P<0.05	S

The results of the correlation co-efficient between cord length and foetal and maternal variables studied are shown in Table 3.

TABLE 3: **Correlation co-efficient between paired foetal and maternal variables.**

PAIRED VARIABLES	Pearson's (r) value	R^2	p-value	comments
UCL& PWt	0.250**	0.062	0.0001	p< 0.01 S
UCL& BWt	0.228**	0.053	0.0001	p< 0.01 S
UCL & PDIA	0.091	0.008	0.0610	p> 0.05 NS
UCL & PT	0.118*	0.014	0.0150	p< 0.05 S
UCL & HC	0.121*	0.014	0.0130	p< 0.05 S
UCL & FL	0.234**	0.046	0.0001	p< 0.01 S
UCL& parity	- 0.062	0.001	0.2050	p> 0.05 NS
UCL& MA	0.012	0.001	0.8040	p> 0.05 NS
PWt & BWt	0.539**	0.290	0.0001	p< 0.01 S
PWt & FL	0.394**	0.155	0.0001	p< 0.01 S
BWt &FL	0.567**	0.913	0.0001	p< 0.01 S
BWt & HC	0.463**	0.214	0.0001	p< 0.01 S
PDIA & BWt	0.441**	0.294	0.0001	p< 0.01 S
PT& BWt	0.385**	0.022	0.0001	p< 0.01 S

Correlation is significant at 0.01 level *, Correlation is significant at 0.05 level ** NS = not significant and S = significant (2 tailed).

From the results in Table 3, the correlation between umbilical cord length and placenta weight ($r = 0.250$, $p<0.01$), cord length with birth weight ($r = 0.228$, $p<0.01$), cord length and placenta thickness ($r = 0.118$, $p<0.05$), cord length with head circumference ($r = 0.121$, $p<0.05$) and also cord length with baby full length ($r = 0.234$, $p<0.01$) respectively, were positive and statistically significant.

Correlation between cord length with parity of mother was negative and non-significant ($r = 0.062$, $p>0.05$). Umbilical cord length and maternal age ($r = 0.012$, $p>0.05$) was non-significant as shown in Table 3.

A significant positive correlation exists between birth weight and placenta weight ($r = 0.539$, $p<0.01$). Placenta weight and full length of baby have a positive correlation ($r = 0.394$, $p<0.01$), placenta diameter and birth weight show a significant positive correlation($r = 0.441$, $p<0.01$), placenta thickness and birth weight show a positive correlation ($r = 0.385$, $p<0.01$).

Birth weight and full length correlate positively ($r = 0.567$, $p<0.01$). Birth weight and head circumference also show a positive significant correlation ($r = 0.463$, $p<0.01$).

Figures 4 to 15 show the graphic representations of the association between the variables measured and recorded for the 424 neonates sampled.

For example, Cord length and placenta weight follow the regression equation:

$y = 0.0208x + 42.504$, with an r^2 value of 0.2902 as shown in Fig 4. Where y = cord length and x = placenta wt.

Placenta weight and baby weight also follow the equation y = 0.0019x +2.0768 (R^2=0.2902) Where y = baby wt and x = placenta wt. shown in Fig 5.

Umbilical cord length and birth weight are related by the equation y = 9.6427x + 23 (R^2 = 0.0529) where y = umbilical cord length and x = birth weight (Fig 6).

Cord length and baby's full length are related by the equation y = 0.6483x +23(R^2 =0.045) x == baby's full length and y = umbilical cord length (Fig 7). Fig 8 shows the relationship between cord length and parity etc.

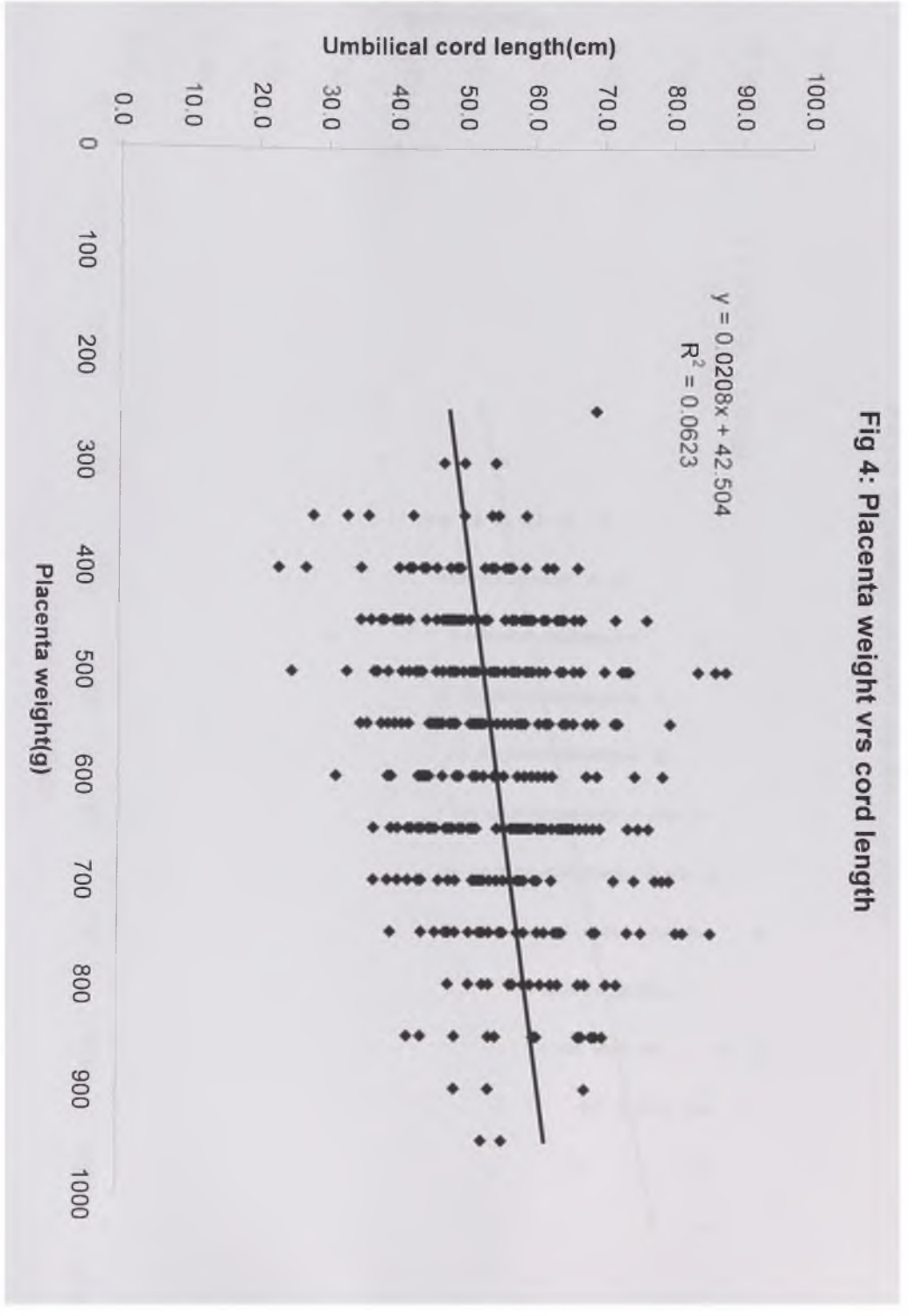

Fig 4: Placenta weight vrs cord length

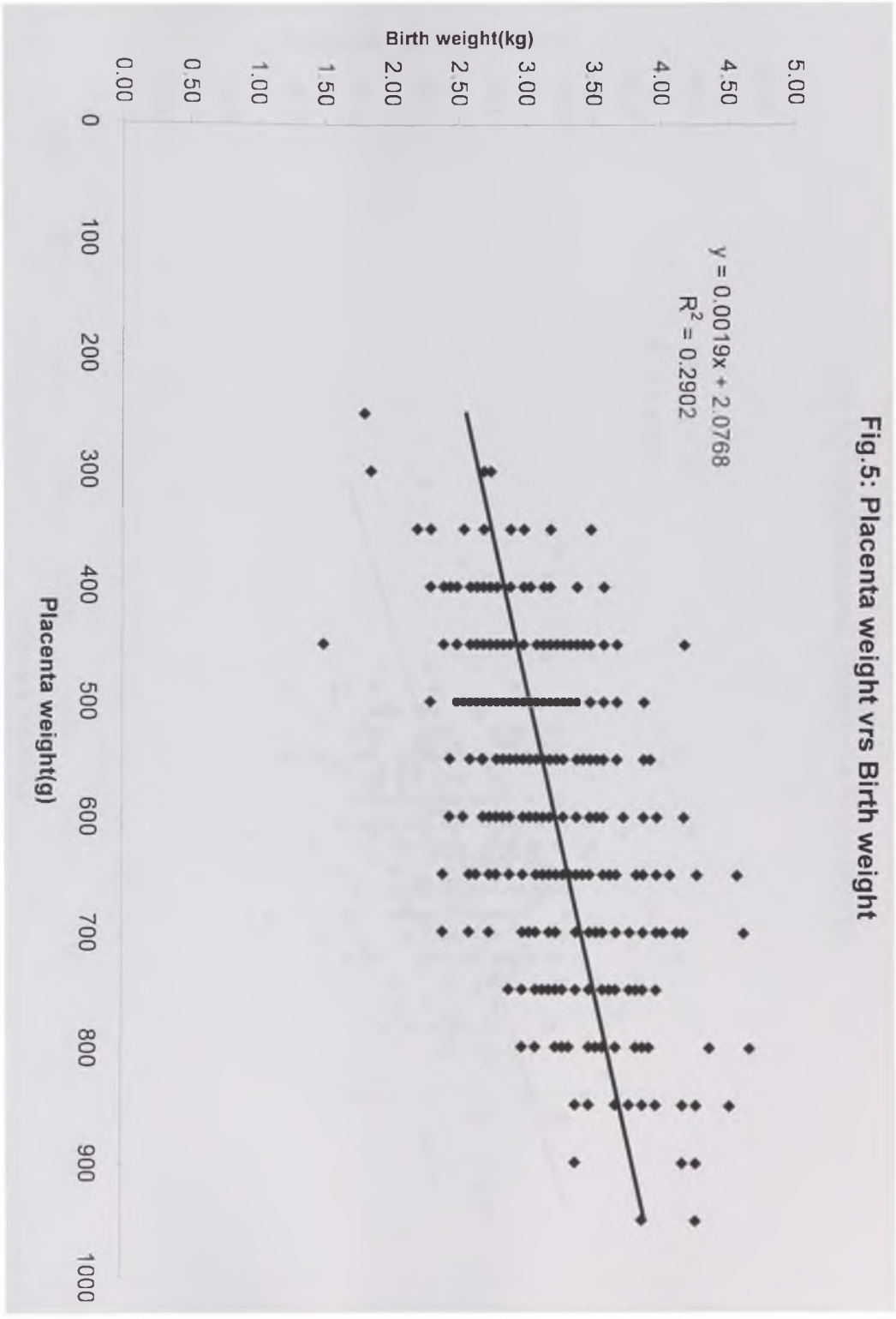

Fig.5: Placenta weight vrs Birth weight

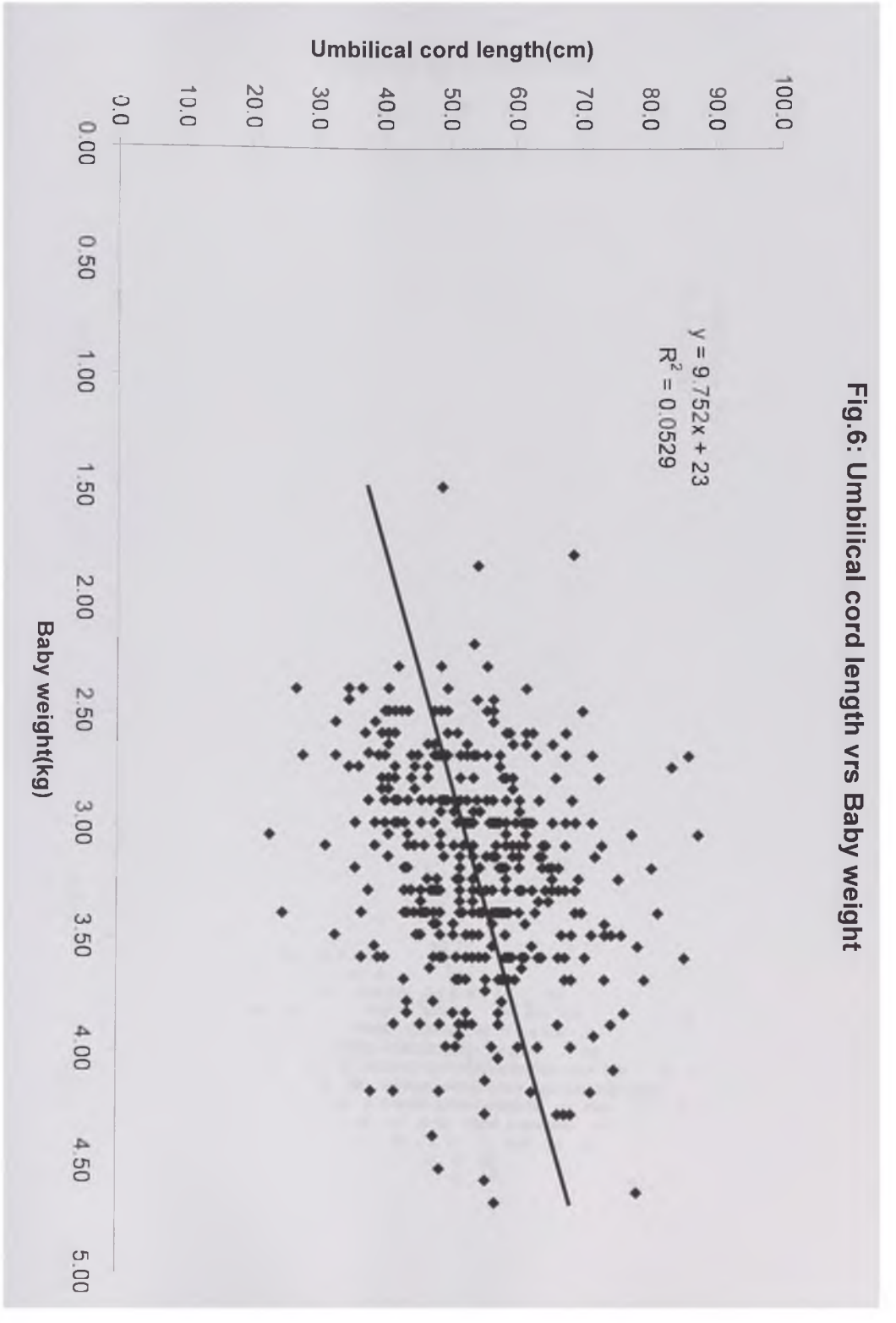

Fig.6: Umbilical cord length vrs Baby weight

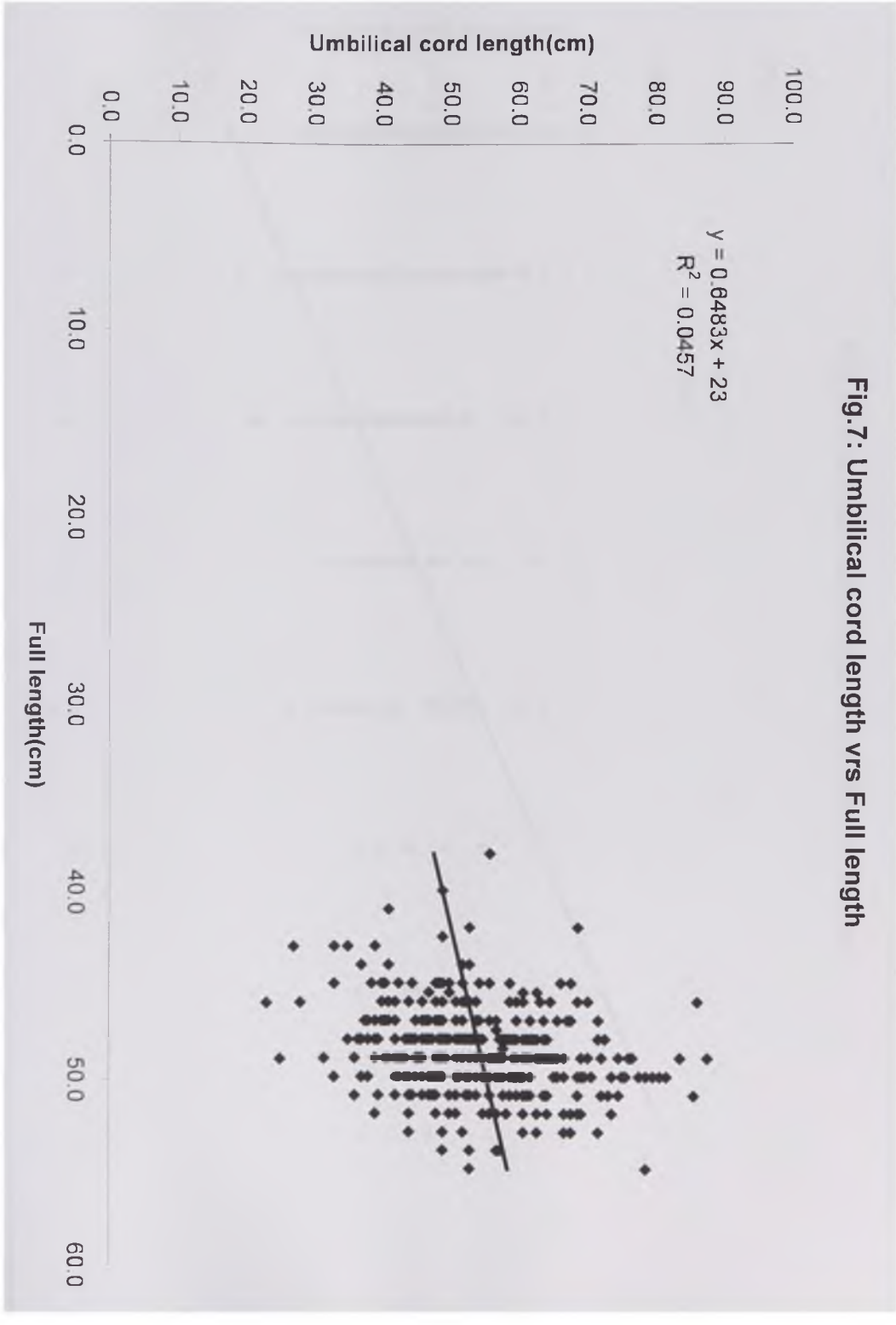

Fig.7: Umbilical cord length vrs Full length

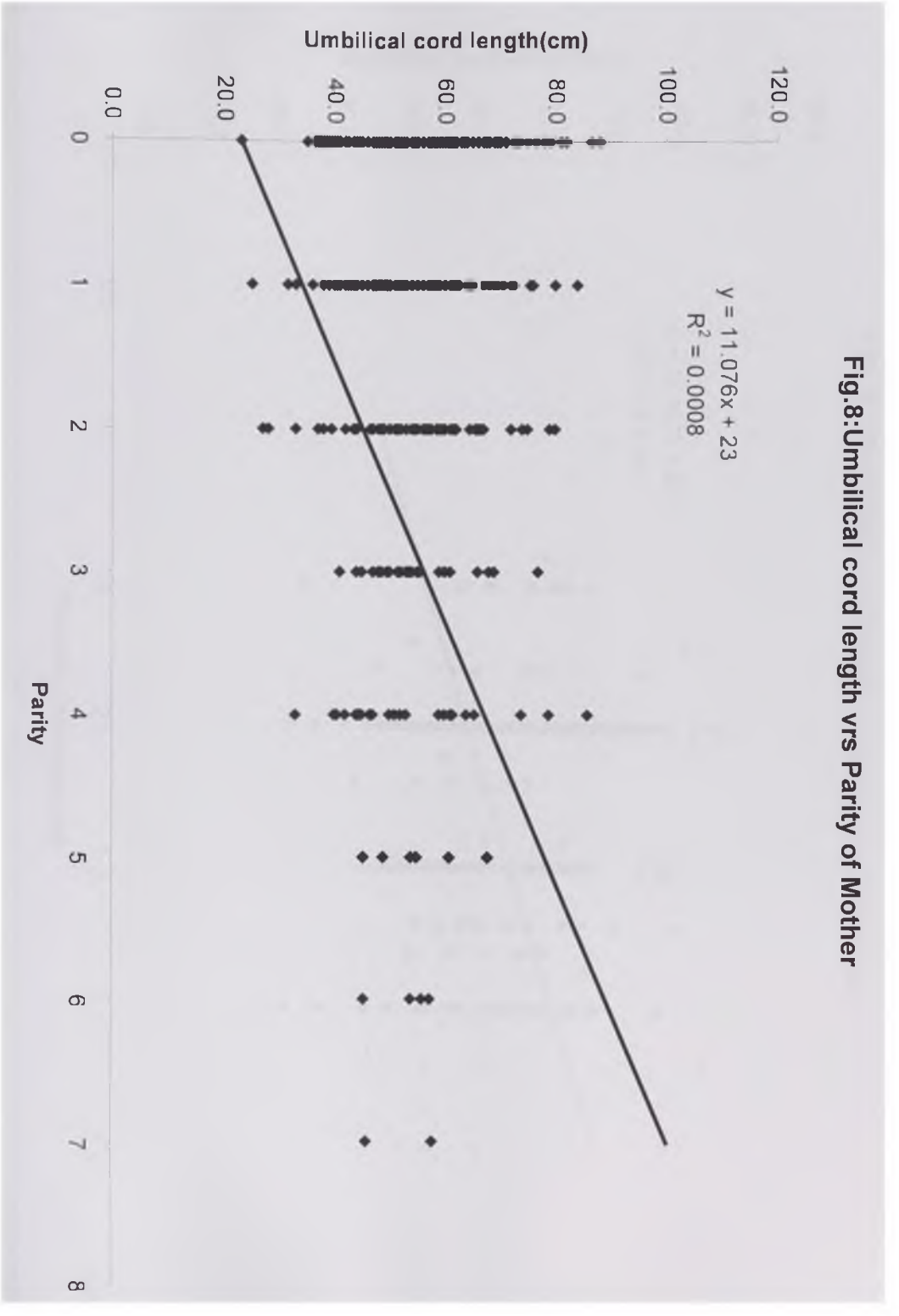

Fig.8:Umbilical cord length vrs Parity of Mother

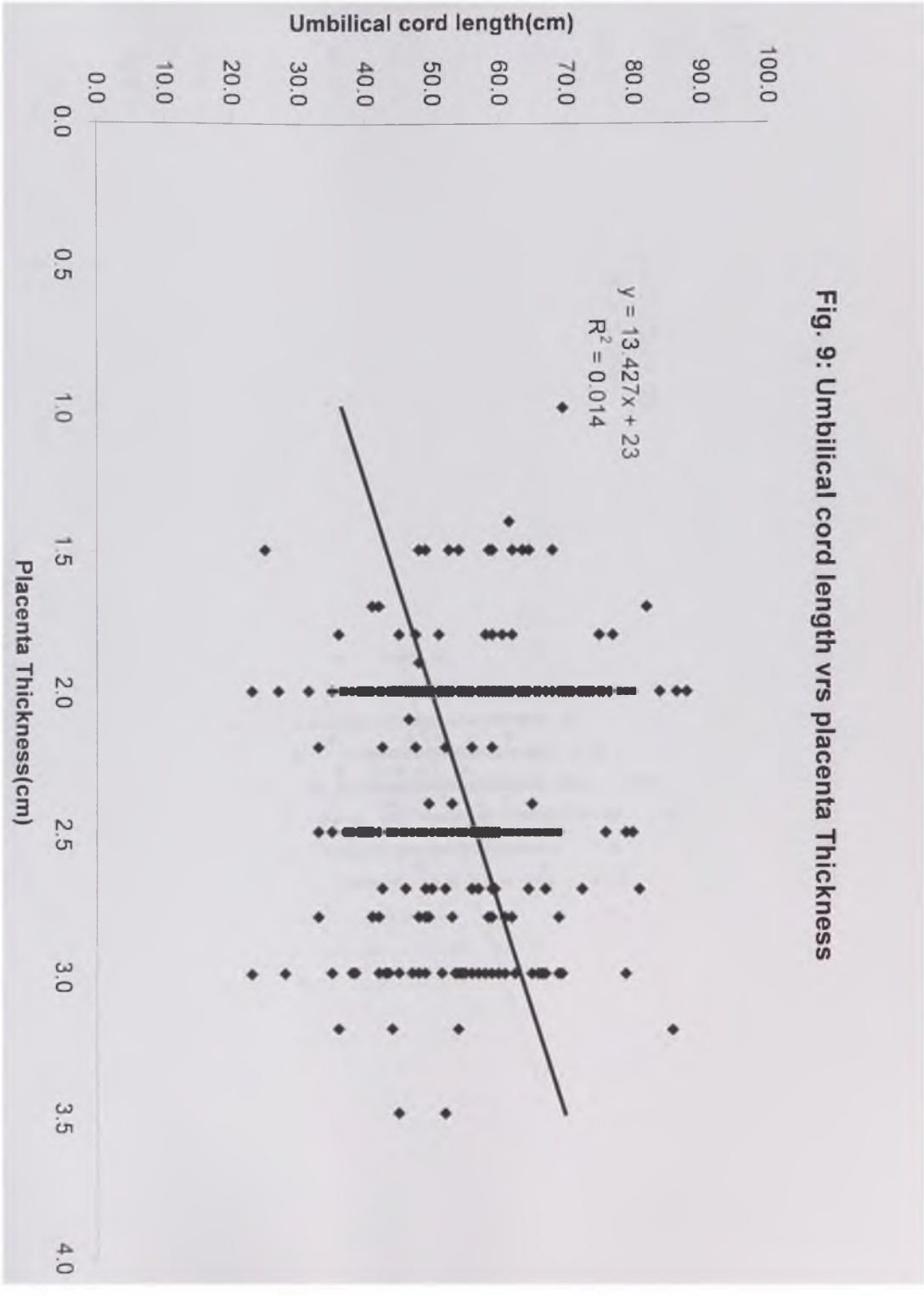

Fig. 9: Umbilical cord length vrs placenta Thickness

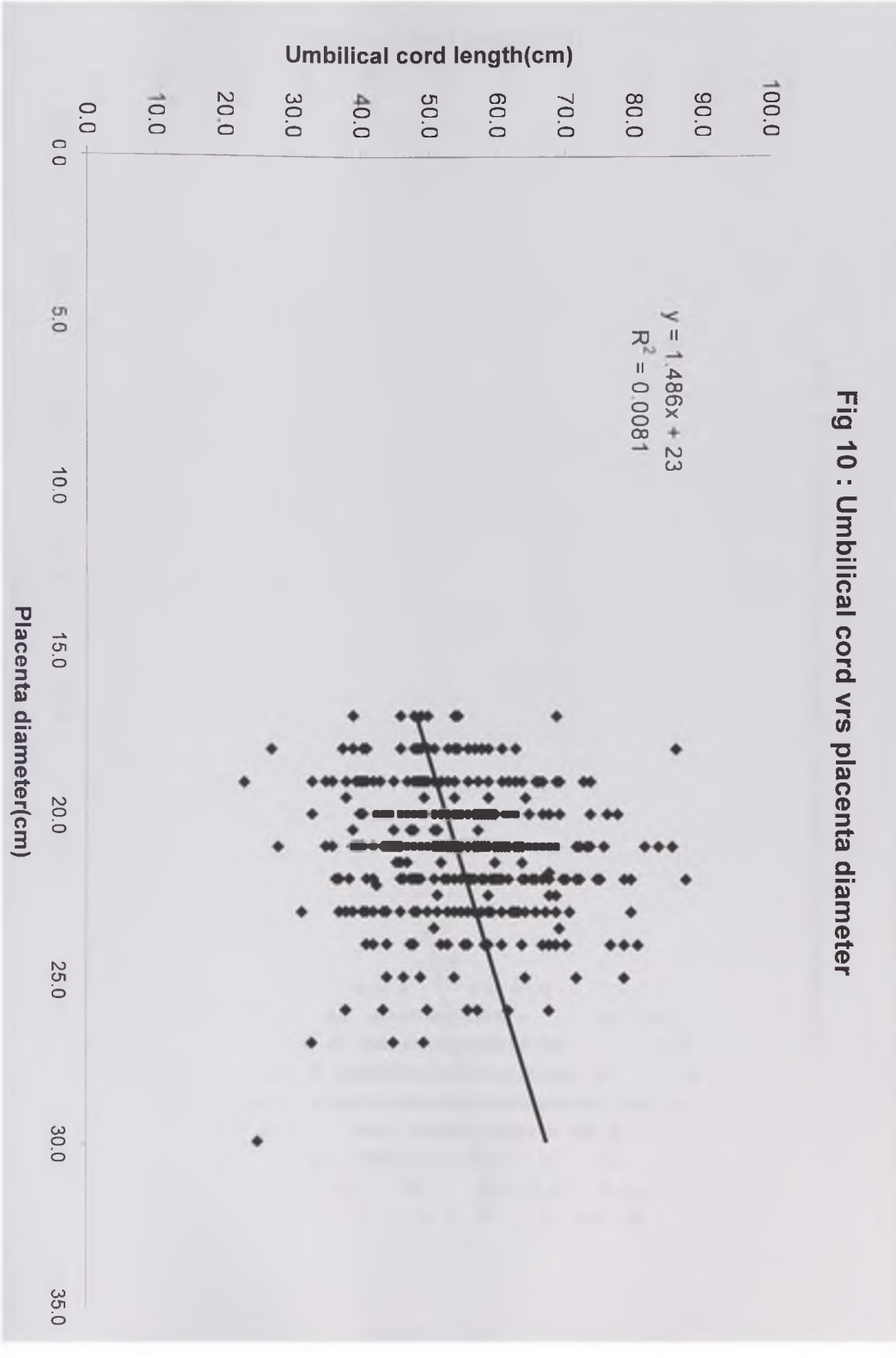

Fig 10 : Umbilical cord vrs placenta diameter

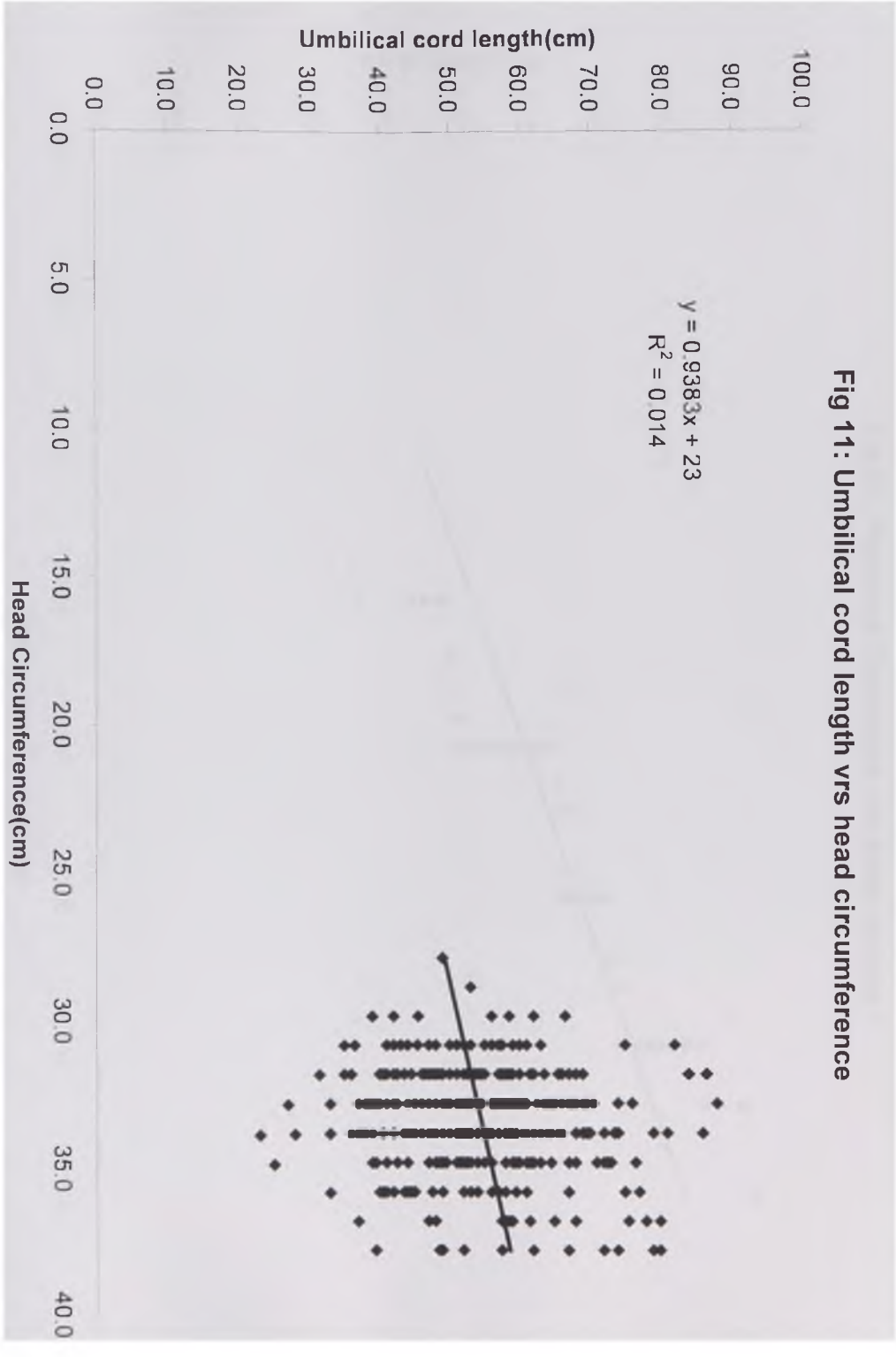

Fig 11: Umbilical cord length vrs head circumference

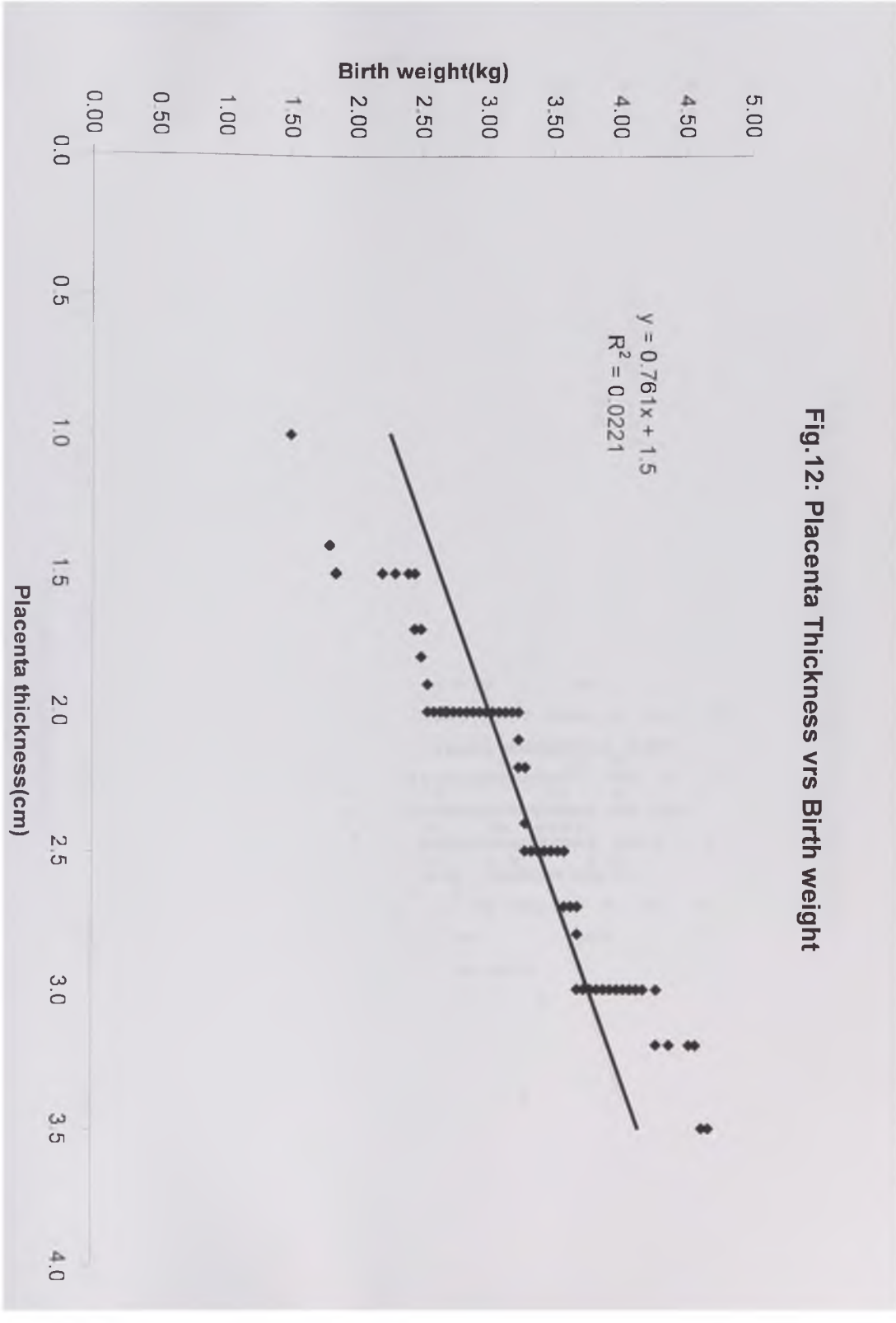

Fig.12: Placenta Thickness vrs Birth weight

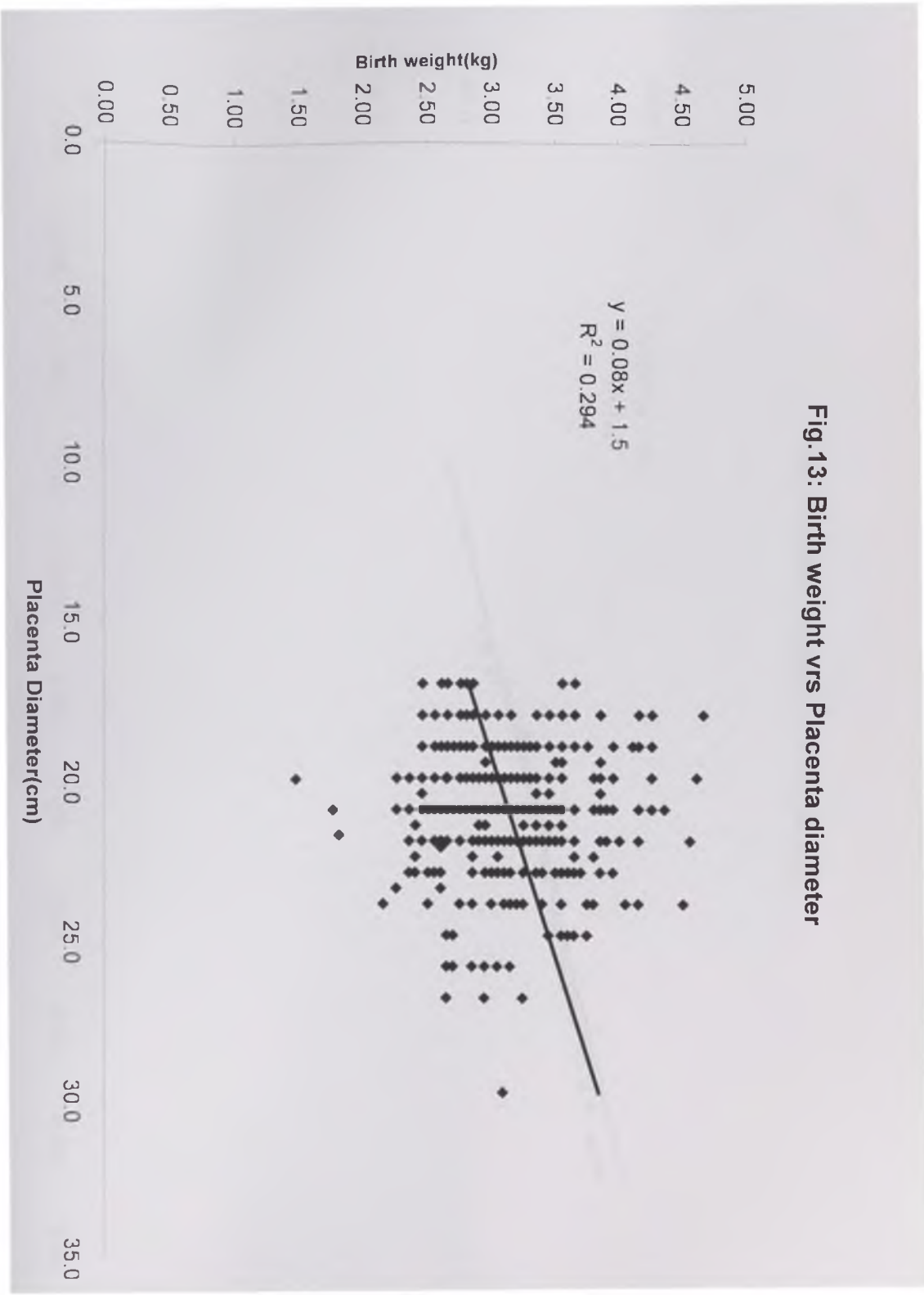

Fig.13: Birth weight vrs Placenta diameter

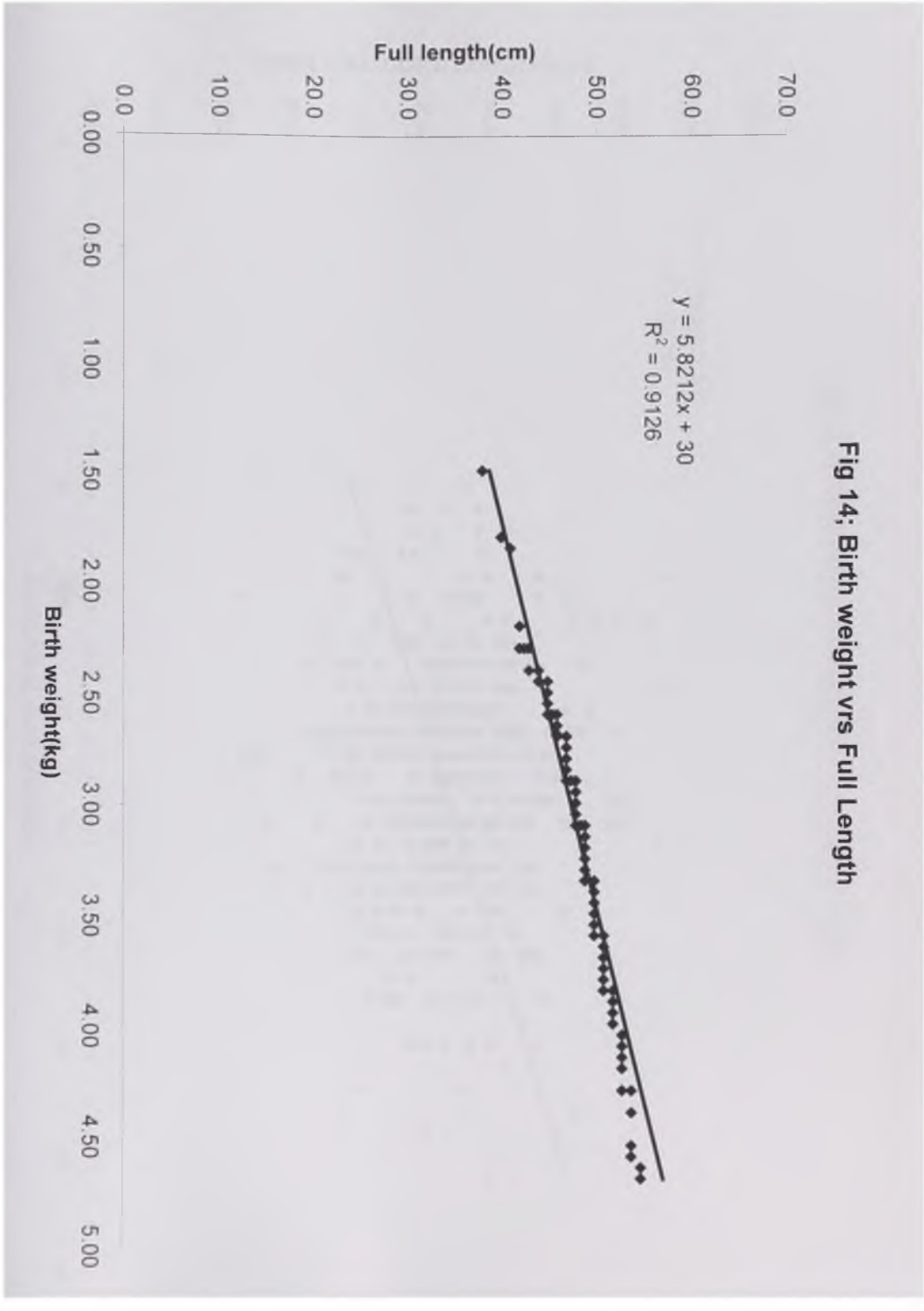

Fig 14; Birth weight vrs Full Length

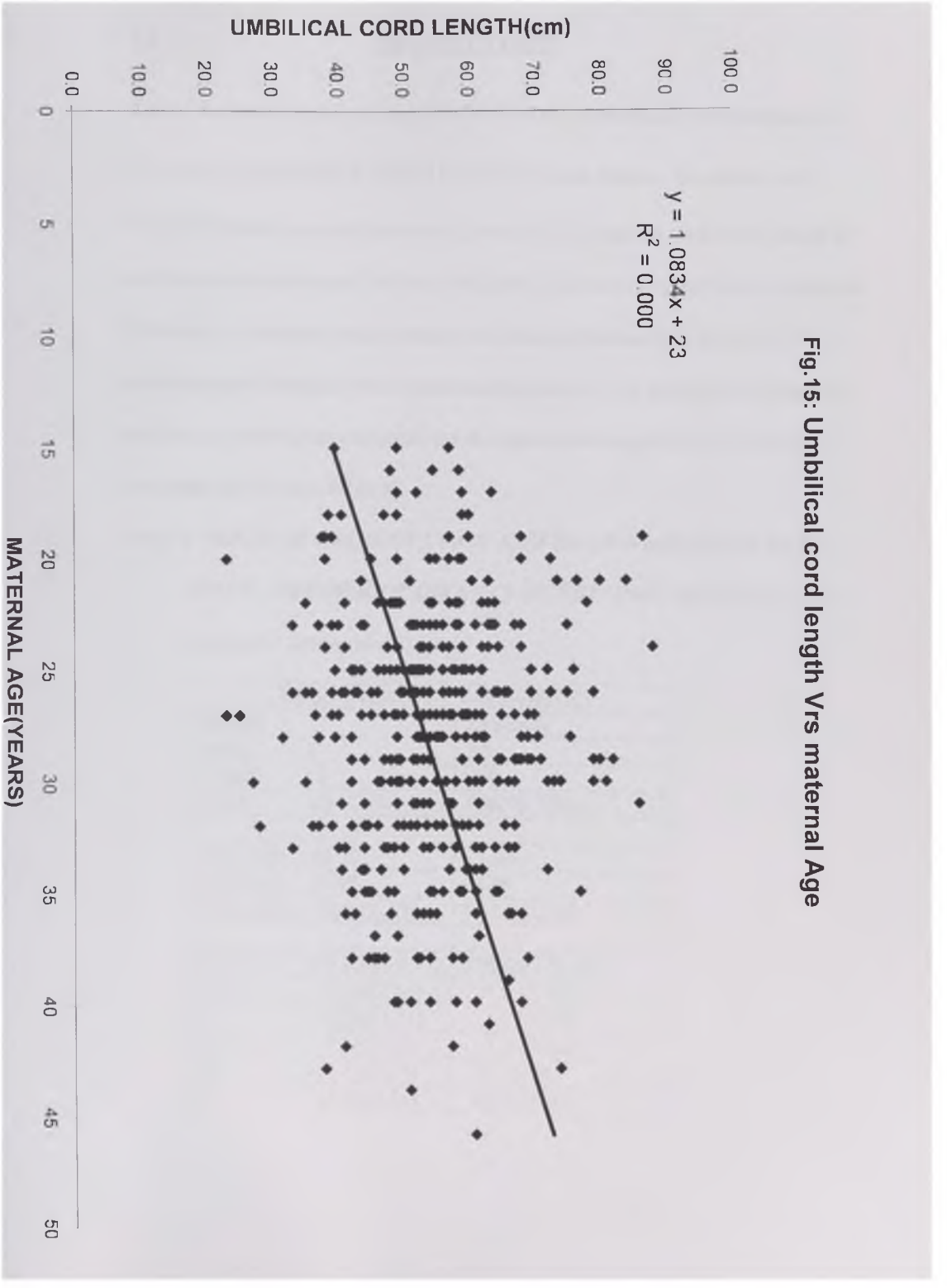

Fig.15: Umbilical cord length Vrs maternal Age

3.2 PRESENTATION.

Out of the total **424** deliveries, 406 (95.75%) of the neonates were cephalic in their mode of presentation while 18 (4.25%.) were breech . The mean cord length for breech presentation was 54.14±6.85(1*Sd*) and the mean cord length for cephalic presentation was 54.56±10.96(1*Sd*). There was no statistically significant difference in the mean cord lengths in the mode of presentation ($p>0.05$). The minimum cord length in breech presentation was 43.5 cm and the maximum was 69.0 cm. In cephalic presentation, the minimum cord length was 23.0 cm and a maximum of 88.0cm (Table 4).

Table 4: **MODE OF PRESENTATION AND MEAN VARIABLES WITH TOTAL NUMBER OF INFANTS OF THE TWO SEXES** (*Standard deviations in brackets*).

PRESENTATION/VARIABLES		
Breech		**Cephalic**
Male:	8	206
Female:	10	200
Total :	18(4.25%)	406(95.75%)
UCL Min	43.50	23.00
UCL Max	69.00	88.00
UCL Mean	54.14(6.85)	54.56(10.96)
PWT	597.22(139.82)	578.64(129.57)
PDIA	21.19(2.46)	21.09(1.94)
PT	2.28(O.31)	2.27(0.40)
BW	3.16(0.59)	3.20(0.50)
HC	34.17(2.31)	33.56(65)
FL	47.94(2.51)	48.70(2.31)

3.3 MODE OF DELIVERY

Out of the total 424 neonates, 312 (68.28%) were delivered by SVDs whose mean cord length was 54.73±11.2cm. 108(25.47%) were delivered by C/S with a mean cord length of 54.03±9.7cm. Four (6.25%) deliveries were by Vacuum Extraction, with a mean cord length of 51.38±7.8cm. There was no statistically significant difference in the mean cord lengths for SVDs and C/S modes of delivery ($p>0.05$) (Table 5).

TABLE 5: **MODE OF DELIVERY AND MEAN CORD LENGTH**

How Delivered	Numbers	Percentage	Mean cord length
SVD	312	68.28	54.73 ± 11.20
C/S	108	25.47	54.03 ± 9.70
VAC	4	6.25	51.38 ± 7.80
OVERALL TOTAL	424	100.00	

TABLE 6a: **MEAN CORD LENGTH IN PARITY GROUPS.**

ANOVA (p-value = 0.580, F = 0.826, (p>0.05).

	Parity							
	0	1	2	3	4	5	6	7
Total	168	121	69	30	22	8	4	2
Mean cord length(cm)	54.3	55.6	55.4	53.6	48.4	54.3	56.8	44.0
Standard deviation	(10.74)	(11.8)	(9.0)	(9.7)	(11.5)	(9.2)	(16.0)	(0.0)

Table 6b. **Comparison of mean cord length between primipara (p0) and multipara (p4+p5+p6+p7).**

Parity	Minimum(cm)	Maximum(cm)	Mean ± (1SD)
Primipara N=168	27.0	86.5	54.33±10.74
Multipara N=36	25.0	78.0	60.97±11.46

A calculated t-test using a Welch correction, gave t = 0.485, p = 0.7126, (p>0.05). There is no significant difference in the mean cord lengths between primipara and multipara in the study.

3.4 POSITION OF CORD ON PLACENTA

The cords on the placenta were accentric or eccentrically located in 370 (87.3 %) out of the total 424 placentas examined. The umbilical cords of 38 (8.9%) of the placentas were centric whereas in the remaining 16 (3.8 %) placentas, the cords were peripheral (marginal) There was no observed velamentous cords insertion in this study. The distributions of the position of the cord are shown in Table 7.

An analysis of variance (one-way ANOVA) gave an F-value of 0.645 and a $p = 0.525$ which was not significant. Thus it appears that there is no association between cord length and cord position on the placenta.

Table 7. **INCIDENCE OF THE POSITION OF UMBILICAL CORDS ON PLACENTAS.**

Position on placenta	Number	Percentage	Mean cord length
Centric	38	8.9	52.63±8.6
Accentric	370	87.3	54.66±10.9
Marginal/peripheral	16	3.8	55.68±12.4
Total	424	100.0	

3.5 FURCATE AND NON-FURCATE CORDS.

Furcate umbilical cord were observed in 68 (16.04%) cases, while the remaining 356 (83.96%) were non-furcate. Table 8 shows descriptive statistics of both types of cords with mean values and one standard deviation (in brackets).

By an independent sample test (assuming equal variance), the means of the cord lengths, placenta weights, placenta diameter, placenta thickness, and birth weight were tested statistically for significance. There were no significant differences between in any pairs of variables ($p>0.05$). There were no observed bleeding in deliveries involving furcate cords as compared with non-furcate cords.

TABLE 8: **RESULTS OF TEST OF SIGNIFICANT DIFFERENCES BETWEEN FURCATE AND NON-FURCATE CORDS.**

Cord vessel separation	Total observed (%)	Mean UCL (cm)	Mean PWt (kg)	Mean PT (cm)	Mean PDIA (cm)	Mean BWt (kg)
FURCATE	68 (16.04%)	55.75 (11.61)	569.85 (146.38)	2.35 (0.44)	21.46 (2.42)	3.27 (0.54)
NON-FURCATE	356 (83.96%)	54.28 (10.65)	580.76 (126.83)	2.25 (0.39)	21.02 (1.86)	3.19 (0.45)
TOTAL	424 (100%)	P = 0.30 NS	P = 0.52 NS	P = 0.07 NS	P = 0.09 NS	P = 0.20 NS

3.6 EXPERIMENTAL FINDINGS

A true knot of the umbilical cord was observed in this study in a Spontaneous vaginal delivery (see plate 10). The cord length was 48.5cm and the baby weighed 3.45kg and delivered alive.

There were 5 stillbirths (babies born dead) in the series with a mean cord length of 57.4cm.They were made up of 3 females and 2 males. 419 out of the 424 were born alive.

Umbilical cord entanglements, especially around foetal neck, were observed in 10% of the deliveries without observed risk to the live of baby. It was observed that some cords showed spiralisation whiles others did not. The tendency of the cord to prolapse was observed in three deliveries (two were through C/S with footling breech complications and one SVD). The cord lengths associated with these cases were (68.0 cm, 49.5 cm and 64.0 cm) belonging to a male and two females respectively.

The number of formed cotyledons or lobules (NOC) were subjectively counted in 126 (29.7%) out of 424 complete placentas, ranging between 9 and 22 cotyledons, whereas in 298(70.3%) the cotyledons could not be counted. This was because the maternal surfaces of these placentas were either highly lobulated or had incomplete sulci.

CHAPTER FOUR

DISCUSSION

4.1 VARIATIONS IN THE LENGTHS OF THE UMBILICAL CORD

The results obtained from the measurements of umbilical cord lengths in this study, confirm previous findings (Walker and Pye, 1960; Malpas, 1964; Purola, 1968; Agboola, 1978; and Nnatsu, 1991), that the length of the human umbilical cord at birth varies widely.

This compares closely with the ranges and means of cord lengths reported by earlier investigators in other countries.

Agboola, (1978) obtained a range of 20 to 100 cm and a mean cord length of 57.48 cm for a sample size of 538 umbilical cords. Nnatsu, (1991) reported a range of 34 to 105cm and a mean of 58.39 ± 12.02 cm for a sample size of 661. Walker and Pye, (1960) with a relatively smaller sample size of 177, reported a range of 18 to 122 cm and a mean of 54.0cm. Malpas, (1964) gave a range of cord lengths at or near term to be 30 to 129cm and a mean of 61.0 ± 10.0 cm for a sample of 538. Purola, (1968) obtained a range of 22 to 130cm and a mean of 59.0 ± 12.0cm for a relatively larger sample size of 1,713 umbilical cords.

The pattern of the frequency distribution of the umbilical cords when plotted into a histogram (Fig 3), appears to follow closely the normal Gaussian curve. It can be seen that there is a rise in frequency of the cord length from 23.0 cm to a peak, followed by a gradual fall to a maximum length of 88.0 cm.

These observations compare favourably to those made by Walker and Pye, (1960) and Malpas,(1964) who studied non- African populations and that of Agboola, (1978) and Nnatsu, (1991) who studied typical African populations.

The assertion (Agboola, 1978 and Nnatsu, 1991), that human umbilical cord length does not vary with race, when they compared their results with those of Walker and Pye, (1960) and Malpas, (1964) whose samples were Caucasian, might be a correct one. Perhaps a comparison between cord lengths of humans and other mammalian species, might reveal some differences.

4.2 SEXUAL DIFFERENCES IN UMBILICAL CORD LENGTH

Some investigators (Walker and Pye, 1960, Sornes & Bakke 1986, Wu *et al*., 1996) have reported some observed sexual differences in the length of the human umbilical cord. Walker and Pye, (1960) measured the lengths of 177 cords, and found that the 87 male cords had a mean cord length of 56.4 ± 1.68 cm (SE) while the 90 female cords had a mean cord length of 52.1 ± 1.24 cm (SE). Though Walker and Pye,(1960) did not test the statistical significance of their observed sexual differences in cord length, it was clear from the comparison of mean cord lengths that male infants had about 4.0 cm longer cords than female infants.

Sornes & Bakke (1986) reported similar observation from a very large sample size of 9,601 infants. A calculated mean difference of 1.56

cm in vertex presentation and 1.26 cm in breech presentation in favour of the males ($p<0.01$) was found.

Wu *et al.*, (1996) have observed sexual differences in mean cord lengths in 1,087 deliveries in Taiwan. They reported that males had slightly longer cords than their female counterparts ($p<0.001$).

In this study, a similar result was obtained with mean cord lengths in male infants being slightly higher than female infants. The observed difference of about 2.0 cm was not statistically significant (Table 2).

A non-significant statistical result does not mean that there is no such difference but only that data are compatible with there being no such difference (Altman *et al.*, 1983).

One may support from the hypotheses of Miller *et al.*, (1981) and Mossenger *et al.*, (1982) that male foetuses were probably more hyperactive during pregnancy, and therefore might have applied greater tension on their umbilical cords leading to their relatively longer cords.

The observed differences in the mean measurement of the BWt, FL, and HC in which male babes were significantly heavier, taller and had wider head circumferences than female babes (Table 2) appear to confirm the reports of Thomson *et al.*, (1969). According to Thomson *et al.*, (1969) the weight of male and female foetuses are practically the same at about 32 weeks but then the males thereafter grow slightly faster than the females. Probably, differences in endocrine constitution might have affected the growth rate.

Thomson *et al.*, (1969) have reported that birth weight and placenta weight tend to be slightly greater in male than in female infants. This was not the case in this study, because although males were heavier than females, mean placenta weights favoured the females. Probably, the gross weight of the placenta has no bearing on its functional activity.

4.3 UMBILICAL CORD LENGTH AND FOETAL PRESENTATION

Umbilical cords have been found to be longer in vertex or cephalic presentation, and relatively shorter in breech (Walker and Pye, 1960, Sornes and Bakke, 1986, Wu *et al.*,1996). In the present study, the mean cord length in cephalic presentations was found to be 54.56 ± 10.96cm compared with 54.14 ± 6.86 cm in breech (Table 4). This shows a slight difference of 0.42 cm longer in cephalic than in breech.

In this study, an observation of the range of cord lengths in both presentations appears to show some difference. In breech, the range was between a minimum of 43.5 cm and a maximum of 69.0 cm (a difference of 25.5cm) compared with a minimum of 23.0cm to a maximum of 88.0cm in cephalic (a difference of 65.0cm).

Walker and Pye,(1960) observed that in four cases of breech presentation, the mean cord length was 44.5 cm compared with 54.3 cm in vertex (cephalic) in 173 cases. Sornes and Bakke, (1986) and Wu *et al.*, (1996) with their large sample sizes of 9,601 and 1,087 respectively linked breech presentation with relatively shorter cords.

Foetuses with relatively shorter cords probably might have difficulty in turning round prior to delivery in breech presentations whereas in cephalic, longer cord lengths presumably allow the foetuses to turn easily and to present with the head during foetal descent.

Clinically, cephalic presentation is more common since it is easier for the head of the foetus to lie in the narrow lower pole of the uterus than the bulky breech, which can find more space in the fundus of the uterus. It is therefore not suprising to observe an incidence of 95.75% for cephalic presentation in the study compared with 4.25% for breech, out of 424 deliveries (Table 4).

4.4 POSITION OF UMBILICAL CORDS ON PLACENTAS

The results (Table 7) indicate that umbilical cords are commonly positioned accentrically on placentas (Plate 2). This observation appears to confirm previous reports Krone *et al.*, (1964), Purola, (1968), Uyanwah-Akpom and Fox, (1977).

The 87.3% incidence of accentric, 8.9% for centric and 3.8% for peripheral could be compared with the observations of Addai *et al.*, (1994), who reported an overall incidence of 74% for a combined centric/ accentric cord insertion , and 26% for marginal/peripheral cords out of 121 sampled umbilical cords. The differences in incidence could be attributed to the relatively larger sample size (424) in this study.

The 3.8% occurrence of peripheral/marginal cord position in the study, compared with incidences of 2% and 15% reported by Fox, (1978) in a review of data from seven different sources, appears to fall within the range. However, the occurrence of marginal position fell outside the range quoted by Robinson *et al.*,(1983) of 5.9 to 10.3%. Their data included all placentas delivered at a particular hospital in five consecutive years. Perhaps the race of the mothers in the study, who were all African negroes, may be important in the interpretation of the observed differences in prevalence of marginal cord insertions.

Fox, (1978) attributed the wide variations in the reported incidence of marginal cord positions on placentas to the different interpretations of the rather subtle distinction between an extremely accentric and a marginal or peripheral positioned umbilical cords. Similarly, Purola, (1968) suggested that the difficulty in separating a centric cord from an accentric cord could lead to different reports of incidence. Thus, different people observing a structure at different times could be one factor that has led to different figures being quoted at different times.

Though marginal cords have been clinically linked to various anomalies in the literature (Brody and Frenkel, 1953; Raaflaub, 1959; Monie,1965) there is no possibility of reconciling this present finding with that of Brody and Frenkel (1953) that about 70% of placentas with marginal cord positions were from women who had suffered a premature onset of labour.

4.5 UMBILICAL CORD LENGTH AND ITS POSITION ON PLACENTA

From the results in Table 7, the mean cord length on the placentas appears to indicate that when the cord is positioned at the margin.

This implies that there was no association between umbilical cord length and the position of the cord on the placenta surface.

Purola,(1968) statistically analysed 1,713 umbilical cords at various positions on the placenta and found that there was no positive correlation between cord length and its position on the placenta.

Probably, the human umbilical cord grows in length irrespective its site or position on the placenta.

4.6 CORD LENGTH IN PARITY GROUPS

This study found revealed no statistically significant differences ($p>0.05$) in the mean cord lengths of babies born to the different parity groups (Table 6a). This is contrary to the report of Sornes *et al.*, (1989) that babies of primiparous mothers have comparatively shorter mean cord lengths compared with those of multiparous mothers.

Probably, parity has no major influence on the length of the umbilical cord. This observation is in agreement with those of Agboola, (1978), Nnatsu, (1991). It stands to reason that first time mothers have the same chance of giving birth to a baby with a relatively longer or shorter cord as multiparous mothers.

The relatively large numbers of primiparous mothers in the sample (Table 6a) appear to show that first time mothers deliver more at the hospitals compared with multiparous ones. This is due to effective antenatal care and family planning education given at the hospitals. Inversely, multiparous mothers appear to deliver at the private maternity homes than the big hospitals hence their low numbers encountered in the study.

4.7 FURCATE AND NON-FURCATE UMBILICAL CORDS.

The point prevalence of furcate and non-furcate umbilical cords (Plates 4 & 5 respectively) observed in the study, appears to show that the non-furcate condition occurs more frequently in umbilical cords delivered at term (gestation > 36weeks). Hence the observed high incidence of 83.96% out of 424 umbilical cords and 16.06% for furcate type (Table 8).

Addai *et al.*, (1994) reported an incidence of 68% for non-furcate and 32% for furcate cords out of 121 cords observed. Clinically, a furcate cord with the umbilical vessels exposed a few centimeters before inserting on the placenta is highly susceptible to damage because of excessive foetal movements. A damaged umbilical vessel could lead to a fatal foetal haemorrhage (Kesseler, 1960). The difference in the prevalence of furcate and non-furcate cords thus suggests a morphological advantage of the non-furcate type over the furcate. Perhaps if the type of cord (whether furcate or non-furcate) could be ascertained earlier in pregnancy, appropriate obstetric management could be given in furcate cases, which are relatively susceptible to vessel damage.

Addai *et al.*, 1994, stereologically, found that the trophoblastic and syncytial knot components grew faster in placentas with furcate cords leading to the congestion of the uterus, with the placenta becoming heavy in some pregnancies and presumably leading to early parturition.

In the present study, only the gross weights of the placentas were taken and this was not statistically significant. There were no significant

differences in the birth weights, placenta thickness, placenta diameter, and umbilical cord lengths in both types of umbilical cords (Table 8).

Probably, the dimensions of the placenta, birth weight, and the length of the cord, are not influenced by the morphological form of the cord (i.e. furcate or non-furcate).

The thickness of the placenta has been reported to be about 2.5cm and about 20.0cm in diameter on the average by many investigators (Boyd and Hamilton, 1970; O'Rahilly, 1973; Johannigmann *et al.*, 1972).

In this study, the thickness of the placenta was measured at the points of cord insertion on the placenta. The minimum thickness of the placentas measured was 1.0 cm and the maximum value was 3.5 cm with a mean value of 2.27 cm (Table 1). Perhaps the thickness of the placenta at the point of insertion on the placenta may be important in the foeto-maternal exchange and not the morphological form of the cord. However, this was not the original area of investigation and only a future detailed study could reveal some useful information.

The widest placenta diameter fell within the range of 17.0 cm and 30.0 cm with an average of 21.0 cm (Table 1). Appearing to conform to recorded standard measurements at term (Bernischke and Kaufmann,1990).

4.8 CORRELATES OF THE LENGTH OF THE HUMAN UMBILICAL CORD

Table 3, shows a generally low correlation between umbilical cord lengths measured at birth with some foetal variables. Some observed results correspond with similar reports of Malpas (1964), Agboola (1978) and Nnatsu, (1991) but differ from the findings of Walker and Pye, (1960) who did not find any correlation between cord length and foetal variables.

In this study, there was low positive correlation between cord length and placenta weight (Pearson's correlation coefficient (r) = 0.250), and between cord length and birth weight (r= 0.228).

Malpas, (1964) reported a Pearson's correlation coefficient (r) = 0.253 between umbilical cord length and placenta weight and (r = 0.254) between cord length and birth weight. Agboola, (1978) obtained (r =0.250) for cord length and placenta weight and an (r =0.218) for cord length and birth weight.

Of the related placental dimensions recorded and foetal variables (i.e. placenta thickness, placenta diameter, placenta weight, baby's full length, and baby's head circumference), only placenta diameter showed a non-significant correlation with cord length. According to Daly *et al.*, (1991) statistical significance obtained in a study could be explained in two main ways:

1). That is if a larger number were studied, similar results to those obtained in the smaller study actually carried out would be expected.

Or

2.) perhaps the sampling variation was not sufficient to explain the observed results.

The above points are supported by this study because although a smaller sample size was involved in the present study compared to those of Malpas, (1964), Agboola, (1978) and Nnatsu, (1991), a similar statistical significance ($p< 0.05$) was however obtained. On the other hand, one could also observe small sample variations in the measurements of placenta thickness, baby crown-heel lengths, head circumference and baby weight which could have affected the observed significant results (SD on Table1).

The negative correlation between cord length and parity ($r = - 0.062$) is statistically non-significant, ($p> 0.05$). Since the correlation was non-significant, it might be ignored because cord length does not decrease or increase with increasing parity of the mother. The non-significant finding of the mean cord length in the parity groups (Table 6a), confirms that cord length has no inverse relationship with maternal parity.

The R^2 values which are the square of the r-values give a ready interpretation in terms of the strength of a relationship that exists between two continuous variables (Table 3 and Fig4 to Fig15) (Daly *et al.*, 1991). Thus an R^2 value of 0.0623 as in fig 2 between umbilical cord length and placenta weight suggests for example, that variation in placenta weight

explain just about 6% of the total variation in cord length. The remaining 94% of the cord length variations are unexplained and might be due to many other factors not considered in the analysis. Figure 5 also shows that about 29% of the variability of birth weights could be explained by placenta weight (R^2 value of 0.2902).

According to Sanin *et al.*, (2001) together with gestational age and maternal age, placenta weight could explain 32% of the variability of birth weight.

An R^2 value of 0.0529 between cord length and birth weight (fig.6), implies that the variation in birth weight could explain just 5% of the total variation in cord lengths. The R^2 values in Table 3, further confirm the seemingly low correlation that exist between the factors considered in the study.

Umbilical cord length in this study correlated significantly with baby full length ($r = 0.234$, $p < 0.01$ Table 3). This observation contradicts the report of Agboola, (1978) who reported that the correlation between cord length and the crown-heel length at birth was non- significant ($r = 0.0878$, $p > 0.0001$).

The low correlation between cord length and foetal and placental dimensions that was interpreted by Malpas, (1964) that the human umbilical cord grows in one dimension independent of other structures of embryonic growth (the foetus and the placenta) as a possible explanation

is debatable. This is because correlation co-efficient values are not causative and their use to explain biological phenomina are inconclusive.

From the results and graph representations, it seem likely that foetal weight and placenta weight for instance, could probably be predicted if the umbilical cord length can be measured during intrauterine life by the use of a marker and ultrasonography as has been reported by Nnatsu, (1991). This is an area that requires further research.

The present study confirms that placenta weight and birth weight are correlated (Table 3 & Fig 5). This may mean no more than that a small foetus tends to have a small placenta. The cause of the smallness of the foetus may be genetic or some external influence such as uteroplacental circulation of low capacity (Thomson *et al.*, 1969). The moderate correlation between placenta weight and birth weight Table 3 & Fig.5 is in agreement with the findings of Thomson *et al.*, 1969, Younoszai and Haworth, 1969, and Sanin *et al.*, 2001 that placenta weight and size are known to increase as birth weight increases. Various conclusions seem to depend mainly on data being interpreted. According to Thomson *et al.*, (1969) birth weight is predicted rather poorly by placenta weight with an r – value often between 0.5 to 0.6 at term. In this work, an r-value of 0.539 was obtained. Probably, the gross placenta weight is a poor indicator of placenta adequacy as has been stated already. However, its importance as a "sentinel" indicator of

nutritional and/ or environmental problems cannot be ignored (Sanin *et al.*, 2001).

A moderate correlation between baby's birth weight and its full length is to be expected (Table 3 & Fig.14), since normally a heavy baby is more likely to be taller, but as has been mentioned earlier, other factors could account for the correlation. Correlation between birth weight and head circumference is also expected in a normal baby.

4.9 TRUE KNOTS

The observed true knot in the study (Plate 10) although a single case, clearly indicates how a cord can become knotted and be a risk to the baby if the cord vessels were to be occluded.

The male baby whose cord was knotted was delivered alive by SVD to a mother who had already given birth to three children. The length of the cord was 48.5.0 cm and the baby weighed 3.45 kg.

It therefore implies that a true knot, when formed, could be carried through pregnancy till delivery at term without fatality to the foetus. The number of children of the mother could suggest that the uterus might have been spacious to allow the formation of the knot.

The five stillbirths encountered in the study cannot be attributed solely to their umbilical cords being extremely short or

extremely long, since some other factors might have caused their death.

The observation of three cord prolapse cases during the survey whose mean cord length was 60.5 cm compared with the overall mean cord length of 54.5 cm, suggests that when the cord is relatively long there is the possibility of it prolapsing. In this study, two out of the three cases with cord prolapse had to undergo emergency C/S. This observation supports the idea that when the cord prolapses, it may result in an emergency situation, which might require immediate obstetric intervention to save the foetus (Durand *et al.*, 1996).

5.0 SUMMARY AND CONCLUSION

The major objective of this study was to find out whether there were any possible correlations between umbilical cord length and various variables of birth. There were significant positive correlations between umbilical cord length and foetal birth weight($r = 0.228$, $p<0.01$), cord length and placenta weight($r = 0.240$, $p<0.01$), cord length and placenta thickness ($r = 0.118$, $p< 0.05$), cord length and foetal head circumference ($r = 0.121$, $p< 0.05$), and cord length with baby's full length ($r = 0.234$, $p<0.01$).

There were no significant correlations between cord length and maternal age, parity and placenta diameter

Morphologically, the human umbilical cord shows clear variations in its gross features. In length, the cord showed a normal distribution in the population. The cord most often situated a little away from the center of the placenta (accentric). The vessels in the cord could be separated a little distance before inserting on the placenta (Furcate) or remain close together without significant difference in birth outcome.

Recording the morphological findings of the cord, for example True knot formation or the Furcate nature of the cord, could be informative and important in obstetric management and help explain some birth complications.

Further studies on the umbilical cord, using ultrasound guided techniques before birth and histological methods after birth to ascertain the number of vessels in the cord are proposed to provide additional information.

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