

Sudan University of Science&Technology
College Of Graduate Studies

**Curve of Normal Fetal Weight Values Estimated by
Ultrasound According to Gestational Age in Sudan**

منحنى قيم الأوزان الطبيعية للأجنة وفقاً لمراحل الحمل باستخدام

الموجات فوق الصوتية في السودان

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الآية

وَقُلْ رَبِّ زِدْنِي عِلْمًا قَالَ ((

((تعالى

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Dedication

**To my family, friends and colleagues, whom I love and
respect
I dedicate this work**

Acknowledge

the first and last thank to ALLA.

Our all and deep thank to our families for their support, bearing and encouragement .

Deep thank to my supervisor DR. Mona Ahmed Mohammed for his great effort and patience.

Great appreciation to Dr. Mohammed Ahammed Ali Omer and my friends whom pushed me forward and encouraged me .

Abstract

The objective of this study To detect the normal intrauterine fetal growth in pregnant Sudanese women and compared with international intrauterine fetal growth using serial ultrasound investigation .

This was statistical study for pregnant women came to ultrasound department for routine check up ,the study was covered 150 cases between the age (14-45)years .

This results shows the correlation between the mother Height in cm and Fetus weight in Kg . It reveals that: the mother Height has no correlation with Fetus weight which are insignificance ($R^2 = 0.03$). in(Figure 1), shows the correlation between the mother weight in Kg and Fetus weight in Kg . It reveals that: the mother weight has no correlation with Fetus weight which are insignificance ($R^2 = 0.03$). in (Figure 2), shows the correlation between the BMI (Kg/m^2) versus fetal weight standard and measured. It reveals that: the fetal weight has been increases as the BMI increases in forms of linear correlation which are insignificance ($R^2 = 0.05$ and 0.02) for the influence of BMI in

measured fetal weight and standard one respectively in(Figure 3), shows the correlation between the GA in weeks and versus measured parameters (FL, AC and BPD) in cm. It reveals that: the GA has has been increases as measured parameters (BPD ,FL, and AC) increases in forms of linear correlation which are significance ($R^2 = 0.692, 0.33, 0.768$) for the influence of GA in measured parameters (BPD ,FL, and AC) respectively in (Figure 4) and shows the correlation between the GA in weeks versus fetal weight standard and measured. It reveals that: the fetal weight has been increases as the GA increases in forms of linear correlation which are significance ($R^2 = 0.996$ and 0.738) for the influence of GA in standard fetal weight and measured one respectively in(Figure 5).

المستخلص

تهدف هذه الدراسة لإكتشاف النمو الطبيعي للأطفال داخل الرحم للنساء الحوامل السودانيات

. ومقارنتها مع الأوزان المعيارية للأجنة داخل الرحم باستخدام الموجات فوق الصوتية

إعتمدت الدراسة علي المسح الروتيني للنساء الحوامل من عمر 14-45 سنة ل 150 حالة

أوضحت نتيجة الدراسة أن ليس هنالك علاقة بين أطوال الأمهات وأوزان الأجنة وكان معامل

الارتباط (0.03) في مخطط رقم 1

وأيضا ليس هنالك علاقة بين أوزان الأمهات وأوزان الأجنة معامل الارتباط (0.03) في مخطط

(رقم 2)

كما أوضحت أن هناك علاقة طردية بين معدل كتلة الجسم للأمهات وأوزان الأجنة المعيارية

والمقاسة على التوالي في مخطط رقم 3

معامل الارتباط (0.02 and 0.05)

وبينت أن هناك علاقة طردية بين أعمار الأجنة بالأسابيع والمعايير المقاسة (مقاس الرأس , طول

(عظمة الفخذ ومحيط البطن) على التوالي في مخطط رقم 4)

معامل الارتباط (0.692, 0.33, 0.768)

وأوضحت النتيجة الأساسية أن هناك علاقة طردية بين أعمار الأجنة بالأسابيع وأوزانها

المعيارية والمقاسة معامل الارتباط (0.738 - 0.996

(في مخطط رقم 5)

Contents

الآية	I
Dedication	II
Acknowledgement	III
Abstract	IV
Arabic abstract	V
Contents	VI
List of figures	IX
Abbreviation	XI
Chapter one: Introduction	
Introduction	2
Chapter two: Literature review	
2.1fetal anatomy :-	5
2-1-1Skeletal System	4
2-1-2 Muscular System	5
2-1-3 Body Cavities	7
2-1-4 Cardiovascular System	8
2-1-5 Respiratory System	11
2-1-6 Digestive System	12
2-1-7 Urogenital System	14
2-1-8 Head and Neck	16
2-1-9 Ear	18
2-1-10 Eye	19
2-1-11 Integumentary System	19
2-1-12 Central Nervous System	20

2-2 Fetal pathology	23
2.2.1 Causes of Slow Fetal Growth in the Third Trimester	23
2.2.1 .1 Maternal Factors	23
2.2.1 .2 Placental Factors	24
2.2.1 .3 Fetal Factors	24
2.2.2 Causes of Fetal loss in the Scand Trimester:	24
2.2.3 The third Trimester of Pregnancy Weight Gain¹⁹	25
2.2.4 Weight gain should I expect in the second trimester:	26
2.3 Fetal growth	26
2.3 .1 Defining growth restriction	28
2.3.1 Symmetric growth restriction	29
2.3 .2 A Symmetric growth restriction	29
2.3 Fetal measuring parameters	30
2.3.1 Measuring the biparietal diameter (BPD)	32
2.3.1.1 Measuring the BPD from the lateral ventricles view	32
2.3 .1.2 Problems	34
2.3 .1.3 Skull shape	36
2.3 .2 Measuring the abdominal circumference (AC)	38
2.3.2 .1 Method	38
2.3 .2 .2 Problems	38
2.3.3 Measuring the femur length	42
2.3 .3 .1 Method	42

2.3.3.2 Problems	43
2.4 Evaluation amniotic fluid volume	45
2.5 Confirming or assign gestational age	46

Chapter three: Material and method	
3.1 material and method	41
Chapter four: Result	
Result	56
Chapter five	
Discussion and conclusion and recommendation	
5.1 Discussion	61
5.2 Conclusion	63
5.3 Recommendation	64
Reference	65
Appendixes	67

List of Figures

Figure	Topic	Bag NO
Figure3.1	A. Mild cardiomegaly. B. Hyperechoic small bowel in a 30-weeks fetus with severe intrauterine growth restriction.	21
Figure 3.2	Transverse section of the fetal head demonstrating the landmarks required to measure the BPD using the lateral ventricles view	24
Figure3.3	Transverse section of the fetal head demonstrating the landmarks required to measure theBPD using the thalami view	25
Figure3.4	Transverse section of the fetal head withthe callipers placed on the outer border of both theproximal and distal parietal bones (diameter 1)	27
Figure 3.5	Measurement of the abdominal Circumference using the two diameter method	33
Figure 3.6	Transverse section of the fetal abdomendemonstrating the landmarks required to measure theabdominal circumference	34
Figure 3.7	Measurement of the fetal femur. Note thatsoft tissue is visible beyond both ends of the bone	

		37
Figure4.1	the correlation between the mother Height in cm and Fetus weight in Kg.	44
Figure4.2	the correlation between the mother weight in Kg and Fetus weight in Kg	45
Figure4.3	the correlation between the BMI (Kg/m ²) versus fetal weight standard and measured	46
Figure4.4	the correlation between the GA in weeks versus measured parameters (FL, AC and BPD) in cm	47
Figure4.5	the correlation between the GA in weeks versus Fetal weight standard and measured	48

Abbreviation

FL	femur length
AC	abdominal circumference
BBD	Bi Barital diameter
GA	gestational age
MH	Maternal height
MW	Maternal weight
BMI	Body mass index
FW	Fetal weight

Chapter One

Introduction

Chapter one

1.1 Introduction

Many researchers have attempted to estimate fetal weight using single or combined ultrasound measurements of the fetus. Knowledge of expected birth weight is attractive to clinicians as it is an important variable affecting perinatal mortality. Fetal weight estimation is thought to be helpful in predicting fetal survival and making management decisions in the very low birth weight group (< 1000 g) and in managing the delivery of the large baby, where complications may occur.

The most successful early approach was a simple correlation between abdominal circumference (AC) and birth weight. Numerous further attempts have combined measurements in regression equations or volumetric formulae, with varying degrees of accuracy. Several of these methods have insignificant systematic errors, but random errors (as measured by the standard deviation of errors) of less than about 7% are rarely reported. John Wiley & Sons, 2004

The aim of this study was to estimate the normal fetal weight according to gestational age in Sudanese pregnant women .John Wiley & Sons, 2004

1.2 The problem:-

To generate Sudanese graph to determine weight for gestational age

1.3 General objective:-

To detect normal intrauterine growth using serial us investigation

1.4 Specific objective:-

- To compare Sudanese fetus weight with international intrauterine growth curve
- To determine the prevalence of IUGR IN Khartoum

1.5 The rationale and importance of the study:-

To facilitate for a physician a direct assessment & to determine weight for gestational age.

Chapter two

Literature review

Chapter two

Literature review

2.1fetal anatomy:-

2-1-1Skeletal System

The skeletal system develops from mesenchyme, which is derived from the mesodermal germ layer and from neural crest. Some bones, such as the flat bones of the skull, undergo membranous ossification; that is, mesenchyme cells are directly transformed into osteoblasts. In most bones, such as the long bones of the limbs, mesenchyme condenses and forms hyaline cartilage models of bones . Ossification centers appear in these cartilage models, and the bone gradually ossifies by endochondral ossification. The skull consists of the neurocranium and viscerocranium (face). The neurocranium includes a membranous portion, which forms the cranial vault, and a cartilaginous portion (chondrocranium), which forms the base of the skull. Neural crest cells form the face, most of the cranial vault, and the prechordal part of the chondrocranium (the part that lies rostral to the notochord). Paraxial mesoderm forms the remainder of the skull.

Limbs form as buds along the body wall that appear in the fourth week.

Lateral plate mesoderm forms the bones and connective tissue, while muscle cells migrate to the limbs from the somites. The AER regulates limb outgrowth, and the ZPA controls anteroposterior patterning. Many of the genes that regulate limb growth and patterning have been defined.

The vertebral column and ribs develop from the sclerotome compartments of the somites, and the sternum is derived from mesoderm in the ventral body wall. A definitive vertebra is formed by condensation of the caudal half of one sclerotome and fusion with the cranial half of the subjacent sclerotome.

The many abnormalities of the skeletal system include vertebral (spina bifida), cranial (cranioschisis and craniosynostosis), and facial (cleft palate) defects.

Major malformations of the limbs are rare, but defects of the radius and

digits are often associated with other abnormalities (syndromes). (Sadler (

2-1-2 Muscular System

Most muscles arise from the mesoderm. Skeletal muscles are derived

from paraxial mesoderm, including (a) somites, which give rise to muscles

of the axial skeleton, body wall, and limbs, and (b) somitomeres, which give rise to muscles of the head. Progenitor cells for muscle tissues are derived from the dorsolateral and dorsomedial portions of the somites. Cells in the dorsolateral portion express *MYO-D* and migrate to form hypomeric muscle; cells in the dorsomedial portion express *MYF5*, migrate ventral to the dermatome to form the myotome, and ultimately form epimeric musculature. (Sadler(

By the fifth week muscle precursor cells are divided into a small dorsal portion, the epimere, innervated by a dorsal primary

ramus, and a larger ventral portion, the hypomere, innervated by a ventral primary ramus. Myoblasts from epimeres form extensor muscles of the vertebral column, while those of the hypomere form limb and body wall musculature. Connective tissue derived from somites, somatic mesoderm, and neural crest (head region) provide a template for establishment of muscle patterns. Most smooth muscles and cardiac muscle fibers are derived from splanchnic mesoderm. Smooth muscles of the pupil, mammary gland, and sweat glands differentiate from ectoderm. (Sadler)

2-1-3 Body Cavities

At the end of the third week, intercellular clefts appear in the mesoderm on

each side of the midline. When these spaces fuse, the intraembryonic cavity (body cavity), bordered by a somatic mesoderm and a splanchnic mesoderm layer. With cephalocaudal and lateral folding of

the embryo, the intraembryonic cavity extends from the thoracic to the pelvic region. Somatic mesoderm will form the parietal layer of the serous membranes lining the outside of the peritoneal, pleural, and pericardial cavities. The splanchnic layer will form the visceral layer of the serous membranes covering the lungs, heart, and abdominal organs. These layers are continuous at the root of these organs in their cavities (as if a finger were stuck into a balloon, with the layer surrounding the finger being the splanchnic or visceral layer and the rest of the balloon, the

somatic or parietal layer surrounding the body cavity). The serous membranes in the abdomen are called peritoneum.

The diaphragm divides the body cavity into the thoracic and peritoneal

cavities. It develops from four components: (a) septum transversum (central tendon); (b) pleuroperitoneal membranes; (c) dorsal mesentery of the esophagus; and (d) muscular components of the body wall. Congenital

diaphragmatic hernias involving a defect of the pleuroperitoneal membrane on the left side occur frequently. (Sadler, chapter 10, Body Cavities, 223)

The thoracic cavity is divided into the pericardial cavity and two pleural

cavities for the lungs by the pleuropericardial membranes.

Double layers of peritoneum form mesenteries that suspend the gut tube and provide a pathway for vessels, nerves, and lymphatics to the organs. Initially, the gut tube from the caudal end of the foregut to the end of the hindgut is suspended from the dorsal body wall by dorsal mesentery. Ventral mesentery derived from the septum transversum exists only in the region of the terminal part of the esophagus, the stomach, and upper portion of the duodenum (Sadler (

2-1-4 Cardiovascular System

The entire cardiovascular system—heart, blood vessels, and blood

cells—originates from the mesodermal germ layer. Although initially

paired, by the 22nd day of development the two tubes form a single, slightly bent heart tube consisting of an inner endocardial tube and a surrounding myocardial mantle. During the 4th to 7th weeks the heart divides into a typical four-chambered structure. (Sadler (

Septum formation in the heart in part arises from development of endocardial cushion tissue in the atrioventricular canal (atrioventricular cushions) and in the conotruncal region (conotruncal swellings). Because of the key location of cushion tissue, many cardiac malformations are related to abnormal cushion morphogenesis. (Sadler (

Septum Formation in the Atrium. The septum primum, a sickle-shaped crest descending from the roof of the atrium, begins to divide the atrium in two but leaves a lumen, the ostium primum, for communication between the two sides. Later, when the ostium primum is obliterated by fusion of the septum primum with the endocardial cushions, the ostium secundum is formed by cell death that creates an opening in the septum primum. Finally, a septum secundum forms, but an interatrial opening, the oval foramen, persists. Only at birth, when pressure in the left atrium increases, do the two septa press against each other and close the communication between the two. Abnormalities in the atrial septum may vary from total absence to a small opening known as probe patency of the oval foramen.

Septum Formation in the Atrioventricular Canal. Four endocardial cushions

surround the atrioventricular canal. Fusion of the opposing superior and inferior cushions divides the orifice into right and left atrioventricular canals. Cushion tissue then becomes fibrous and forms the mitral (bicuspid) valve on the left and the tricuspid valve on the right . Persistence of the common atrioventricular canal and abnormal division of the canal are well-known defects. (Sadler (

Septum Formation in the Ventricles. The interventricular septum consists of a thick muscular part and a thin membranous portion formed by (a) an inferior endocardial atrioventricular cushion, (b) the right conus swelling, and (c) the left conus swelling . In many cases these three components fail to fuse, resulting in an open interventricular foramen. Although this abnormality may be isolated, it is commonly combined with other compensatory defects . (Sadler (

Septum Formation in the Bulbus. The bulbus is divided into (a) the truncus

(aorta and pulmonary trunk), (b) the conus (outflow tract of the aorta and

pulmonary trunk), and (c) the trabeculated portion of the right ventricle. The

truncus region is divided by the spiral aortopulmonary septum into the two

main arteries . The conus swellings divide the outflow tracts of the aortic and pulmonary channels and with tissue from the inferior endocardial

cushion close the interventricular foramen . Many vascular abnormalities,

such as transposition of the great vessels and pulmonary valvular atresia, result from abnormal division of the conotruncal region; they may involve neural crest cells that contribute to septum formation in the conotruncal region. (Sadler (

The aortic arches lie in each of the five pharyngeal arches.

Four important derivatives of the original aortic arch system are (a) the carotid arteries (third arches); (b) the arch of the aorta (left fourth aortic arch); (c) the pulmonary artery (sixth aortic arch), which during fetal life is connected to the aorta through the ductus arteriosus; and (d) the right subclavian artery formed by the right fourth aortic arch, distal portion of the right dorsal aorta, and the seventh intersegmental artery . The most common vascular aortic arch abnormalities include (a) open ductus arteriosus and coarctation of the aorta and (b) persistent right aortic arch and abnormal right subclavian artery , both causing respiratory and swallowing complaints. (Sadler (

The vitelline arteries initially supply the yolk sac but later form the celiac, superior mesenteric, and inferior mesenteric arteries, which supply the foregut, midgut, and hindgut regions, respectively.

The paired umbilical arteries arise from the common iliac arteries. After birth the distal portions of these arteries are obliterated to form the medial umbilical ligaments, whereas the proximal portions persist as the internal iliac and vesicular arteries. (Sadler ,c h a p t e r 11,Cardiovascular System,274)

Venous System. Three systems can be recognized: (a) the vitelline system, which develops into the portal system; (b) the cardinal system, which forms the caval system; and (c) the umbilical system, which disappears after birth. The complicated caval system is characterized by many abnormalities, such as double inferior and superior vena cava and left superior vena cava .

Changes at Birth. During prenatal life the placental circulation provides the fetus with its oxygen, but after birth the lungs take on gas exchange. In the circulatory system the following changes take place at birth and in the first postnatal months: (a) the ductus arteriosus closes; (b) the oval foramen closes; (c) the umbilical vein and ductus venosus close and remain as the ligamentum teres hepatis and ligamentum venosum; and (d) the umbilical arteries form the medial umbilical ligaments. (Sadler (

Lymphatic System. The lymphatic system develops later than the cardiovascular system, originating as five sacs: two jugular, two iliac, one retroperitoneal, and one cisterna chyli. Numerous channels form to connect the sacs and provide drainage from other structures. Ultimately the thoracic duct forms from anastomosis of the right and left thoracic ducts, the distal part of the right thoracic duct, and the cranial part of the left thoracic duct. The right lymphatic duct develops from the cranial part of the right thoracic duct.(Sadler (

2-1-5 Respiratory System

The respiratory system is an outgrowth of the ventral wall of the foregut, and the epithelium of the larynx, trachea, bronchi, and alveoli originates in the endoderm. The cartilaginous,

muscular, and connective tissue components arise in the mesoderm. In the fourth week of development, the tracheoesophageal septum separates the trachea from the foregut, dividing the foregut into the lung bud anteriorly and the esophagus posteriorly. Contact between the two is maintained through the larynx, which is formed by tissue of the fourth and sixth pharyngeal arches. The lung bud develops into two main bronchi: the right forms three secondary bronchi and three lobes; the left forms two secondary bronchi and two lobes. Faulty partitioning of the foregut by the tracheoesophageal septum causes esophageal atresias and tracheoesophageal fistulas . After a pseudoglandular (5–16 weeks) and canalicular (16–26 weeks) phase, cells of the cuboidal lined bronchioles change into thin, flat cells, type I alveolar epithelial cells, intimately associated with blood and lymph capillaries. In the seventh month, gas exchange between the blood and air in the primitive alveoli is possible. Before birth the lungs are filled with fluid with little protein, some mucus, and surfactant, which is produced by type II alveolar epithelial cells and which forms a phospholipid coat on the alveolar membranes. At the beginning of respiration the lung fluid is resorbed except for the surfactant coat, which prevents the collapse of the alveoli during expiration by reducing the surface tension at the air-blood capillary interface. Absent or insufficient surfactant in the premature baby causes respiratory distress syndrome (RDS) because of collapse of the primitive alveoli (hyaline membrane disease). (Sadler (

Growth of the lungs after birth is primarily due to an increase in the number

of respiratory bronchioles and alveoli and not to an increase in the size of the alveoli. New alveoli are formed during the first 10 years of postnatal life.

(Sadler (

2-1-6 Digestive System

The epithelium of the digestive system and the parenchyma of its derivatives originate in the endoderm; connective tissue, muscular components, and peritoneal components originate in the mesoderm. Differentiation of the gut and its derivatives depends upon reciprocal interactions between the gut endoderm (epithelium) and its surrounding mesoderm. *HOX* genes in the mesoderm are induced by *sonic hedgehog (SHH)* secreted by gut endoderm and regulate the craniocaudal organization of the gut and its derivatives.

The gut system extends from the buccopharyngeal membrane to the

cloacal membrane and is divided into the pharyngeal gut, foregut, midgut, and hindgut. The pharyngeal gut gives rise to the pharynx and related glands .The foregut gives rise to the esophagus, the trachea and lung buds, the stomach, and the duodenum proximal to the entrance of the bile duct. In addition, the liver, pancreas, and biliary apparatus develop as outgrowths of the endodermal epithelium of the upper part of the duodenum . Since the upper part of the foregut is divided by a septum (the tracheoesophageal septum) into the esophagus posteriorly and

the trachea and lung buds anteriorly, deviation of the septum may result in abnormal openings between the trachea and esophagus. The epithelial liver cords and biliary system growing out into the septum transversum differentiate into parenchyma. Hematopoietic cells (present in the liver in greater numbers before birth than afterward), the Kupffer cells, and connective tissue cells originate in mesoderm. The pancreas develops from a ventral bud and a dorsal bud that later fuse to form the definitive pancreas . Sometimes, the two parts surround the duodenum (annular pancreas), causing constriction of the gut . The midgut forms the primary intestinal loop ,gives rise to the duodenum distal to the entrance of the bile duct, and continues to the junction of the proximal two-thirds of the transverse colon with the distal third. At its apex the primary loop remains temporarily in open connection with the yolk sac through the vitelline duct. During the sixth week, the loop grows so rapidly that it protrudes into the umbilical cord (physiological herniation) . During the 10th week, it returns into the abdominal cavity. While these processes are occurring, the midgut loop rotates 270° counterclockwise. Remnants of the vitelline duct, failure of the midgut to return to the abdominal cavity, malrotation, stenosis, and duplications of parts of the gut are common abnormalities.

The hindgut gives rise to the region from the distal third of the transverse

colon to the upper part of the anal canal; the distal part of the anal canal originates from ectoderm. The hindgut enters the posterior region of the cloaca (future anorectal canal), and the

allantois enters the anterior region (future urogenital sinus). Breakdown of the cloacal membrane covering this area provides communication to the exterior for the anus and urogenital sinus. Abnormalities in the size of the posterior region of the cloaca shift the entrance of the anus anteriorly, causing rectovaginal and rectourethral fistulas and atresias (Sadler).

2-1-7 Urogenital System

The urinary and genital systems both develop from mesodermal tissue.

Three urinary systems develop in a temporal sequence from cranial to caudal segments:

The pronephros, which forms in the cervical region, is vestigial.

The mesonephros, which forms in the thoracic and lumbar regions, is large

and is characterized by excretory units (nephrons) and its own collecting duct, the mesonephric or wolffian duct. In the human it may function briefly, but most of the system disappears. Ducts and tubules from the mesonephros form the conduit for sperm from the testes to the urethra. In the female, these ducts regress.

The metanephros, or permanent kidney, develops from two sources. It forms its own excretory tubules or nephrons like the other systems, but its collecting system originates from the ureteric bud, an outgrowth of the mesonephric duct. This bud gives rise to the ureter, renal pelvis, calyces, and the entire collecting system. Connection between the collecting and excretory tubule systems is essential for normal development. *WT1*, expressed by the mesenchyme, makes this tissue

competent to respond to induction by the ureteric bud. Interactions between the bud and mesenchyme occur through production of GDNF and HGF by the mesenchyme with their tyrosine kinase receptors RET and MET, respectively, produced by the ureteric epithelium. PAX2 and WNT4, produced by the ureteric bud, cause epithelialization of the metanephric mesenchyme in preparation for excretory tubule differentiation. Early division of the ureteric bud may lead to bifid or supernumerary kidneys with ectopic ureters. Abnormal positions of the kidney, such as pelvic and horseshoe kidney, are also well known. The genital system consists of (a) gonads or primitive sex glands, (b) genital ducts, and (c) external genitalia. All three components go through an indifferent stage in which they may develop into either a male or a female. The *SRY* gene on the Y chromosome produces testes-determining factor and regulates

Male sexual development. Genes downstream from *SRY* include *steroidogenesis factor (SF1)* and *SOX9* that stimulate differentiation of Sertoli and Leydig cells in the testes. Expression of the *SRY* gene causes (a) development of the medullary (testis) cords, (b) formation of the tunica albuginea, and (c) failure of the cortical (ovarian) cords to develop. In the absence of the *SRY* gene, the combination of *DAX1* expression, to downregulate *SF1*, and continued expression of *WNT4* in the gonadal ridge, causes formation of ovaries with (a) typical cortical cords, (b) disappearance of the medullary (testis) cords, and (c) failure of the tunica albuginea to develop. When primordial germ cells fail to reach the indifferent gonad, the gonad remains indifferent or is

absent. The indifferent duct system and external genitalia develop under the influence of hormones. Testosterone produced by Leydig cells in the testes stimulates development of the mesonephric ducts (vas deferens epididymis),

While MIS produced by Sertoli cells in the testes causes regression of the

paramesonephric ducts (female duct system).

Dihydrotestosterone stimulates development of the external genitalia, penis, scrotum, and prostate. (Sadler (

Estrogens influence development of the paramesonephric female system, including the uterine tube, uterus, cervix, and upper portion of the vagina. They also stimulate differentiation of the external genitalia, including the clitoris, labia, and lower portion of the vagina. Errors in production of or sensitivity to hormones of the testes lead to a predominance of female characteristics under influence of the maternal and placental estrogens. (Sadler, chapter 14, Urogenital System, 362)

2-1-8 Head and Neck

Pharyngeal (branchial) arches, consisting of bars of mesenchymal tissue separated by pharyngeal pouches and clefts, give the head and neck their typical appearance in the fourth week. Each arch contains its own artery, cranial nerve, muscle element, and cartilage bar or skeletal element. Endoderm of the pharyngeal pouches gives rise to a number of endocrine glands and part of the middle ear. In subsequent order the pouches give rise to (a) the middle ear cavity and auditory tube (pouch 1), (b) the stroma of the palatine tonsil (pouch 2), (c) the inferior parathyroid glands

and thymus (pouch 3), and (d) the superior parathyroid glands and ultimobranchial body (pouches 4 and 5) .

Pharyngeal clefts give rise to only one structure, the external auditory meatus. Molecular control of arch development resides in *HOX* genes, whose pharyngeal arch code is carried to the arches by neural crest cells migrating from hindbrain segments known as rhombomeres .This code is then maintained by interactions between crest cells and arch mesoderm.

The thyroid gland originates from an epithelial proliferation in the floor of the tongue and descends to its level in front of the tracheal rings in the course of development.

The paired maxillary and mandibular prominences and the frontonasal prominence are the first prominences of the facial region. Later, medial

and lateral nasal prominences form around the nasal placodes on the frontonasal prominence. All of these structures are important, since they determine, through fusion and specialized growth, the size and integrity of the mandible, upper lip, palate, and nose.

Formation of the upper lip

occurs by fusion of the two maxillary prominences with the two medial nasal prominences .The intermaxillary segment is formed by merging of the two medial nasal prominences in the midline.

This segment is composed of (a) the philtrum, (b) the upper jaw component, which carries the four incisor teeth, and (c) the palatal component, which forms the triangular primary palate.

The nose is derived from (a) the frontonasal prominence, which forms the bridge, (b) the medial nasal prominences, which

provide the crest and tip, and (c) the lateral nasal prominences, which form the alae . Fusion of the palatal shelves, which form from the maxillary prominences, creates the hard (secondary) and soft palate. A series of cleft deformities may result from partial or incomplete fusion of these mesenchymal tissues, which may be caused by hereditary factors and drugs (diphenylhydantoin). (Sadler ,h a p t e r 15,Head and Neck,401)

The adult form of the face is influenced by development of paranasalsinuses, nasal conchae, and teeth. Teeth develop fromepithelial mesenchymal interactions between oral epithelium and neural crest derived mesenchyme. Enamel is made by ameloblasts . It lies on a thick layer of dentin produced by odontoblasts, a neural crest derivative. Cementum is formed by cementoblasts, another mesenchymal derivative found in the root of the tooth. The first teeth (deciduous teeth, or milk teeth) appear 6 to 24 months after birth, and the definitive or permanent teeth, which supplant the milk teeth, are formed during the third month of development .(Sadler(

2-1-9 Ear

The ear consists of three parts that have different origins, but that function

as one unit. The internal ear originates from the otic vesicle, which

in the fourthweek of development detaches from surface ectoderm. This vesicle divides into a ventral component, which gives rise to the saccule and cochlear duct, and a dorsal component, which gives rise to the utricle, semicircular canals,

and endolymphatic duct .The epithelial structures thus formed are known collectively as the membranous labyrinth. Except for the cochlear duct, which forms the organ of Corti, all structures derived from the membranous labyrinth are involved with equilibrium.

The middle ear, consisting of the tympanic cavity and auditory tube,

is lined with epithelium of endodermal origin and is derived from the first

pharyngeal pouch. The auditory tube extends between the tympanic cavity and nasopharynx. The ossicles, which transfer sound from the tympanic membrane to the oval window, are derived from the first (malleus and incus) and second (stapes) pharyngeal arches.

The external auditory meatus develops from the first pharyngeal cleft and

is separated from the tympanic cavity by the tympanic membrane (eardrum). (Sadler)

The eardrum consists of (a) an ectodermal epithelial lining, (b) an intermediate layer of mesenchyme, and (c) an endodermal lining from the first pharyngeal pouch.

The auricle develops from six mesenchymal hillocks along the first and second pharyngeal arches. Defects in the auricle are often associated with other congenital malformations.(Sadler (

2-1-10 Eye

The eyes begin to develop as a pair of outpocketings that will become the optic vesicles on each side of the forebrain at the end

of the fourth week of development. The optic vesicles contact the surface ectoderm and induce lens formation. When the optic vesicle begins to invaginate to form the pigment and neural layers of the retina, the lens placode invaginates to form the lens vesicle. Through a groove at the inferior aspect of the optic vesicle, the choroid fissure, the hyaloid artery (later the central artery of the retina) enters the eye . Nerve fibers of the eye also occupy this groove to reach the optic areas of the brain. The cornea is formed by (a) a layer of surface ectoderm, (b) the stroma, which is continuous with the sclera, and (c) an epithelial layer bordering the anterior chamber . *PAX6*, the master gene for eye development, is expressed in the single eye field at the neural plate stage. The eye field is separated into two optic primordia by *SHH*, which up-regulates *PAX2* expression in the optic stalks while down-regulating *PAX6*, restricting this gene's expression to the optic cup and lens. Epithelial-mesenchymal interactions between prospective lens ectoderm, optic vesicle, and surrounding mesenchyme then regulate lens and optic cup differentiation .(Sadler)

2-1-11 Integumentary System

The skin and its associated structures, hair, nails, and glands, are derived from surface ectoderm. Melanocytes, which give the skin its color, are derived from neural crest cells, which migrate into the epidermis. The production of new cells occurs in the germinative layer. After moving to the surface, cells are sloughed off in the horny layer . The dermis, the deep layer of the skin, is derived from lateral plate mesoderm and from dermatomes of the

somites. .(Sadler) Hairs develop from downgrowth of epidermal cells into the underlying dermis. By about 20 weeks, the fetus is covered by downy hair, lanugo hair, which is shed at the time of birth. Sebaceous glands, sweat glands, and mammary glands all develop from epidermal proliferations. Supernumerary nipples (polythelia) and breasts (polymastia) are relatively common . (Sadler (

2-1-12 Central Nervous System

The CNS originates in the ectoderm and appears as the neural plate at the middle of the third week . After the edges of the plate fold, the neural folds approach each other in the midline to fuse into the neural tube .The cranial end closes approximately at day 25, and the caudal end closes at day 27. The CNS then forms a tubular structure with a broad cephalic portion, the brain, and a long caudal portion, the spinal cord. Failure of the neural tube to close results in defects such as spina bifida and anencephaly ,defects that can be prevented by folic acid.

The spinal cord, which forms the caudal end of the CNS, is characterized

by the basal plate containing the motor neurons, the alar plate for the sensory neurons, and a floor plate and a roof plate as connecting plates between the two sides . *SHH* ventralizes the neural tube in the spinal cord region and induces the floor and basal plates. Bone morphogenetic proteins 4 and 7, expressed in nonneural ectoderm, maintain and up-regulate expression of *PAX3* and *PAX7* in the alar and roof plates.

The brain, which forms the cranial part of the CNS, consists originally of three vesicles: the rhombencephalon (hindbrain), mesencephalon (midbrain), and prosencephalon (forebrain).

The rhombencephalon is divided into (a) the myelencephalon, which forms the medulla oblongata (this region has a basal plate for somatic and visceral efferent neurons and an alar plate for somatic and visceral afferent neurons) , and (b) the metencephalon, with its typical basal (efferent) and alar (afferent) plates . This brain vesicle is also characterized by formation of the cerebellum , a coordination center for posture and movement, and the pons, the pathway for nerve fibers between the spinal cord and the cerebral and the cerebellar cortices .

The mesencephalon, or midbrain, resembles the spinal cord with its basal

efferent and alar afferent plates. The mesencephalon's alar plates form the

anterior and posterior colliculi as relay stations for visual and auditory reflex

centers, respectively . (Sadler)

The diencephalon, the posterior portion of the forebrain, consists of a thin roof plate and a thick alar plate in which the thalamus and hypothalamus develop . It participates in formation of the pituitary gland, which also develops from Rathke's pouch . Rathke's pouch forms the adenohypophysis, the intermediate lobe, and pars tuberalis, and the diencephalon forms the posterior lobe, the neurohypophysis, which contains neuroglia and receives nerve fibers from the hypothalamus.

The telencephalon, the most rostral of the brain vesicles, consists of two lateral outpocketings, the cerebral hemispheres, and a median portion, the lamina terminalis . The lamina terminalis is used by the commissures as a connection pathway for fiber bundles between the right and left hemispheres . The cerebral hemispheres, originally two small outpocketings , expand and cover the lateral aspect of the diencephalon, mesencephalon, and metencephalon . Eventually, nuclear regions of the telencephalon come in close contact with those of the diencephalon. The ventricular system, containing cerebrospinal fluid, extends from the lumen in the spinal cord to the fourth ventricle in the rhombencephalon, through the narrow duct in the mesencephalon, and to the third ventricle in the diencephalon. (Sadler)

By way of the foramina of Monro, the ventricular system extends from the third ventricle into the lateral ventricles of the cerebral hemispheres. (Sadler)

Cerebrospinal fluid is produced in the choroid plexus of the third, fourth, and lateral ventricles. Blockage of cerebrospinal fluid in the ventricular system or subarachnoid space may lead to hydrocephalus.

The brain is patterned along the anteroposterior (craniocaudal) and

dorsoventral (mediolateral) axes. *HOX* genes pattern the anteroposterior axis in the hindbrain and specify rhombomere identity. Other transcription factors containing a homeodomain pattern the anteroposterior axis in the forebrain and midbrain

regions, including *LIM1* and *OTX2*. Two other organizing centers, the anterior neural ridge and the rhombencephalic isthmus, secrete FGF-8, which serves as the inducing signal for these areas. In response to this growth factor, the cranial end of the forebrain expresses *BF1*, which regulates development of the telencephalon, and the isthmus expresses *engrailed* genes that regulate differentiation of the cerebellum and the roof of the midbrain. As it does throughout the central nervous system, SHH, secreted by the prechordal plate and notochord, ventralizes the forebrain and midbrain areas. Bone morphogenetic proteins 4 and 7, secreted by nonneural ectoderm, induce and maintain expression of dorsalizing genes. (Sadler, chapter 19, Central Nervous System, 479)

Most muscles arise from the mesoderm. Skeletal muscles are derived

from paraxial mesoderm, including (a) somites, which give rise to muscles of the axial skeleton, body wall, and limbs, and (b) somitomeres, which give rise to muscles of the head. Progenitor cells for muscle tissues are derived from the dorsolateral and dorsomedial portions of the somites. Cells in the dorsolateral portion express *MYO-D* and migrate to form hypomeric muscle; cells in the dorsomedial portion express *MYF5*, migrate ventral to the dermatome to form the myotome, and ultimately form epimeric musculature. (Sadler).

By the fifth week muscle precursor cells are divided into a small dorsal portion, the epimere, innervated by a dorsal primary ramus, and a larger ventral portion, the hypomere, innervated by

a ventral primary ramus. Myoblasts from epimeres form extensor muscles of the vertebral column, while those of the hypomere form limb and body wall musculature. Connective tissue derived from somites, somatic mesoderm, and neural crest (head region) provide a template for establishment of muscle patterns. Most smooth muscles and cardiac muscle fibers are derived from splanchnic mesoderm. Smooth muscles of the pupil, mammary gland, and sweat glands differentiate from ectoderm. (Sadler)

2-2 Fetal pathology:-

2.2.1 Causes of Slow Fetal Growth in the Third Trimester

2.2.1 .1 Maternal Factors

The main maternal cause of IUGR is high blood pressure, causing around one third of all cases of IUGR. Other maternal factors associated with IUGR include kidney disease, diabetes, lung or heart disease. Cigarette smoking during pregnancy may cause IUGR, and the risk increases the more a mother smokes. Alcohol consumption, poor diet, low maternal weight and poor weight gain also are implicated in IUGR, along with young maternal age, poverty, recent pregnancy and high number of previous births. (Sharon Perkins,2015)

2.2.1 .2 Placental Factors:

The placenta supplies nutrients to the fetus and removes waste products, so diseases that interfere with its functioning, such as high blood pressure, diabetes and kidney disease can cause IUGR. Other causes of decreased blood flow include a placenta that

implants too low on the uterine wall, part or all of the placenta detaching prematurely from the uterine wall and infection.

(Sharon Perkins,2015)

2.2.1 .3 Fetal Factors

Genetic and chromosomal defects can cause IUGR in a fetus. Fetal exposure to infections, which include toxoplasmosis, rubella, cytomegalovirus and herpes simplex or varicella can lead to IUGR. Prenatal infections often have a poor long term prognosis. Multiple pregnancies also increase the risk of IUGR -- identical twins who share a placenta develop twin to twin transfusion syndrome, where one twin receives too much of the blood supply and one receives too little, developing IUGR.(Sharon Perkins,2015)

2.2.2 Causes of Fetal loss in the Scand Trimester:

Conditions Associated with Intrauterine Growth

RetardationMedicalChronic hypertension Preeclampsia early in gestation Diabetes mellitus Systemic lupus erythematosusChronic renal diseaseInflammatory bowel diseaseSevere hypoxic lung diseaseMaternalSmokingAlcohol useCocaine useWarfarin (Coumadin, Panwarfin)Phenytoin (Dilantin) THOMAS,2007

-Malnutrition

Prior history of pregnancy with intratuterie growth retardation

Residing at altitude above 5,000 feet

-Infectious

-Syphilis

-Cytomegalovirus

-Toxoplasmosis

- Rubella
- Hepatitis B
- HSV-1 or HSV-2
- HIV-1
- Congenital
- Trisomy 21
- Trisomy 18
- Trisomy 13
- Turner's syndrome. THOMAS,2007

2.2.3 The third Trimester of Pregnancy Weight Gain

- fat stores
- fluids
- more blood
- amniotic fluid
- a larger uterus
- the placenta.)Michele Drehmer,.etal/_2013)

Complications of too much weight gain can show up during the third trimester, and may include:

- premature birth (baby is born at 37 weeks or earlier)
- gestational diabetes
- high blood pressure
- Baby is born large.)Michele Drehmer,.etal/_2013)

2.2.4 Weight gain should I expect in the second trimester:

At the start of the second trimester, your baby weighs nearly 1.5 ounces. When you reach the end of this trimester, they'll weigh almost 2 pounds. That's a lot of growth in a few months. The rate

of growth will only increase in your next trimester. (Rachel Nall 2016)

The increase in your baby's weight will cause an increase in your own weight. Your body will continue to increase your blood and fluid volume, which adds weight. Soon, you will start to feel your baby move. (Rachel Nall 2016)

The amount of weight you can expect to gain during the second trimester will vary based on your pre-pregnancy weight. Your doctor should calculate your body mass index (BMI) early in your pregnancy. Based on your BMI, your doctor can estimate how much weight you should gain. According to the Institute of Medicine, women who are:

- underweight, or have a BMI under 18.5, should gain 28-40 pounds
- normal weight, or have a BMI between 18.5-24.9, should gain 25-35 pounds
- overweight, or have a BMI between 25-29.9 , should gain 15-25 pounds
- obese, or have a BMI over 30, should gain 11-20 pounds

If you were very sick in your first trimester of pregnancy, you may have lost weight or your weight may have stayed the same. You may gain weight in the second trimester to compensate for this loss.

Your doctor will weigh you and estimate your baby's weight with each monthly visit. Ask them if you're concerned you're gaining too much or too little weight. (Rachel Nall 2016)

2.3 Fetal growth:-

Abnormalities of fetal growth are encountered far more frequently in clinical practice than fetal abnormalities. Fetal growth restriction, whatever the cause, is associated with a wide variety of adverse outcomes, from minor neonatal morbidity to intrauterine fetal death. The most common reason for fetal growth restriction is uteroplacental insufficiency. Chromosome abnormalities, some fetal abnormalities and fetal infection can be associated with a reduction in growth velocity of varying severity. It is therefore important to be aware of the ultrasound features that aid in distinguishing between uteroplacental insufficiency, fetal abnormality and fetal infection as possible causes for fetal growth restriction. Many ultrasound features are associated with uteroplacental insufficiency, and these will vary depending on the severity and duration of the problem.

Early features include a reduction in growth velocity and oligohydramnios followed by a reduced biophysical profile, mild cardiomegaly, hyperechoic bowel and bowel dilatation. Doppler studies typical of uteroplacental insufficiency include uterine artery notches, absent/reversed end-diastolic flow in the umbilical artery and arterial redistribution. Similar features are seen in fetal abnormality or infection, but usually the uterine artery Doppler values and the amniotic fluid volume are normal (unless the urinary tract is involved). In these situations, consideration should be given to prenatal diagnosis of chromosomal abnormality or congenital viral infection. (Elsevier 2004)

The relative frequency of fetal growth restriction and the understandable anxiety it causes in both parents and medical carers necessitates a thorough understanding of the differential diagnosis and management of this condition.

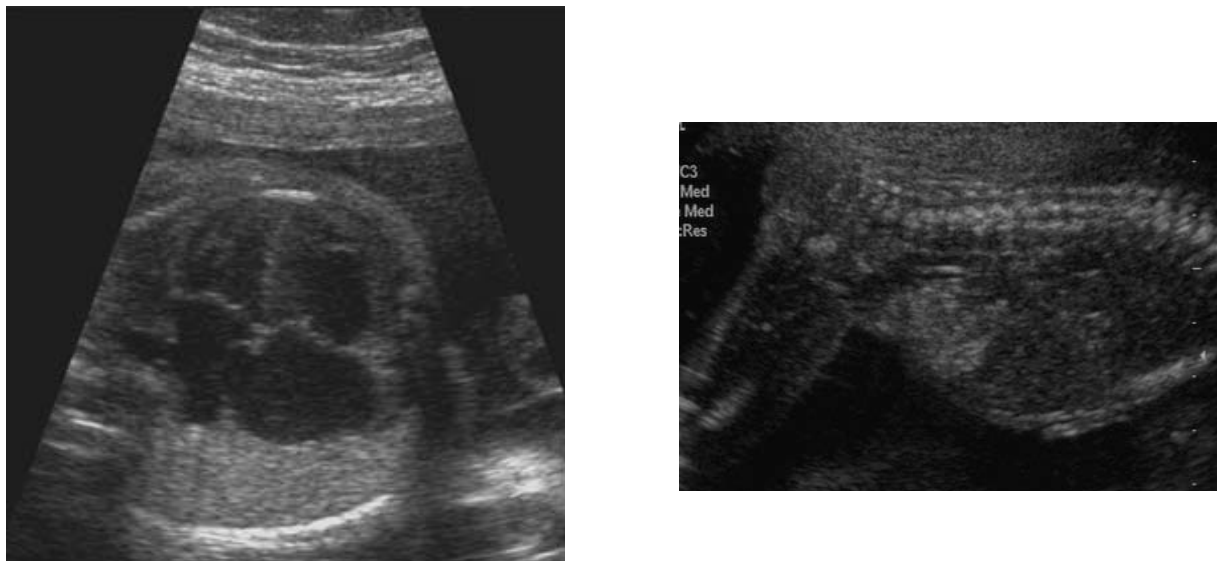


Figure 3.1 A. Mild cardiomegaly. B. Hyperechoic small bowel in a 30-weeks fetus with severe intrauterine growth restriction.
(Elsevier 2004)

2.3 .1 Defining growth restriction :-

Intrauterine growth restriction (IUGR) is often defined as birthweight less than the 10th centile for gestation. However, this simple definition has many drawbacks. First, 10% of the normal population will be identified by this classification. Second, despite being growth-restricted, the majority of fetuses affected by uteroplacental insufficiency have biometry measurements that plot above the 10th centile. (Elsevier 2004)

It would seem better to use individualized growth charts to determine whether a fetus is growth restricted . These are based primarily on the mother's height, weight, parity and the birthweights of her previous children.

For example, a fetus that weighs 2.9 kg at 39 weeks is probably growth restricted if the mother is 6 feet tall, weighs 65 kg and has had a previous child weighing 4.2 kg at birth. Unfortunately, such individualized or customized antenatal growth charts are not in common usage, and they remain to be validated in large prospective studies. (Elsevier 2004)

A simpler alternative would be to use growth velocity to determine the effect of uteroplacental function. For example, a fetus with a falling growth velocity that is crossing centile lines is likely to be compromised, whereas another that has a consistent growth rate on the 5th centile is probably constitutionally small. However, this still remains a subjective assessment because there is no agreed rate of fall or threshold for determining pathologic from physiologic pregnancies. Before growth restriction can be diagnosed by any of the methods described above, correct pregnancy dating is required. Incorrect or unknown pregnancy dating will result in an increased diagnosis of growth restriction and post-term pregnancy, both of which lead to unnecessary pregnancy intervention (Elsevier 2004)

2.3.1 Symmetric growth restriction

Symmetric growth restriction is the description given to the equivalent reduction in growth velocity of both the fetal head circumference and abdominal circumference.

In the vast majority of cases, the H:A ratio would be in the normal range and this picture would represent a constitutionally small fetus. In a very small number of pregnancies, symmetric growth restriction occurs in pathological pregnancies. In the latter case, the insult or injury has occurred in early pregnancy and results in severe growth restriction and very poor pregnancy outcome .

(Elsevier 2004)

2.3 .2 A Symmetric growth restriction

Asymmetric growth restriction is the description given to differential reduction in growth velocity of the fetal head to abdominal circumference . In the vast majority of cases, asymmetric fetal growth restriction is a consequence of uteroplacental insufficiency. Ultrasound features of fetal growth restriction include placental abnormality (lakes, calcification, jelly-like consistency) and reduction in amniotic fluid volume. Fetal features include mild cardiomegaly, hyperechoic bowel and small bowel dilatation.

The presence of these features would support the diagnosis of uteroplacental insufficiency . (Elsevier 2004)

2.3 Fetal measuring parameters:-

2.3.1 Measuring the biparietal diameter (BPD)

The BPD has traditionally been the most widely used ultrasound parameter in the estimation of gestational age. Although more recent data suggest that head circumference (HC) should be used in preference to BPD for dating purposes, the BPD is easy to obtain and, on a routine basis, is more accurate than the crown-

rump length. A single optimal measurement of the BPD will predict the gestational age to within ± 5 days.

This last point has justified its use in all pregnancies. The BPD is the maximum diameter of a transverse section of the fetal skull at the level of the parietal eminences. The BPD, occipitofrontal diameter (OFD) and head circumference can be measured from one of the following two sections:

- *Lateral ventricles view*: the correct section is demonstrated in, and should include

the following features:

- a rugby-football-shaped skull, rounded at the back (occiput) and more pointed at the front (sinciput)
- a long midline equidistant from the proximal and distal skull echoes
- the cavum septum pellucidum bisecting the midline one-third of the distance from the sinciput to the occiput
- the two anterior horns of the lateral ventricles, symmetrically placed about the midline
- all or part of the posterior horns of the lateral ventricles symmetrically placed about the midline. In earlier gestations (15–20 weeks), (Elsevier 2004)

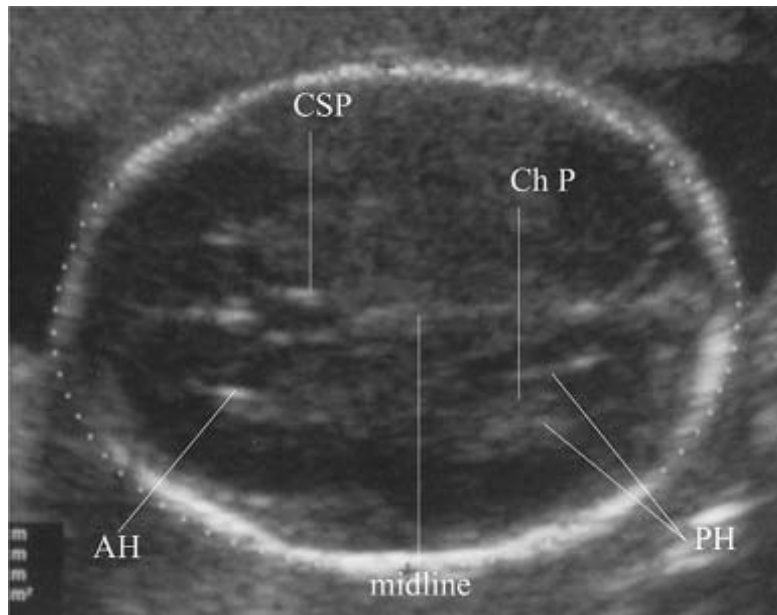


Figure 3.2 Transverse section of the fetal head demonstrating the landmarks required to measure the BPD using the lateral ventricles view. Note the rugby football shape, the centrally placed midline, the presence and position of the cavum septum pellucidum (CSP), and the appearance and position of the anterior horns (AH) of the lateral ventricles. Note the choroid plexus (ChP) within the distal posterior horn (PH) of the lateral ventricle and reverberation causing visualization of the proximal posterior horn. (Elsevier 2004)

The optimal view of the posterior horn is usually obtained in this section (see below). At later gestations (20–24 weeks), the optimal section for visualizing the posterior horn is slightly lower than the BPD section.

● **Thalami view:** include the following features:

● a rugby-football-shaped skull, rounded at the back (occiput) and more pointed at the front (synciput)

- a short midline equidistant from the proximal and distal skull echoes
- the cavum septum pellucidum bisecting the midline one-third of the distance from the sinciput to the occiput
- The thalami
- **The basal cisterns.**

There is no consensus as to which section is preferable.

We recommend using the lateral ventricles view because this enables the anterior and posterior horns of the lateral ventricles to be examined and the head measurements to be taken from the same section. However, the thalami view is the section of choice in the American literature and in many departments in the United Kingdom. The BPD and HC measurements obtained from both sections are comparable.

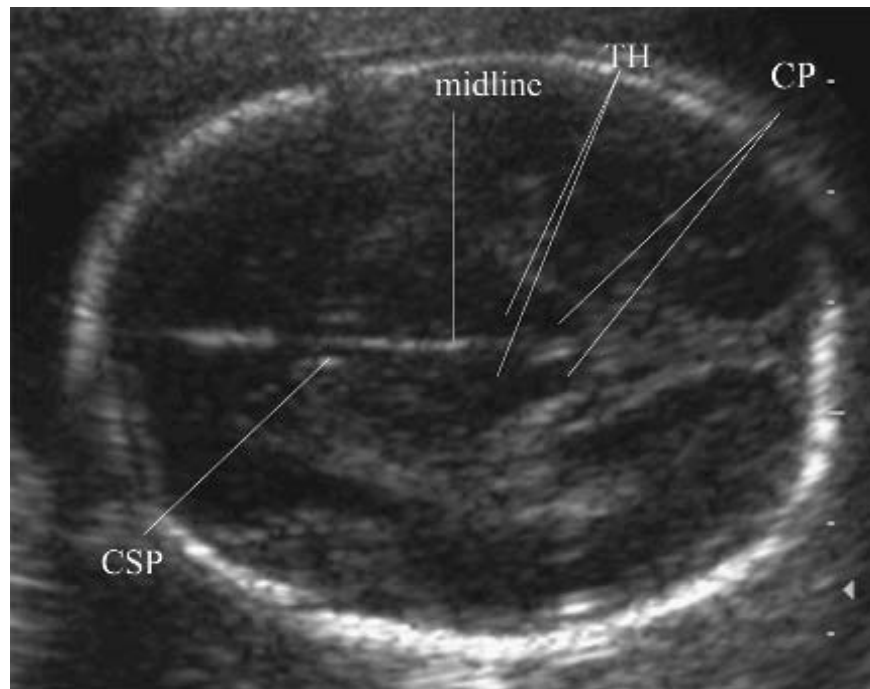


Figure 3.3 Transverse section of the fetal head demonstrating the landmarks required to measure the BPD using the thalami view. CP, cerebral peduncles; CSP, cavum septum pellucidum; TH, thalami. (Elsevier 2004)

2.3.1.1 Measuring the BPD from the lateral ventricles view:-

Obtain a longitudinal section of the fetus as described above. Small sliding movements of the transducer on each side of the fetal spine will give a longitudinal section of the fetal head that will demonstrate a strong midline echo. By rotating the transducer through 90° a transverse section of the fetal head is obtained. If the midline is not in the exact middle of the section, alter the angle of the probe slightly on the maternal abdomen. This corrects for the angle of asynclitism. Once the midline is centrally placed do not alter the angle of the probe. Now assess the shape of the fetal skull. The required shape is that of a rugby football, with the more pointed end at the synclitism. As the cavum lies one-third of the distance from the synclitism to the occiput, identifying the cavum will allow you to determine which is the front and the back of the head. If the section is not the required ovoid shape, make minor rotational adjustments. If the landmark features listed above are not evident when the midline and shape are correctly imaged then the level of the section is wrong and should be corrected by small sliding movements of the probe up or down the fetal head. (Elsevier 2004)

The BPD is then measured on the frozen image. Place the intersection of the two arms of the first onscreen caliper on the

outer aspect of the proximal skull surface. Place the intersection of the two arms of the second caliper on the outer aspect of the distal skull surface at right angles to the midline and at the widest diameter. This BPD measurement includes the thickness of both parietal bones and is commonly described as an 'outer to outer' measurement. Some centers include the thickness of only one (the upper) parietal bone when measuring the BPD. This measurement is commonly described as an 'outer to inner' measurement. The two techniques will produce BPD measurements that differ typically by 2-3 mm in the second trimester, approximately equivalent to 1 week of gestation. There is no consensus regarding which technique is more acceptable, although the 'outer to inner' method finds greater favor with the physicists. This is because the anterior edge of the parietal echo is less influenced by the equipment's controls than the posterior edge. The 'outer to inner' measurement is thus a more accurate representation of the true distance selected for measurement than the 'outer to outer'. What is critically important is that the technique used (including the section selected for measurement) corresponds to that employed when the reference data, that is the biometry charts, were produced? We recommend using the 'outer to outer' technique because this measurement can be combined with the occipitofrontaldiameter measurement to estimate the 'derived' HC measurement. (Elsevier 2004(

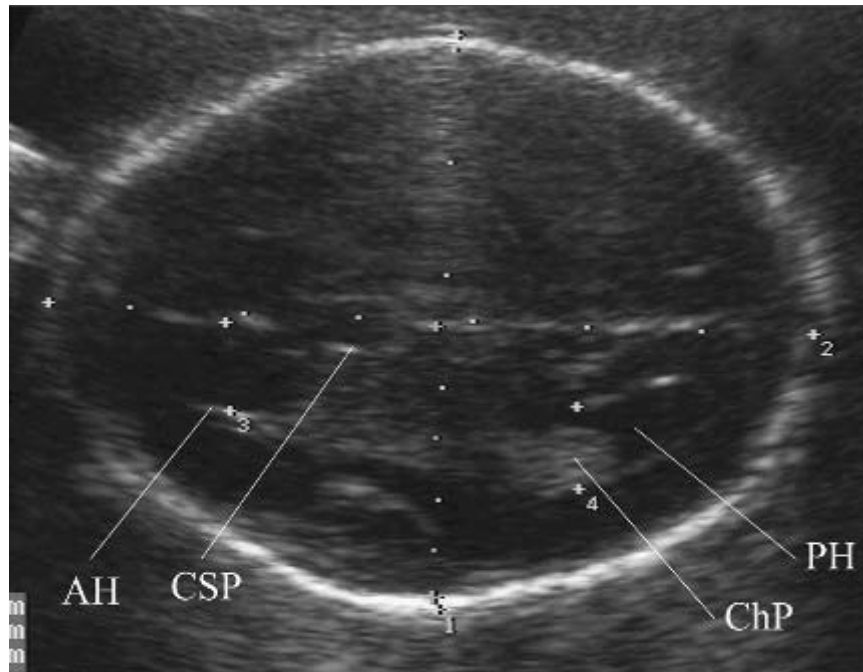


Figure 3.4 Transverse section of the fetal head with Thecallipers placed on the outer border of both the proximal and distal parietal bones (diameter 1). The measurement therefore produces an ‘outer to outer’ BPD measurement. The occipitofrontal diameter has also been measured in this image (diameter 2). Note the placement of the calipers to produce an ‘outer to outer’ OFD measurement. Measurements of the anterior and posterior horns of the distal lateral ventricle and distal hemisphere have also been taken (diameters 3, 4 and 1, respectively). (Elsevier 2004)

2.3 .1.2 Problems

Incorrect angle. If the angle of the probe on the maternal abdomen is incorrect, the midline echo does not lie centrally within the fetal skull . Similarly, the echoes from the lateral ventricles will not be visualized symmetrically about the midline. The angle of the probe to the maternal abdomen should be altered, without sliding or rotating the probe. Incorrect rotation.

This is readily recognized because the visualized shape of the fetal skull is not that of a rugby football – it is usually too round – and/or not all the landmark features are seen. For example, visualizing the anterior horns together with the cerebellum indicates that the selected level at the back of the head is too low. This, in fact, is the suboccipitobregmatic view required for measurement of the TCD. (Elsevier 2004)

Visualizing the orbits together with the posterior horns of the lateral ventricles indicates that the selected level at the front of the head is too low. A slightly higher level at the front of the head, between the orbits and the anterior horns, can produce the false impression of a lemon-shaped skull. Rotating the probe will correct the shape but you must be careful to maintain the correct angle. Incorrect level. Sliding movements of the probe will alter the level of section. A continuous midline indicates the selected level is too high. A section demonstrating the orbits and the cerebellum is too low. Be careful not to rotate or change the angle of the probe as you slide. Midline not horizontal. Having obtained the correct section displaying the required landmarks check that the midline is not lying at an angle to the horizontal. (Elsevier 2004)

Dipping one end of the probe will orientate the head into the correct position. BPD measurements in breech and transverse presentations. In the second half of pregnancy BPD measurements obtained from fetuses presenting transversely or by the breech can be unreliable. In these presentations the fetal head might be dolichocephalic (long and narrow) in shape. This

produces a BPD measurement that is artifactually small for gestational age. The head circumference measurement, however, is unaltered by presentation and is therefore a reliable indicator of gestational age irrespective of fetal presentation. OP/OA position. Measurement of the BPD should only be taken when the fetal head is in the occipitotransverse (OT) position, because the landmarks are best recognized when the midline echo and the other landmarks are at 90° to the ultrasound beam. The BPD should therefore not be measured if the fetal head is directly occipitoposterior (OP), directly occipitoanterior (OA) or deep in the maternal pelvis. Tilting the woman into a 45° head-down position and/or partially filling the maternal bladder might displace and rotate the fetal head such that it can be measured. Alternatively, transvaginal imaging could be attempted. If these options are unsuccessful, estimation of gestational age could be made from measurement of femur length, but we recommend rescanning the woman at a later date to ensure that the intracranial anatomy is normal. (Elsevier 2004)

Evaluating the intracranial anatomy from the lateral ventricles view It is important to observe the information presented in a section selected for measurement and thus to familiarize yourself with the range of normal appearances at a specific gestation. Once you have this experience you will immediately identify a finding that is not normal, even if you do not have the expertise to make the diagnosis. For example, make sure the cavum is normal in size and position. Similarly, make sure that the choroid plexus fills the posterior horn of the lateral ventricle and is of a

homogeneous echogenicity. Evaluate the echo pattern in both halves of the brain to exclude any unusual echo-poor or echo-bright areas, ensuring that the echo pattern in both halves of the brain is similar and symmetrical about the midline. Are the intracranial contents of normal echogenicity or do they appear brighter than usual, such that you may have reduced the gain slightly? Abnormally echo-bright skull contents are unlikely to indicate a brain abnormality but rather skull hypo mineralization, as seen in osteogenesis imperfecta type 11 or hypophosphatemia (Elsevier 2004)

2.3 .1.3 Skull shape

It is important to observe the normal shape of the fetal skull and appreciate how this varies, albeit subtly, with increasing gestation. As discussed above, the shape of the normal skull at 20–22 weeks is that of a rugby football. Abnormal scalloping of the frontal bones produces a more angular appearance to the front of the skull in this view. (Elsevier 2004)

This is known as the ‘lemon’ sign and is associated with spina bifida. However, a similar appearance is demonstrated by the skull of the normal fetus early in the second trimester, before 16 weeks. It can also be obtained in a normal 20–22 week fetus from an incorrect BPD section, if the probe is rotated slightly too far toward the fetal orbits at the front of the skull. Conversely, fetuses with spina bifida rarely demonstrate a lemon-shaped skull after 24–26 weeks. These factors should be remembered if a lemon-shaped skull is suspected. Measuring the BPD from the thalami view. Follow the same procedure as outlined above. From

the anterior horns section, make a very slight rotation of the probe toward the fetal neck (i.e. the back of the head) to image the basal cisterns in preference to the posterior horns of the lateral ventricles. This is followed by a very slight sliding movement of the probe downward, toward the fetal body so that the lower border of the cavum is just visible together with the optimal view of the thalami. The BPD is measured using the technique described above. The issue of ensuring the technique used (including the section selected for measurement) corresponds to that employed when the reference data were produced also applies. The range of problems described above are also relevant for this view. (Elsevier 2004)

Evaluating the intracranial anatomy from the thalami view As discussed above, it is important to observe the information presented in a section selected for measurement and thus to familiarize yourself with the range of normal appearances at a specific gestation. Once you have this experience you will immediately identify a finding that is not normal, even if you do not have the expertise to make the diagnosis. For example, make sure that the thalami are of normal size, position and have a homogeneous echogenicity. Note that the normal third ventricle is a midline structure that lies between the two thalamic hemispheres and is usually too small to identify in the normal fetus. Exclude any unusual echo-poor or echo-bright areas and ensure that the echo pattern in both halves of the brain is similar and symmetrical about the midline. Are the intracranial contents of normal echogenicity or do they appear brighter than usual such

that you may have reduced the gains slightly? Abnormally echo-bright skull contents are not indicative of a brain abnormality but rather of skull hypomineralization as seen in osteogenesis imperfecta type 11 and hypophosphatasia. (Elsevier 2004)

2.3 .2 Measuring the abdominal circumference (AC)

which the AC is measured. The landmark features are:-

- A circular section of the abdomen demonstrating an unbroken and short rib echo of equal size on each side.
- A cross-section of *one* vertebra visualized as a triangle of three white spots.
- A short length of umbilical vein. This should be imaged so that it is centrally placed between the lateral abdominal walls and is a third of the way along an imaginary line drawn from the anterior abdominal wall to the fetal spine. The stomach, usually visualized as a hypoechoic area in the left side of the abdomen. (Elsevier 2004)

2.3.2 .1 Method

Obtain a longitudinal view of the fetus that demonstrates both the fetal heart and the fetal bladder. Slide the transducer laterally until the fetal spine is visualized. Rotate the transducer through 90° at the level of the fetal stomach to obtain a cross-section. The outline should be circular, if it is ovoid make a small adjustment of the rotation or the angle of the transducer. If the umbilical vein is not visualized as described above, make small sliding movements of the transducer to change the level of the section. Freeze the image. (Elsevier 2004)

The circumference of the abdomen is measured in the same way as the head circumference, using the two-diameter method. The anteroposterior diameter (APAD) is measured from the fetal spine to the anterior abdominal wall. The short section of umbilical vein should lie along this axis. The transverse abdominal diameter (TAD) is measured across the widest part of the abdominal circumference section at 90° to the APAD. Both diameters are measured using the 'outer to outer' technique. (Elsevier 2004)

The machine's software then calculates the abdominal circumference (AC) using the formula derived from the formula for the circumference of a circle ($2\pi r$):

$$AC = 3.14 (TAD + APAD)/2$$

Growth of the AC is illustrated in Appendix 6.

2.3 .2 .2 Problems

Directly anterior fetal spine

The umbilical vein will not be seen in transverse section because it lies in the acoustic shadow produced from the fetal spine . In the second trimester it is nearly always possible to slide the transducer to a more lateral position on the maternal abdomen, or to dip one end of the transducer, to allow the umbilical vein to be imaged . Alternatively, you can complete the remainder of the examination, by which time the fetus might have moved into a more favorable position. (Elsevier 2004(

Non-circular outline

An oval outline indicates an oblique cross-section. This can be rectified by a slight change in rotation or angle – the choice depends on the position of the fetal body relative to the horizontal plane. If the longitudinal fetal spine is in the horizontal plane then rotation is required, if the longitudinal fetal spine is at an angle to the horizontal then angling is required. (Elsevier 2004.capter7. Routine second trimester screening – assessing gestational age.

Long length of umbilical vein

The umbilical vein travels up through the liver at approximately 45° . Thus, if the section on which you intend to measure the AC demonstrates a long length of umbilical vein, you know you have an oblique and incorrect section. If the longitudinal fetal spine is in the horizontal plane then angling is required, if the longitudinal fetal spine is at an angle to the horizontal then rotation is required. (Elsevier 2004(

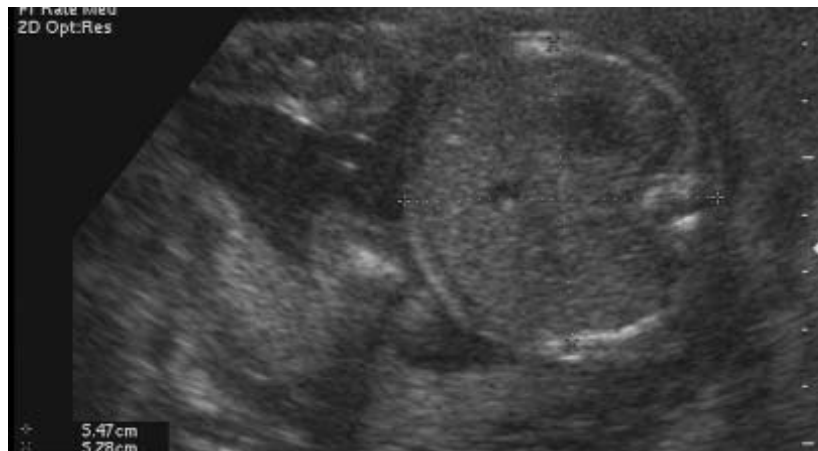


Figure 3.5 Measurement of the abdominal circumference using the two diameter method. The anteroposterior diameter (APAD) is obtained by placing one caliper on the outer border of the skin directly behind the spine and the second on the outer border of the anterior abdominal wall, following the direction of the umbilical vein. The transverse abdominal diameter (TAD) is obtained by placing the calipers on the outer borders of the widest part of the fetal abdomen, at 90° to the APAD. (Elsevier 2004)

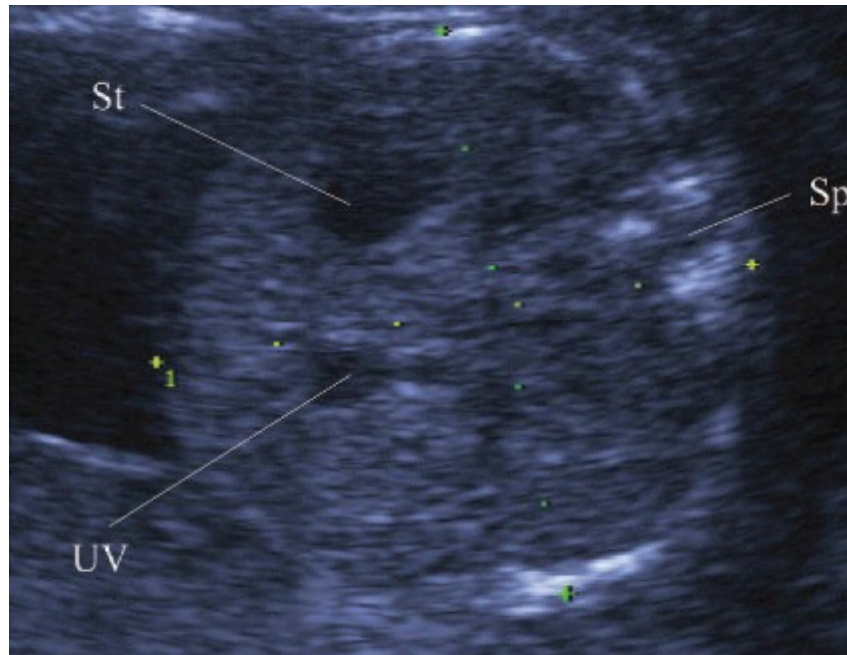


Figure 3.6 Transverse section of the fetal abdomen Demonstrating the landmarks required to measure the Abdominal circumference. Note the appearance of the normal single vertebra (Sp), the short length of umbilical vein (UV) and its position. Note also the appearance and position of the normally sized stomach (St). (Elsevier 2004)

2.3.3 Measuring the femur length

This measurement is as accurate as the BPD in the prediction of gestational age. It is useful in confirming the gestational age estimated from BPD or HC measurements and can often be obtained when fetal position prevents measurement of the BPD or HC. As examination of intracranial anatomy is an important part of all ultrasound examinations, measurement of femur length should not replace that of the BPD or HC as the sole predictor of

gestational age. The femur can be measured from 12 weeks to term. (Elsevier 2004)

2.3 .3 .1 Method

Measuring the femur is ideally undertaken after the AC has been measured. Slide the probe caudally from the AC section until the iliac bones are visualized. At this point, a cross-section of one or both femurs is usually seen. The upper femur should be selected for measurement. The lower femur is frequently difficult to image clearly because of acoustic shadowing from fetal structures anterior to it. Keeping the echo from the anterior femur in view, rotate the probe slowly until the full length of the femur is obtained. You might need to make a *small* sliding movement after each rotational movement to bring the probe back onto the femur. To ensure that you have the full length of the femur and that your section is not oblique, soft tissue should be visible beyond both ends of the femur and the bone should not appear to merge with the skin of the thigh at any point. The end-points of the femur are often difficult to define when the femur is imaged lying horizontally but are much easier to define when the bone lies at a slight angle (5–15° to the horizontal). The angle of the bone relative to the horizontal can be manipulated by dipping one end of the probe gently into the maternal abdomen. The measurement of the femur is made from the center of the 'U' shape at each end of the bone. (Elsevier 2004)

This represents the length of the metaphysis. It is good practice to obtain measurements from three separate images of the same femur. These should be within 1 mm of each other. Growth of the femur is illustrated in Fig. 7.16 and Appendix 9 As with the BPD, a growth chart should be used to determine if the FL measurement is within the normal range for the gestational age as calculated from a reliable menstrual history and/or a first trimester dating scan. In practical terms, you should measure the FL and then you should plot it on a growth chart according the gestational age. If the measurement lies outside the normal range for the menstrual age then you must consider whether it is valid to ignore the menstrual dates and reassign EDD based on the ultrasound measurements or whether the menstrual dates should be retained . In cases where the gestational age is unknown or the menstrual history is unreliable, estimation of gestational age should be made from a dating chart, in which the FL (independent variable) is plotted on the x-axis and the gestational age (dependent variable) is plotted on the y-axis, or from tables derived in the same manner . In practical terms, this means that, having measured the FL, you should estimate the gestational age by use of the tables. The measurement can then be plotted on the growth chart according to the gestational age derived from the dating tables. We recommend that dating charts are used in look-up table format and that all measurements are represented graphically on growth charts. (Elsevier 2004)

2.3.3.2 Problems

-Fetal movements

Most problems arising with measuring the FL are due to a combination of fetal movements and slow use of the freeze button. The cine loop might be useful in such situations. If the end-points of the femur cannot be adequately visualized, unfreeze the image and seek another, better image. It is very easy to under- or overestimate the FL by 3–5mm if a suboptimal image is measured. (Elsevier 2004)

-One or both end-points are difficult to define

Dip one end of the probe gently in to the maternal abdomen, as described above. (Elsevier 2004)

-The upper femur appears straight but the lower femur appears bowed

The slight bowing seen in the lower limb is a normal artifact of the imaging process. Unilateral femoral abnormalities are very rare but should always be considered as a possible, if unlikely, explanation for significant dissimilarity in the appearance of the two femurs. An experienced second opinion should be sought if necessary. (Elsevier 2004)

-Gestational age equivalents of the BPD or HC and femur disagree

The estimation of gestational age obtained from measurements of femur length should agree with that obtained from the measurement of the BPD, HC and/or the TCD. If the femur length is small (below the 5th centile) compared to the BPD or HC (on the 50th centile) and TCD then all the long bones and the plantar view of the feet should be carefully measured to exclude skeletal

dysplasia . A short femur is also a minor marker for chromosomal abnormalities, including trisomy 21. (Elsevier 2004)

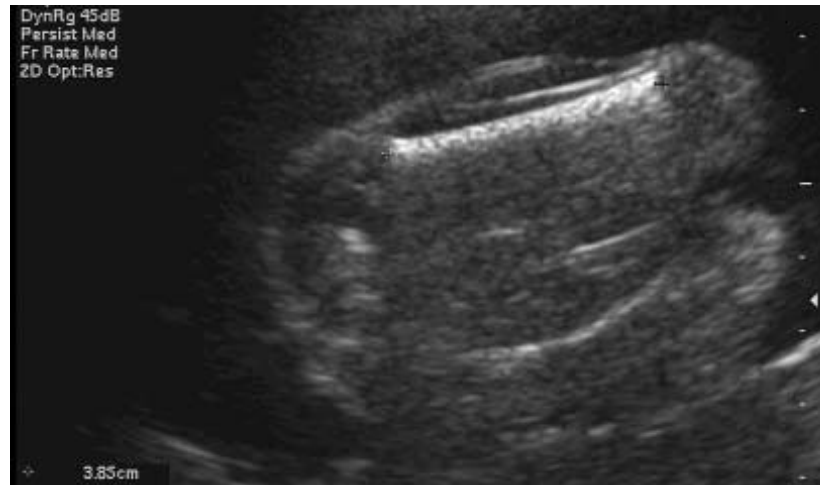


Figure 3.7 Measurement of the fetal femur. Note that soft tissue is visible beyond both ends of the bone. The femur length is the distance between the caliper markers (Elsevier 2004)

2.4 Evaluation amniotic fluid volume :-

From early in the second trimester most of the amniotic fluid is fetal urine. Amniotic fluid is therefore produced by the fetal kidneys and removed by fetal swallowing and subsequent absorption by the fetal bowel. Disruption of this pathway will cause an abnormal reduction (oligohydramnios) or increase (polyhydramnios) in amniotic fluid volume. Abnormal amniotic fluid volume is therefore an important indicator of a range of varying fetal abnormalities, and one that can be readily assessed during every examination. Measuring amniotic fluid volume Three methods of assessing amniotic fluid volume can be used:

1. visual assessment

2. measurement of the maximum vertical depth (MVD)
3. measurement of the amniotic fluid index (AFI).

2.5 Confirming or assign gestational age :-

Interpretation of the measurements taken at the time of the routine 20–22 week ultrasound examination will depend on whether measurements have been taken at an earlier examination. If reliable measurements were taken at an earlier examination, then the EDD should have already been assigned, or confirmed, at that time. The second trimester measurements are thus used to assess fetal growth. If the second trimester examination is the first of the pregnancy then the measurements are used: (i) to confirm the gestational age and the EDD based on the menstrual history; or (ii) to assign the gestational age and the EDD when the menstrual history is unknown or unreliable .

(Elsevier)

We recommend that the minimum measurements that should be taken for gestational age assessment are those of the BPD or HC and the FL. Confirming gestational age in the second Trimester Confirmation of gestational age at the second trimester examination is based either on a reliable menstrual history or/and on measurements obtained from an earlier and reliable ultrasound examination. In both instances the gestational age of the second examination is known. Four outcomes are possible:

1. The measurements of the BPD or HC and the FL fall within the normal range for the gestational age when plotted on appropriate charts. This confirms the previously assigned EDD based either on the LMP or the early scan.

2. Measurements of the BPD or HC and the FL fall outside the normal range for menstrual age. In this case you should consider carefully the reliability of the menstrual history/early scan:

- If there is uncertainty concerning the reliability of the previously assigned EDD then, in the majority of cases, it is considered acceptable practice to reassign the EDD based on the second trimester measurements. Estimate the gestational age from the BPD or HC using dating tables (see Appendices 3 and 5) and then replot the FL for the estimated gestation. If the FL is within normal range you should then assign the new EDD from the BPD-derived or HC-derived gestational age. Your AC measurement should further confirm the ultrasound date you have assigned. (Elsevier 2004)
- If there is no reason to doubt either the optimal menstrual history (for example if the date of conception is indisputable) or the measurements from the earlier scan then the previously assigned EDD should be kept. The second trimester measurements in this case therefore indicate poor fetal growth. As early onset growth restriction is associated with chromosomal anomalies and/or poor outcome, karyotyping should be considered and the pregnancy should be monitored carefully.

3. The BPD or HC falls within normal range for the known gestational age but the FL is below the normal range. In this case you should repeat the measurements of the BPD or HC and FL and measure the TCD and AC. If all the measurements but that of the FL agree with the known

gestational age, you should suspect a skeletal dysplasia or trisomy 21.

4. The FL falls within the normal range for known gestational age but the BPD is below the normal range. Again, you should repeat the measurements of the BPD or HC and FL and also measure the TCD and AC. If the HC and BPD are both below the normal range you should look for spina bifida or microcephaly, remembering that the majority of fetuses with spina bifida will have an abnormally shaped or absent cerebellum. If all the measurements are appropriate, with the exception of the BPD, look at the shape of the head

- The most usual cause for a small BPD is dolichocephaly (narrow head) due to a breech or transverse presentation. Such circumstances emphasize why it is more appropriate to use the HC instead of the BPD with the FL to confirm the known gestational age. Some authors recommend the use of the cephalic index:

$$\text{Cephalic index} = \text{BPD}/\text{OFD} = 80 \pm 5$$

An index of less than 75 is seen in cases of dolichocephaly and makes the BPD measurement unreliable for estimating gestational age. An index of more than 85 is seen in brachycephalic heads (wide and short) and also makes use of the BPD for estimating gestational age unreliable. The cephalic index is constant throughout pregnancy. (Elsevier 2004)

New Sudanese Reference Chart of Fetal Biometry and Weight Using Ultrasonography

Abstract

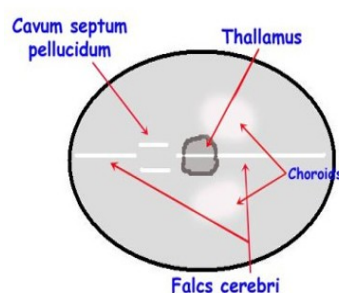
Background: Many centers in Sudan use the reference data for fetal biometry. The recently published population-based reference either overestimated or underestimated the weight of the fetuses.

Objective: To establish a national reference for fetal biometry, and weight by gestational age for singleton fetuses in Sudan.

Methods: Data were collected on all singleton live births documented in the data collection sheet done at Saudi Hospital from 2015 to 2016 ($n = 225$). Gestational age estimation was based on the last menstrual period and fetal ultrasound thereafter. Fetal biometry and weight and other 6 fetal weight formulae were assessed. Reference data for fetal growth by gestational age were created. **Results:** New charts and reference equations are reported in Sudanese population for fetal biparietal diameter, head circumference, abdominal circumference and femur length and fetal weight. **Conclusion:** We advocate that these reference charts and equations for fetal biometry and weight might be valuable in the clinical use for appropriate ethnic Sudanese. Ayad, *et al*, 2016

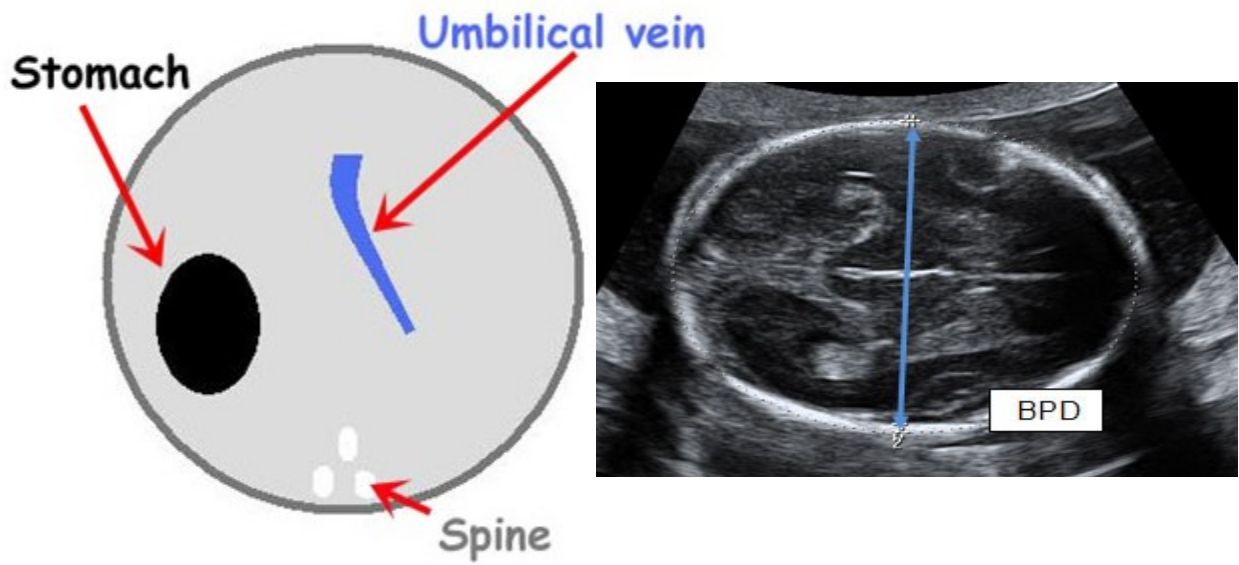
Keywords

Fetal Biometry, Fetal Weight, Gestational Age, Ultrasonography





TAS of FL 36 weeks fetus

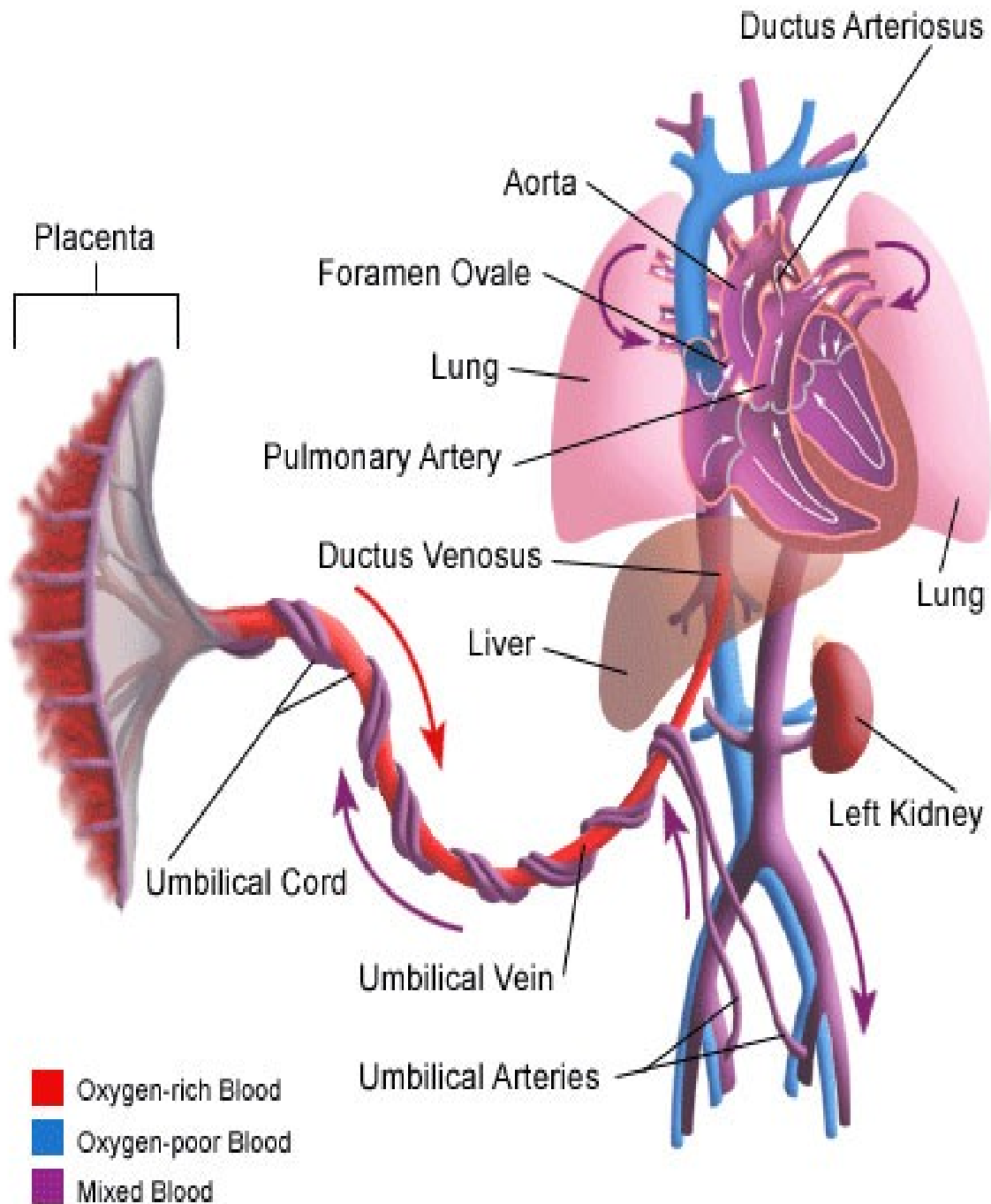


TAS of BBD 34 weeks fetus



TAS of AC 35 weeks fetus

Fetal Circulation



chapter3
material andmothed

chapter3

material andmothed

Study area :

This study was be carried out in Khartoum state .

Study period:

This study conducted within period between

Sample techniques:

Simple Randum technique

Sample size :

150 pregnant women from second to third trimester came to ultrasound department foe routine examination

Data collection :

Direct interview by use Questionnaire

Data analysis:

Collected data wase analyzed by using statistical package of social science (Axial) program

Ethical consideration:

Permission to conduct this study was obtained from college of post graduate insudan university of science and technology and verble consent form group under study

mothed :

Instrumentation :-

Ultrasound machines Toshiba- power vision – 600 with transabdominal 3.5MHZ convex probes with facility computerize reporting system .

Techniques of Data collection :-

The position of the patient is supine Scanning technique is sagittal ,oblique and coronal viow.

Chapter Four

Results

Chapter Four

Results

Figure 1: shows the correlation between the mother Height in cm and Fetus weight in Kg.

Figure 1: shows the correlation between the mother Height in cm and Fetus weight in Kg. It reveals that: the mother Height has no correlation with Fetus weight which are insignificance ($R^2 = 0.03$).

Figure 2: shows the correlation between the mother weight in Kg and Fetus weight in Kg

Figure 2: shows the correlation between the mother weight in Kg and Fetus weight in Kg . It reveals that: the mother weight has no correlation with Fetus weight which are insignificance ($R^2 = 0.03$).

Figure 3: shows the correlation between the BMI (Kg/m^2) versus fetal weight standard and measured

Figure 3: shows the correlation between the BMI (Kg/m^2) versus fetal weight standard and measured. It reveals that: the fetal weight has been increases as the BMI increases in forms of linear correlation which are insignificance ($R^2 = 0.05$ and 0.02) for the influence of BMI in measured fetal weight and standard one respectively, such finding could be ascribed to different factors (e.g. nutrition, environment, gender, race, genetics, ... etc) as the author assumed.

Figure 4: shows the correlation between the GA in weeks versus measured parameters (FL, AC and BPD) in cm.

Figure 4: shows the correlation between the GA in weeks and versus measured parameters (FL, AC and BPD) in cm. It reveals that: the GA has has been increases as measured parameters (BPD ,FL, and AC) increases in forms of linear correlation which are significance ($R^2 = 0.692, 0.33, 0.768$) for the influence of GA in measured parameters (BPD ,FL, and AC) respectively, such finding could be ascribed to different factors (e.g. nutrition, environment, gender, race, genetics, ... etc) as the author assumed.

Figure 5: shows the correlation between the GA in weeks versus Fetal weight standard and measured

Figure 5: shows the correlation between the GA in weeks versus fetal weight standard and measured. It reveals that: the fetal weight has been increases as the GA increases in forms of linear correlation which are significance ($R^2 = 0.996$ and 0.738) for the influence of GA in standard fetal weight and measured one respectively, such finding could be ascribed to different factors (e.g. nutrition, environment, gender, race, genetics, ... etc) as the author assumed.

Chapter Five

Chapter Five

5.1 Dissection :-

One hundred and fifty pregnant women on the second and third trimester to assesses the fetal weight according to gestational age .

On this study we found that the relation between mother height and fetus weight had no significance $R^2 = 0.03$ figure (4_1).

The mother weight had no effect on fetal weight $R^2 = 0.3$ figure (4_2).

On our study we found that the gestational age parameters (BPD, FL, AC) where ($R^2 = 0.69, 0.33, 0.76$) respectively so the relationship between GA more accurate on AC ($R^2 = 0.76$) than BPD ($R^2 = 0.69$) and the FL ($R^2 = 0.33$) was less accurate Table(4-3).

The most strong relationship between GA, weight standard and measured $R^2 = 0.99, R^2 = 0.73$ respectively this result was almost accurate as Ayad, et al, 2016, table (4.4). We found that there was no relation between BMI and fetal weight.

5.2 Conclusion:

the correlation between the GA in weeks and versus measured parameters (FL, AC and BPD) in cm. It reveals that: the GA has been increases as measured parameters (BPD, FL, and AC) increases in forms of linear correlation which are significance ($R^2 = 0.692, 0.33, 0.768$) for the influence of GA in measured parameters (BPD, FL, and AC) respectively, such finding could be ascribed to different factors (e.g. nutrition, environment, gender, race, genetics, ... etc) as the author assumed. that describe in figure 5

the correlation between the GA in weeks versus fetal weight standard and measured. It reveals that: the fetal weight has been increases as the GA increases in forms of linear correlation which are significance ($R^2 = 0.996$ and 0.738) for the influence of GA in standard fetal weight and measured one respectively, such

finding could be ascribed to different factors (e.g. nutrition, environment, gender, race, genetics, ... etc) as the author assumed. that describe in figer (6) shows the correlation between the BMI (Kg/m^2) versus fetal weight standard and measured. It reveals that: the fetal weight has been increases as the BMI increases in forms of linear correlation which are insignificance ($R^2 = 0.05$ and 0.02) for the influence of BMI in measured fetal weight and standard one respectively, such finding could be ascribed to different factors (e.g. nutrition, environment, gender, race, genetics, ... etc) as the author assumed. in figer7

5.3 Recommendation:

5.3.1 we have to use three parameter at least e.g (AC,FL,BBD)for accurate fetal weight

5.3.2 for standard fetal weight we must increase the data volume

5.3.3 for the study must include the type of nutrition, occupation and type of work **5.3.4** the study of placenta measurement in relation to fetal weight

References:

- 1/ Sharon Perkins,2015
- 2/ THOMAS,2007 Madigan Army Medical Center, Tacoma, Washington, 1;76(9):1341-1346.
- 3/ Michele Drehmer, BruceBartholow Duncan, Gilberto Kac, Maria Inês Schmidt
Published: January 30,
2013<http://dx.doi.org/10.1371/journal.pone.0054704>
- 4/Nutr Res Pract.2009 nutrition research and practice Jun 30. doi: [10.4162/nrp.2009.3.2.134](https://doi.org/10.4162/nrp.2009.3.2.134)Summer; 3(2): 134-140.
- 5/ Elsevier 2004, Obstetric_Ultrasound,How,Why,and,When, Fetal growth
3rd,Edition,(180-182),
- 6/ Michele Drehmer, BruceBartholow Duncan, Gilberto Kac, Maria Inês Schmidt
Published: January 30,
2013<http://dx.doi.org/10.1371/journal.pone.0054704>
- 7/ Elsevier 2004, Obstetric,Ultrasound,How,Why,and,When,Fetal growth
3rd,Edition,(180-182),
- 8/ Elsevier 2004, Obstetric_Ultrasound,How,Why,and_When ,Routine second trimester screening ,assessing gestational age, 3rd_Edition(98-112)
- 9/ Caroline Edward Ayad1, Ahmed Abdelrahim Mohammed Ibrahim2,

Mohammed Elfadil Mohammed GarElnabi¹, Bushra Hussein Ahmed³,
Elsafi Ahmed Abdalla¹, Mohammed Ahmed ElshiekhSaleem,2016,
Open Journal of Radiology, , 6, 131-139, Published Online June
2016 in SciRes. <http://www.scirp.org/journal/ojrad>
<http://dx.doi.org/10.4236/ojrad.2016.62020>
10/ John Wiley & Sons,2004,kudoswiley, 10.1002/uog.1751
12/Rachel. Nall 2016. [Healthline](#). [Healthy Pregnancy](#). Second
Trimester Weight Gain & Physical Change

13/sadler. *Langman's Medical Embryology*. (<http://connection.LWW.com/go/sadler>)

Appendixes:-

Appendix 1

Master sheet of data collection

MH/c	MW/K	BMI	GA	FL	AC	BPD/c	FW/K	F
m	g			/cm	/cm	m	g	W
								-
								S
								t
								d

Keywords:

MH = maternal height

MW = maternal weight

BMI= body mass index

GA=gestational age

FL =femur length

AC=abdominal circumference

BPD= bi parietal diameter

FW=fetal weight

Std = stander

Appendix 2s:-



TAS ;FL of 24-weeks fetus



TAS ;AC of 33-weeks fetus



TAS ;BBD of 30-weeks fetus