Development of Fetus

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ABSTRACT

In Fetal size and fetal growth trajectories are important indicators of fetal health. This article reviews fetal growth from an obstetric perspective and addresses various issues including the physiologic mechanisms that determine fetal growth trajectories, known risk factors for abnormal fetal growth, diagnostic and prognostic issues related to restricted and excessive growth and temporal trends in fetal growth.The perinatal literature contains several potentially confusing terms and concepts related to fetal size and growth. These include distinctions between fetal growth 'standards' and fetal growth 'references', and between fetal growth charts based on estimated fetal weight vs those based on birth weight. There is also a lack of clarity around fundamental concepts such as the incidence of fetal growth restriction in pregnancy: does the frequency of fetal growth restriction increase or decrease as gestation advances (or is it a constant 3 or 10% for each gestational week)? The demonstrated associations between in-utero growth and adult chronic illnesses (such as coronary heart disease) are another potential source of confusion in obstetrics: should pregnancy interventions be predicated on improving such long-term outcomes? Finally, there is the seductive proposition regarding the need for customizing fetal growth standards. Should fetal size and growth be assessed in the context of fetal gender alone or should other physiologic parameters such as maternal height, weight, parity and ethnicity/race provide additional context? This review attempts to clarify these issues by providing a brief synthesis of the prevailing perspectives.the review the development of a fetus is a marvel of nature, encompassing a series of intricate processes that transform a single cell into a fully formed human being. This journey, spanning approximately 40 weeks, is divided into three distinct stages: the germinal stage, the embryonic stage, and the fetal stage.The germinal stage, spanning the first two weeks after conception, marks the beginning of life. It starts with the fusion of a sperm and an egg, resulting in the formation of a zygote. This zygote undergoes rapid cell division, forming a blastocyst, which implants itself into the uterine wall. During this stage, the placenta begins to develop, providing the fetus with essential nutrients and oxygen.[2]The embryonic stage, spanning weeks 3 to 8, is characterized by rapid growth and development. Major organs and structures, including the heart, brain, spinal cord, and limbs, begin to form. By the end of this stage, the embryo has a beating heart and rudimentary facial features, setting the stage for further development.[15]The fetal stage, spanning weeks 9 to birth, is a period of refinement and growth. The fetus continues to develop, with organs and structures maturing

further. It begins to move, and its senses start to develop. By the end of this stage, the fetus is capable of survival outside the womb, although it will continue to grow and develop after birth.[3]Throughout fetal development, the fetus is vulnerable to environmental factors such as nutrition, exposure to toxins, and maternal health.[8]Therefore, it is crucial for pregnant individuals to receive regular prenatal care to ensure the health and well-being of the fetus.

I. INTRODUCTION

The word fetus (plural fetuses or feti) is related to the Latin fētus ("offspring", "bringing forth", "hatching of young")[2][3][4] and the Greek "φυτώ" to plant. The word "fetus" was used by Ovid in Metamorphoses, book 1, line 104.[5]The predominant British, Irish, and Commonwealth spelling is foetus, which has been in use since at least 1594. The spelling with -oe- arose in Late Latin, in which the distinction between the vowel sounds -oe- and -e- had been lost. This spelling is the most common In most Commonwealth nations, except in the medical literature, where fetus is used. The more classical spelling fetus is used in Canada and the United States. In addition, fetus is now the standard English spelling throughout the world in medical journals.[6] The spelling faetus was also used historically.[7]

II. NUTRITION

Fetal growth is determined by many factors, both genetic and environmental. Of the latter, adequate placental perfu sion and placental function are crucial. Maternal nutrition is not a limiting factor except in cases of extreme starva- tion, although chronic undernutrition may be associated with anaemia and may lead to a low-birthweight baby.[15]The fetus, insulated in its protective amniotic sac and relatively weightless, directs most of the energy supplied to it to growth. The energy is derived mainly from glucose. Only small amounts of lipids, as free fatty acids, cross the placenta until the fourth quarter of preg nancy. Any excess carbohydrate, after the growth and metabolic energy needs of the fetus have been met, is converted into lipids, and this conversion increases as term approaches.From the 30th gestational week the fetal liver becomes increasingly efficient and converts glucose into glycogen. Which is stored in the fetal heart muscle, the skeletal mus cle and the placenta. Should fetal hypoxia occur the fetus is able to obtain energy from the heart muscle and pla centa for anaerobic glycolysis.[12]Free fatty acids are formed and stored in brown and white adipose tissue. Brown fat is deposited around the fetal neck and behind the scapulae and the sternum and around the kidneys. It is metabolized to provide energy to maintain the infant's body temperature after birth. White adipose tissue forms the subcutaneous cover of the body of a term fetus, but in preterm babies the layer may be thin. It acts as an insulator and as a lipid store.[9]The fat stores of an 800 g fetus (24-26 weeks gestation) Constitute 1% of its body weight; by the 35th week fat Constitutes 15% of fetal body weight. As the placenta clears the blood of bilirubin and other metabolic products that require a transferase activity, the fetal (and neonatal) liver is deficient in certain trans- ferases. The result is

that unless the deficiencies are corrected in the early neonatal period, bilirubin may accumulate in the neonate's blood, which is of some consequence in haemolytic disease of the newborn.[21]Amino acids cross the placenta by active transfer and are converted into protein. Protein synthesis exceeds protein breakdown, and the fetus uses some of the breakdown amino acids for resynthesis.

Table A: stages & Time frame of development.

Stage	**Time Frame**	**Key Developmental Milestones**
Germinal Stage	Weeks 1-2	Fertilization, formation of zygote, blastocyst development, implantation, formation of placenta.
Embryonic Stage	Weeks 3-8	Development of major organs and structures (heart, brain, spinal cord), limb buds, facial features, heartbeat.
Fetal Stage	Weeks 9- Birth	Continued growth and development, maturation of organs and structures, movement, development of senses, viability.

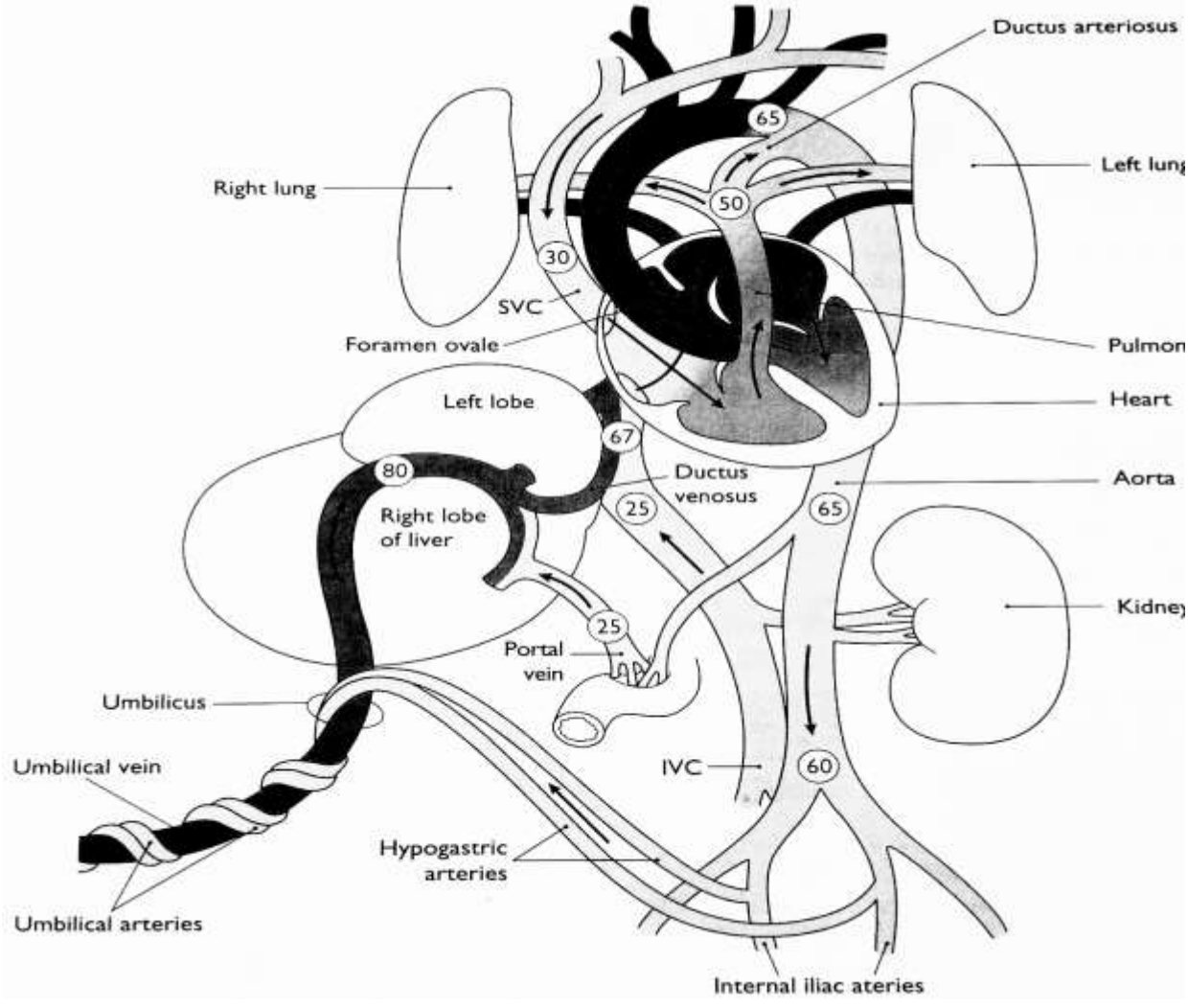

Figure : 01-Normal circulation in the fetus in utero. IVC, inferior vena cava; SVC, superior vena cava. The figures give the approximate oxygen saturation of the blood at given points in the circulatory system.

The fetus also synthesizes a specific protein, e-fetoprotein (AFP) in its liver. The peak of AFPP is reached between the 12^{th} and 16^{th} gestational weeks, after which a decline occurs until term. The protein is secreted in the fetal urine and swallowed by the fetus, to be degraded in its gut. If the fetus is unable to swallow, as in cases of anencephaly, the level of AFP in the amniotic fluid rises.

III. Human Embryonic Development

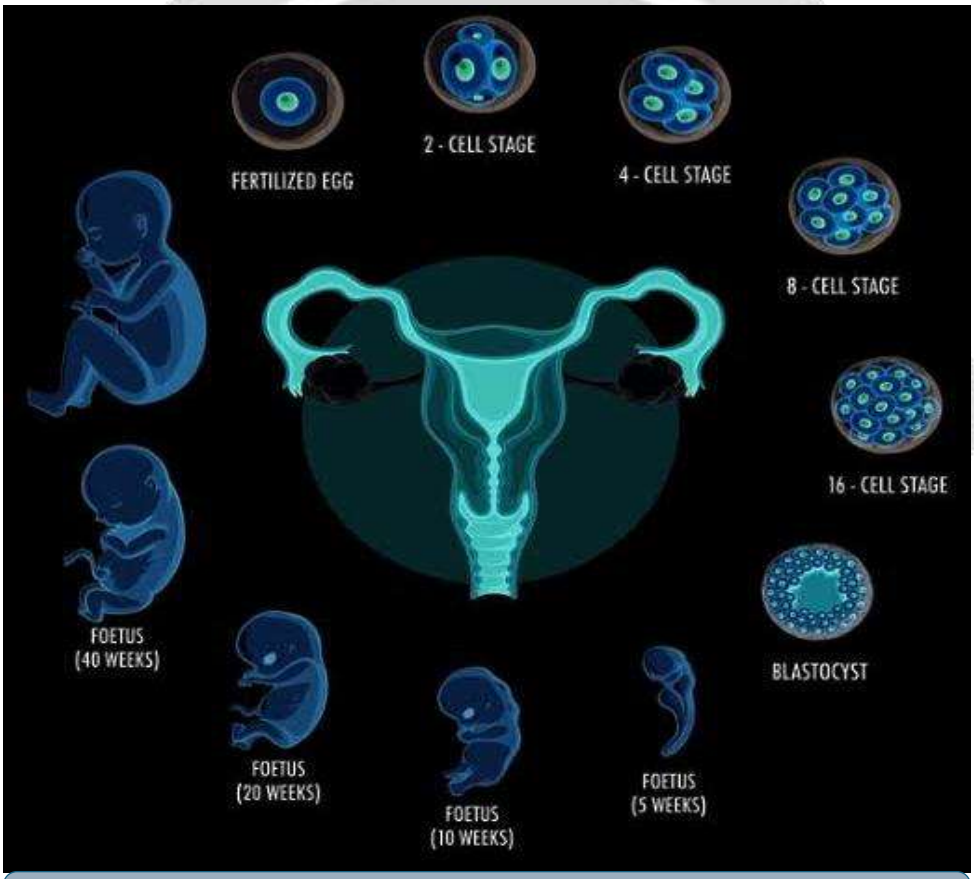

Figure : 2-Human Embryonic Development.

Human embryonic development is a complex and fascinating process that transforms a single fertilized cell into a fully formed human being. It is divided into several stages, each marked by specific milestones. Here is a detailed overview of the stages of human embryonic development:

- ***Fertilization (Week 0):*** Fertilization occurs when a sperm cell penetrates an egg cell, resulting in the formation of a zygote. This usually happens in the fallopian tube.
- ***Cleavage (Week 1):*** After fertilization, the zygote undergoes rapid cell division, forming a solid ball of cells called a morula.
- ***Blastocyst Formation (Week 1-2):*** The morula continues to divide and forms a hollow structure called a blastocyst. The blastocyst consists of two distinct cell populations: the inner cell mass, which will give rise to the embryo, and the outer trophoblast cells, which will form the placenta.
- ***Implantation (Week 2):*** The blastocyst implants into the uterine wall, where it receives nourishment from the mother's blood supply.
- ***Gastrulation (Week 2-3):*** Gastrulation is a crucial stage where the blastocyst undergoes dramatic changes. The inner cell mass differentiates into three germ layers: the ectoderm, mesoderm, and endoderm. The ectoderm gives rise to the nervous system, skin, and hair. The mesoderm gives rise to muscles, bones, and the circulatory system. The endoderm gives rise to the digestive system, liver, and lungs.
- ***Neurulation (Week 3-4):*** Neurulation is the process by which the neural plate folds and fuses to form the neural tube, which will develop into the brain and spinal cord.
- ***Organogenesis (Week 4-8):*** Organogenesis is the stage where the major organ systems begin to develop from the three germ layers. This stage is characterized by rapid cell proliferation, migration, and differentiation. By the end of this stage, all major organs and structures are formed, although they continue to grow and mature throughout the pregnancy.
- ***Fetal Development (Week 9-Birth):*** The fetal stage begins around the 9th week after fertilization and lasts until birth. During this stage, the fetus grows and develops rapidly, and its organs and systems become more specialized and functional. By the end of the fetal stage, the fetus is fully formed and ready for birth.

IV. ABNORMAL FETAL GROWTH

Fetal growth abnormalities are commonly diagnosed using criteria such as low birth weight, macrosomia, small-for-gestational age (SGA) and large-for-gestational age (LGA). Labeling fetuses as SGA or LGA based on normative values from a fetal growth standard is analogous to diagnosing malnutrition in children using a weight-for-age chart. Pediatric weight-for-age percentiles have been developed through the longitudinal follow-up of normal children with serial measurements obtained at regular intervals. Under this formulation, children who fall below the 3rd percentile or above the 97th percentile of weight for age are labeled malnourished. The theoretical basis for using the 3rd and 97th percentile cut-offs from such a weight-for-age standard is similar to the rationale of statistical inference using a P-value cut-off of 0.05 for rejecting the null hypothesis (i.e. a 2.5% error rate that is two tailed). It is also analogous to using the mean weight $\pm$ 2 SD of a standard population as cut-offs for abnormal weight-for-age, since approximately 5% of subjects will fall outside this range. Although by definition 3% of normal

children will fall below the 3rd percentile weight-for-age cut-off (false positives), the probability that a child with undernutrition will fall below this cut-off is higher than 3% (with the magnitude of this latter probability a function of the severity of the pathologic process causing the under nutrition)8, 9. One important feature of this method of identifying abnormal growth is the use of normal children in the creation of the standard. This issue is of particular relevance because many fetal growth references are based on fetuses from normal and abnormal pregnancies, without sufficient acknowledgment of the implications for normative interpretation using percentiles.

• Small-for-gestational age

A fetus is labeled SGA if its size (e.g. estimated fetal weight, estimated abdominal circumference) falls below some cut-off percentile of size for gestational age (e.g. the 3rd percentile). Although the SGA label implies fetal growth restriction, such fetuses will include some normal yet constitutionally small fetuses by definition; the ratio of constitutionally small fetuses to fetuses that are small because of an abnormal, pathologic process will depend on the prevalence of such illnesses in the population. For instance, among normal pregnancies, 3% of fetuses will fall below the 3rd percentile of a fetal growth standard and all of these will be constitutionally small, whereas among women with pre-eclampsia substantially more than 3% of fetuses will fall below the 3rd percentile, and a substantial fraction of such fetuses will be pathologically small (i.e. growth restricted).

• Fetal growth restriction

Fetuses whose growth potential has been compromised are referred to as growth restricted. Just as all SGA fetuses may not be growth restricted (some may be constitutionally small), growth-restricted fetuses may or may not be SGA. For instance, a fetus whose growth trajectory falls sequentially from a stable 60th percentile of estimated fetal weight to the 50th, 40th and 30th percentiles of estimated fetal weight for gestational age over several weeks could be labeled growth-restricted though not SGA10. Similarly, a fetus whose growth is substantially compromised owing to maternal smoking could be labeled growth-restricted even if the estimated fetal weight for gestational age is above the 10th percentile.

• Large-for-gestational age and excessive fetal growth

A fetus is labeled LGA if its size (e.g. estimated fetal weight, estimated abdominal circumference) is above the 90th or 97th percentile (or other cut-off) of size for gestational age. As with SGA fetuses and growth-restricted fetuses, LGA fetuses will include normal (i.e. constitutionally large) fetuses by definition, and fetuses with excessive fetal growth do not have to be LGA. The ratio of constitutionally large fetuses to fetuses that are large because of a pathologic process (such as maternal diabetes mellitus) will depend on the prevalence of such pregnancy complications in the population.

• Low birth weight and macrosomia

Low birth weight (< 2500 g) and macrosomia (birth weight ≥ 4000 or ≥ 4500 g) are indices of size with substantial utility for various reasons including ease of measurement and strong correlation with adverse perinatal outcomes. The 10th percentile value at 37 weeks' gestation of current fetal growth references approximates the low birth weight cut-off (2452 g for females and 2552 g for males in the Canadian reference11 and 2484 g for females and 2596 g for males in the United States reference12). The 90th and 97th percentiles of current fetal growth references loosely approximate the macrosomia cut-offs. The limitation of the low birth weight index arises because discounting gestational age makes the index a heterogeneous entity that includes both fetuses that are preterm and those that are SGA.Morphologic heterogeneity in growth restriction and excessive growth Growth-restricted fetuses have been traditionally categorized as symmetrically or asymmetrically growth restricted because of differences in both etiology and prognosis13. Symmetrical growth restriction is believed to occur as a result of a global insult such as aneuploidy or a viral infection early in pregnancy, while asymmetrical growth restriction – with a brain-sparing effect – is thought to be the result of complications later in pregnancy (e.g. pre-eclampsia). These findings have been challenged, however, with studies showing asymmetrical growth associated with aneuploidy14, symmetrical growth associated with pre-eclampsia15 and evidence of morbidity despite brain sparing in asymmetrical growth-restricted fetuses16.Asymmetric increases in abdominal circumference after 32 weeks' gestation characterize excessive fetal growth seen in diabetic pregnancies. Fetuses of diabetic mothers have an excess deposition of fat and more muscle growth in the interscapular areas and abdomen17. These morphologic differences are responsible for substantially higher rates of shoulder dystocia among macrosomic fetuses of diabetic pregnancies compared to macrosomic fetuses of non-diabetic pregnancies18.

V. VARIATION IN GROWTH

There is much variation in the growth of the human fetus. When the fetal size is less than expected, the condition is known as intrauterine growth restriction also called fetal growth restriction; factors affecting fetal growth can be maternal, placental, or fetal.[19]Maternal factors include maternal weight, body mass index, nutritional state, emotional stress, toxin exposure (including tobacco, alcohol, heroin, and other drugs which can also harm the fetus in other ways), and uterine blood flow.Placental factors include size, microstructure (densities and architecture), umbilical blood flow, transporters and binding proteins, nutrient utilization, and nutrient production.Fetal factors include the fetal genome, nutrient production, and hormone output. Also, female fetuses tend to weigh less than males, at full term.[19]Fetal growth is often classified as follows: small for gestational age (SGA), appropriate for gestational age (AGA), and large for gestational age (LGA).[20] SGA can result in low birth weight, although premature birth can also result in low birth weight. Low birth weight increases the risk for perinatal mortality (death shortly after birth), asphyxia, hypothermia, polycythemia, hypocalcemia, immune dysfunction, neurologic abnormalities, and other long-term health problems. SGA may

be associated with growth delay, or it may instead be associated with absolute stunting of growth.

VI. CARDIOVASCULAR SYSTEM

The circulatory pattern of the fetus is shown in Figure 4.1. It should be noted that over 50% of the cardiac output passes through the umbilical arteries to perfuse the pla centa. The cardiac output increases to term, at which time about 200 ml/kg per minute is usual. The heart rate lies between 110 and 150 bpm to maintain this output. The fetal blood pressure also increases through the pregnancy and, after the 36th week, has a mean of 75 mmlig systolic, 55 mmHg diastolic.Haemopoiesis commences in the villous capillaries but from the second trimester liver production becomes dominant. The red cell count, the haemoglobin level and the packed cell volume increase as pregnancy advances Most of the erythrocytes contain fetal haemoglobin (HbF). At 15 weeks gestation all the cells contain HbF; by the 36th week 70% of the erythrocytes contain HbE, and 30% adult haemoglobin, but there are wide variations. Cells contain ing HbF are able to absorb more oxygen at a given Po They are more resistant to haemolysis but less resistant to trauma than cells containing adult haemoglobin.

VII. LUNGS

In the early embryo the lungs are made up of epithelial tubes surrounded by mesoderm. With further development the epithelium becomes folded and glandular to form primi tive alveoli. By the 22nd gestational week a capillary system has developed and the lungs are capable of gas exchange.By term, three or four generations of alveoli have developed and been replaced. Their epithelium, which has a cuboidal appearance, becomes flattened with the first breath. By the 24th week, fluid fills the alveoli and the passages. There are two principal alveolar cell types, the flatter type I pneumo cytes, which facilitate gas exchange, and the cuboidal type Il pneumocytes that by the 24th week begin to secrete a surface active lipoprotein surfactant. Surfactant facilitates lung expansion at birth and helps the air-containing lung to maintain its normal volume. However, until the 35th week the amount of surfactant may be insufficient for some babies to expand their lungs after birth, and hyaline membrane disease may develop.The fetus makes respiratory movements (breathing) from early in pregnancy. At first they are sporadic, but by mid pregnancy the movements become regular and increase in frequency as the pregnancy advances. Respiratory activity results in the inspiration of amniotic fluid into the bronchi oles but no further, as the fluid secreted into the alveoli is under higher pressure. The reduction in fetal breathing move- ments when the fetus is subjected to chronic hypoxia can be observed during ultrasound examination. Acute episodes of hyponia in late pregnancy of during the birth may stimulate gamping. This fetal gasping draws the amniotic fluid, which often contains meconium, deeper into the lungs.[23]

VIII. GASTROINTESTINAL TRACT

In the uterus the fetal intestinal tract is relatively quiet Some of the swallowed amniotic fluid, and the cellular material it contains, enters the gut, where it is acted upon by the exurymes and bacteria to produce meconium. The meco nium remains in the gut unless an episode of severe hyposia leads to contractions of the goat, at which time the meconium is expelled to mix with the amniotic fluid.

IX. RENAL SYSTEM

The fetal kidney develops from the metanephros, and new glomeruli continue to be formed until the 36th gestational week. Urine is secreted and expelled into the amniotic fhuid from the tith week and probably earlier. Its tate of flow increases as term approaches.[1]

X. IMMUNE SYSTEM

In early pregnancy the fetus has a poor capacity to pro duce antibodies in response to invasion by maternal anti ges or by bacteria. From the 20th week (perhaps earlier) it becomes able to mount an immune response to a chal lenge.[4] The fetal response is supplemented by the transfer of maternal antibody molecules (provided that they are not too large in size) to the ferus, which provide it with passive protection that may persist for some weeks after birth.

XI. MUSCULAR SYSTEM

Almost weightless in its amniotic capsule, the fetus makes movements from an early age. As the pregnancy advances the fetal movements become stronger, and occur more often. Bouts of activity are followed by periods when the fetus seems to be sleeping. These movements strenghen the fetal muscles and a count of theth gives an indication of fetal wellbeing.

XII. ENDOCRINE ACTIVITY

The fetal hypothalamus secretes corticotrophin-releasing hormone (CRH) by the 13th week, thyrotrophin-releasing hormone (TRH) gonadotrophin-telcasing hormone (GnRI) and somatostatin by the 15th week, and growth hormone-releasing hormone (Ghi) by the 18th work after fertilization. Growth hormone (GH) can be released by the fetal pituitary By the 7th week and luteinizing hormone (LH) and follide stimulating hormone (FSH) by 9 weeks. Although adrenocor ticotrophic hormone (ACTH) is detectable by the 10th week the pituitary-adrenal axis remains immature and the adhenal gland only becomes sensitive to ACTH late in pregnancy. This a possibly because the main source of ACTH is the placenta Thyroid stimulating hormone (TSH) is released from the 14th week, but T, and T, levels remain low throughout Pregnancy. Immediately after birth there is a surge of TSH, which leads to a rapid transient rise in T, and T, and a fall in reverse T.The posterior lobe of the fetal pituitary gland secretes oxytocin from the second trimester and levels rise during labour. Arginine vasotocin is detectable from the 9th week but its function is not understood. Arginine vasopressin is secreted from the 12th week and plays

a key role in cardio vascular function under stress conditions.[6]Antinatriuretic factor (ANF) is released from the atria (predominantly from the right) in response to pressure changes, this helps regulate blood volume by increasing glomerular filtration. Another key component for fetal car diovascular homeostasis is the renin-angiotensin-aldos terone system. Fetal renin levels are 20 times greater than adult levels, and renin is released in response to a fall in blood volume. Aldosterone is detectable from the second trimester. Aldosterone increases renal sodium reabsorption and has a negative feedback effect on renin release.Insulin is present in the fetal pancreas by the 10^{th} week, but pancreatic release of insulin is relatively insensitive until 28 weeks. The two growth factors IGF-1 and IGF-2 increase with gestation, especially from 33 weeks, and are related to fetal placental lactogen levels.

XIII. RISK FACTOR

• Genetic Factors: Genetic abnormalities can occur due to mutations or errors in the genetic material passed from the parents to the fetus. These abnormalities can lead to conditions such as Down syndrome, cystic fibrosis, and sickle cell anemia.

• Environmental Factors: Exposure to harmful substances or toxins during pregnancy can affect fetal development. These substances can include alcohol, tobacco, drugs, and certain medications. They can increase the risk of birth defects, developmental delays, and other health problems in the fetus.

• Maternal Health: The health of the mother plays a crucial role in fetal development. Maternal conditions such as diabetes, hypertension, and infections can impact the fetus. Poor maternal nutrition and inadequate prenatal care can also lead to developmental problems in the fetus.

• Placental Issues: Problems with the placenta, such as placental insufficiency or placenta previa, can affect fetal development by reducing the supply of oxygen and nutrients to the fetus.

• Fetal Growth Restriction: This condition occurs when the fetus does not grow at the expected rate. It can be caused by factors such as placental problems, maternal health issues, or genetic factors. Fetal growth restriction can lead to low birth weight and other complications.

• Structural Abnormalities: Structural abnormalities can occur in various organs or body systems of the fetus. These abnormalities can be detected through prenatal screening tests and may require medical intervention after birth.

• Multiple Pregnancies: In cases of multiple pregnancies (e.g., twins, triplets), there is an increased risk of complications such as premature birth, low birth weight, and developmental issues.

• Maternal Age: Advanced maternal age (over 35) is associated with an increased risk of chromosomal abnormalities and other complications that can affect fetal development.

• Infections: Certain infections contracted during pregnancy, such as rubella, cytomegalovirus, and Zika virus, can lead to developmental problems in the fetus.

• Exposure to Radiation: Radiation exposure, whether from medical procedures or environmental sources, can harm fetal development and increase the risk of birth defects.

XIV. CONCLUSION

The fetal growth literature includes some potentially confusing terms and concepts and an enormous body of sometimes conflicting evidence. The confusion derives in part from the relatively inaccessible nature of the fetus, which presents a challenge with regard to accurate measurement of size and growth. Future developments, including qualitatively improved methods for assessing fetal size, will bring coherence to this field through new concepts, better theories and more accurate evidence.

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*****Thank You*****