NAME: OLUSEYI OMOWUMI ROSEMARY.

MATRICULATION NUMBER: 16/MHS07/028.

LEVEL: 400LEVEL.(carryover student).

DEPARTMENT: PHARMACOLOGY.

COURSE CODE: PHS 204.

ASSIGNMENT.

1) Discuss lactation and gestation period in a normal female.

2) Physiology of lactation and details on the physiology of pregnancy in a normal woman.

Answers

1) Lactation describes the secretion of milk from the mammary glands and the period of time that a mother lactates to feed her young. The process occurs in all female mammals, although it predates the origin of mammals.

In humans the process of feeding milk is called breastfeeding or nursing.

The chief function of lactation is to provide nutrition and immune protection to the young after birth. In almost all mammals, lactation induces a period of infertility, which serves to provide the optimal birth spacing for survival of the offspring.

In most species, milk comes out of the mother’s nipples; however, the platypus (a non-placental mammal) releases milk through ducts in its abdomen. In only one species of mammal, the dayak fruit bat, is milk production a normal male function.

In some other mammals, the male may produce milk as the result of a hormone imbalance. This phenomenon may also be observed in newborn infants as well (for instance, witch’s milk).

**Galactopoiesis** is the maintenance of milk production. This stage requires prolactin and oxytocin.

**Preparation for Lactation**

By the fifth or sixth month of pregnancy, the breasts are ready to produce milk. During the latter part of pregnancy, the woman’s breasts enter into the lactogenesis I stage. This is when the breasts make colostrum, a thick, sometimes yellowish fluid.

At this stage, high levels of progesterone inhibit most milk production. It is not a medical concern if a pregnant woman leaks any colostrum before her baby’s birth, nor is it an indication of future milk production.

At birth, prolactin levels remain high, while the delivery of the placenta results in a sudden drop in progesterone, estrogen, and human placental lactogen levels. This abrupt withdrawal of progesterone in the presence of high prolactin levels stimulates the copious milk production of the lactogenesis II stage.

When the breast is stimulated, prolactin levels in the blood rise and peak in about 45 minutes, then return to the pre-breastfeeding state about three hours later. The release of prolactin triggers the cells in the alveoli to make milk.

**Colostrum**

Colostrum is the first milk a breastfed baby receives. It contains higher amounts of white blood cells and antibodies than mature milk, and is especially high in immunoglobulin A (IgA), which coats the lining of the baby’s immature intestines, and helps to prevent pathogens from invading the baby’s system. Secretory IgA also helps prevent food allergies. Over the first two weeks after the birth, colostrum production slowly gives way to mature breast milk.

**THE PHYSIOLOGY OF LACTATION**

The breasts, unlike most of the other organs, continue to increase in size after childbirth. Although mammary growth begins during pregnancy under the influence of ovarian and placental hormones, and some milk is formed, copious milk secretion sets in only after delivery. Since lactation ensues after a premature birth, it would appear that milk production is held back during pregnancy. The mechanism by which this inhibitory effect is brought about, or by which lactation is initiated at delivery, has long been the subject of an argument that revolves around the opposing actions of estrogen, progesterone, and prolactin, as studied in laboratory animals, goats, and cattle. During pregnancy the combination of estrogen and progesterone circulating in the blood appears to inhibit milk secretion by blocking the release of prolactin from the pituitary gland and by making the mammary gland cells unresponsive to this pituitary hormone. The blockade is removed at the end of pregnancy by the expulsion of the placenta and the loss of its supply of hormones, as well as by the decline in hormone production by the ovaries, while sufficient estrogen remains in circulation to promote the secretion of prolactin by the pituitary gland and so favour lactation.

For lactation to continue, necessary patterns of hormone secretion must be maintained; disturbances of the equilibrium by the experimental removal of the pituitary gland in animals or by comparable diseased conditions in humans quickly arrest milk production. Several pituitary hormones seem to be involved in the formation of milk, so that it is customary to speak of a lactogenic (“milk-producing”) complex of hormones. To some degree, the role of the pituitary hormones adrenocorticotropin, thyrotropin, and growth hormone in supporting lactation in women is inferred from the results of studies done on animals and from clinical observations that are in agreement with the results of animal studies. Adrenal corticoids also appear to play an essential role in maintaining lactation.

The stimulus of nursing or suckling supports continued lactation. It acts in two ways: it promotes the secretion of prolactin (and possibly other pituitary hormones of value in milk formation), and it triggers the release of yet another hormone from the pituitary gland—oxytocin, which causes the contraction of special muscle cells around the alveoli in the breast and ensures the expulsion of milk. It is in this way that a baby’s sucking at one breast may cause an increase in milk flow from both, so that milk may drip from the unsuckled nipple. About 30 seconds elapse between the beginning of active suckling and the initiation of milk flow.

The nerve supply to the mammary glands is not of great significance in lactation, for milk production is normal after the experimental severing of nerves to the normal mammary glands in animals or in an udder transplanted to the neck of a goat. Milk ejection, or “the draught,” in women is readily conditioned and can be precipitated by the preparations for nursing. Conversely, embarrassment or fright can inhibit milk ejection by interfering with the release of oxytocin; alcohol, also, is known to block milk ejection in women, again by an action on the brain. Beyond its action on the mammary glands, oxytocin affects uterine muscle, so that suckling can cause contractions of the uterus and may sometimes result in cramp. Since oxytocin release occurs during sexual intercourse, milk ejection in lactating women has been observed on such occasions. Disturbance of oxytocin secretion, or of the milk-ejection reflex, stops lactation just as readily as a lack of the hormones necessary for milk production, for the milk in the breast is then not extractable by the infant. Many instances of nursing failure are due to a lack of milk ejection in stressful circumstances; fortunately, treatment with oxytocin, coupled with the reassurance gained from a successful nursing, is ordinarily successful in overcoming the difficulty.

Suckling can initiate lactation in nonpregnant women. This has been seen most often in women of childbearing age but also has been observed in older persons. A baby who had lost his mother was suckled by his 60-year-old grandmother, who had borne her last child 18 years before. The grandmother produced milk after a few days and continued to nurse the baby until he was a year old and could walk. Rarely, lactation has been reported to set in after operations on the chest; in such instances it is attributed to injury or irritation of the nerves in this region. Such observations argue against the possibility that lactation continues simply as a consequence of emptying the breasts.

**GESTATION PERIOD**

This relates to the age of an embryo or fetus (or newborn infant). In human obstetrics, this age is often defined as the time elapsed since 14 days prior to fertilization; this is approximately the duration since the woman’s last menstrual period began.

In humans, the fetal stage of prenatal development starts at the beginning of the 11th week in gestational age, which is the ninth week after fertilization. Since the precursors of all the major organs are created by this time, the fetal period is described both by organ and a list of changes by weeks of gestational age.

All major structures are already formed in the fetus, but they continue to grow and develop. Therefore, the fetus is not as sensitive to damage from environmental exposures as the embryo, though toxic exposures often cause physiological abnormalities, growth retardation, or minor congenital malformations.

At the start of the fetal stage, the fetus is typically about 30 millimeters in length from crown to rump and weighs about eight grams. The head makes up nearly half of the fetus’ size. The four-chamber heart is finishing development, and the embryonic tail goes away. The breathing-like movement of the fetus is necessary for stimulation of lung development, rather than for obtaining oxygen.

Week 10: Finger nails and hair start to grow. The heart, hands, feet, brain, and other organs are present, but are only at the beginning of development and have minimal operation.

Week 11: Nearly all structures and organs are formed. Fingers and toes are separated and the genitals begin to take on the proper gender characteristics.

Week 12: The digestive system and liver function. The pancreas makes insulin.

Week 13: The fetus begins to get its nourishment from the placenta and the veins and organs are visible through the skin.

Week 14: The kidneys produce urine, and the liver makes bile. In boys, the prostate gland develops, while in girls the ovaries move from the abdomen to the pelvis.

Weeks 15 to 16: The heart pumps out 25 quarts of blood a day and the fetal structures are looking more normal.

Week 17: The fetus start to move its joints and the retina becomes sensitive to light. It weighs about five ounces.

Week 18: The fetus starts to hear and is startled by noise. Its skin is starting to grow a protective, wax-like layer and tiny air spaces begin to form in the lungs and the vocal cords.

Week 19: The brain is designating areas for the five senses.

Week 20: The fetus weighs about 10 ½ oz, swallows more, and produces meconium.

Week 21: The eyebrows and lids are present, and for the female fetus, the vagina begins to form.

Week 22: Tiny tooth buds beneath the gums develops. The eyes form, but the irises lack pigment.

Week 23: The fetus weighs over a pound. It can feel movements and hear sounds. Blood vessels in his/her lungs are developing to prepare for breathing.

Week 24: The fetus is almost a foot long. The lungs are developing branches of the respiratory tree as well as cells that produce surfactant, a substance that will help the air sacs inflate once born.

Week 25: The amount of body fat rapidly increases. The lungs are not fully mature. The bones are fully developed, but are still soft and pliable. Thalamic brain connections form that mediate sensory input. Iron, calcium, and phosphorus become more abundant. The fingernails reach the end of the fingertips. The lanugo, or fine hair, begins to disappear, until it is gone (except on the upper arms and shoulders). Small breast buds are present on both sexes. Head hair becomes coarse and thicker.

Birth is imminent and occurs around the 40th week. The fetus is considered full-term between weeks 37 and 40, which means that the fetus is considered sufficiently developed for life outside the uterus.

**2) PHYSIOLOGY OF PREGNANCY IN A NORMAL WOMAN**

Pregnancy causes physiologic changes in all maternal organ systems; most return to normal after delivery. In general, the changes are more dramatic in multifetal than in single pregnancies.

**Cardiovascular**

Cardiac output (CO) increases 30 to 50%, beginning by 6 weeks gestation and peaking between 16 and 28 weeks (usually at about 24 weeks). It remains near peak levels until after 30 weeks. Then, CO becomes sensitive to body position. Positions that cause the enlarging uterus to obstruct the vena cava the most (eg, the recumbent position) cause CO to decrease the most. On average, CO usually decreases slightly from 30 weeks until labor begins. During labor, CO increases another 30%. After delivery, the uterus contracts, and CO drops rapidly to about 15 to 25% above normal, then gradually decreases (mostly over the next 3 to 4 weeks) until it reaches the prepregnancy level at about 6 weeks postpartum.

The increase in CO during pregnancy is due mainly to demands of the uteroplacental circulation; volume of the uteroplacental circulation increases markedly, and circulation within the intervillous space acts partly as an arteriovenous shunt. As the placenta and fetus develop, blood flow to the uterus must increase to about 1 L/min (20% of normal CO) at term. Increased needs of the skin (to regulate temperature) and kidneys (to excrete fetal wastes) account for some of the increased CO.

To increase CO, heart rate increases from the normal 70 to as high as 90 beats/min, and stroke volume increases. During the 2nd trimester, blood pressure (BP) usually drops (and pulse pressure widens), even though CO and renin and angiotensin levels increase, because uteroplacental circulation expands (the placental intervillous space develops) and systemic vascular resistance decreases. Resistance decreases because blood viscosity and sensitivity to angiotensin decrease. During the 3rd trimester, BP may return to normal. With twins, CO increases more and diastolic BP is lower at 20 weeks than with a single fetus.

Exercise increases CO, heart rate, oxygen consumption, and respiratory volume/min more during pregnancy than at other times.

The hyperdynamic circulation of pregnancy increases frequency of functional murmurs and accentuates heart sounds. X-ray or ECG may show the heart displaced into a horizontal position, rotating to the left, with increased transverse diameter. Premature atrial and ventricular beats are common during pregnancy. All these changes are normal and should not be erroneously diagnosed as a heart disorder; they can usually be managed with reassurance alone. However, paroxysms of atrial tachycardia occur more frequently in pregnant women and may require prophylactic digitalization or other antiarrhythmic drugs. Pregnancy does not affect the indications for or safety of cardioversion.

**Hematologic**

Total blood volume increases proportionally with cardiac output, but the increase in plasma volume is greater (close to 50%, usually by about 1600 mL for a total of 5200 mL) than that in red blood cell (RBC) mass (about 25%); thus, hemoglobin (Hb) is lowered by dilution, from about 13.3 to 12.1 g/dL. This dilutional anemia decreases blood viscosity. With twins, total maternal blood volume increases more (closer to 60%).

WBC count increases slightly to 9,000 to 12,000/mcL. Marked leukocytosis (≥ 20,000/mcL) occurs during labor and the first few days postpartum.

Iron requirements increase by a total of about 1 g during the entire pregnancy and are higher during the 2nd half of pregnancy—6 to 7 mg/day. The fetus and placenta use about 300 mg of iron, and the increased maternal RBC mass requires an additional 500 mg. Excretion accounts for 200 mg. Iron supplements are needed to prevent a further decrease in Hb levels because the amount absorbed from the diet and recruited from iron stores (average total of 300 to 500 mg) is usually insufficient to meet the demands of pregnancy.

**Urinary**

Changes in renal function roughly parallel those in cardiac function. Glomerular filtration rate (GFR) increases 30 to 50%, peaks between 16 and 24 weeks gestation, and remains at that level until nearly term, when it may decrease slightly because uterine pressure on the vena cava often causes venous stasis in the lower extremities. Renal plasma flow increases in proportion to GFR. As a result, blood urea nitrogen (BUN) decreases, usually to < 10 mg/dL (< 3.6 mmol urea/L), and creatinine levels decrease proportionally to 0.5 to 0.7 mg/dL (44 to 62 micromole/L). Marked dilation of the ureters (hydroureter) is caused by hormonal influences (predominantly progesterone) and by backup due to pressure from the enlarged uterus on the ureters, which can also cause hydronephrosis. Postpartum, the urinary collecting system may take as long as 12 weeks to return to normal.

Postural changes affect renal function more during pregnancy than at other times; ie, the supine position increases renal function more, and upright positions decrease renal function more. Renal function also markedly increases in the lateral position, particularly when lying on the left side; this position relieves the pressure that the enlarged uterus puts on the great vessels when pregnant women are supine. This positional increase in renal function is one reason pregnant women need to urinate frequently when trying to sleep.

**Respiratory**

Lung function changes partly because progesterone increases and partly because the enlarging uterus interferes with lung expansion. Progesterone signals the brain to lower carbon dioxide (CO2) levels. To lower CO2 levels, tidal and minute volume and respiratory rate increase, thus increasing plasma pH. oxygen consumption increases by about 20% to meet the increased metabolic needs of the fetus, placenta, and several maternal organs. Inspiratory and expiratory reserve, residual volume and capacity, and plasma PCO2 decrease. Vital capacity and plasma PCO2 do not change. Thoracic circumference increases by about 10 cm.

Considerable hyperemia and edema of the respiratory tract occur. Occasionally, symptomatic nasopharyngeal obstruction and nasal stuffiness occur, eustachian tubes are transiently blocked, and tone and quality of voice change.

Mild dyspnea during exertion is common, and deep respirations are more frequent.

Gastrointestinal (GI) and hepatobiliary

As pregnancy progresses, pressure from the enlarging uterus on the rectum and lower portion of the colon may cause constipation. GI motility decreases because elevated progesterone levels relax smooth muscle. Heartburn and belching are common, possibly resulting from delayed gastric emptying and gastroesophageal reflux due to relaxation of the lower esophageal sphincter and diaphragmatic hiatus. Hydrochloric acid production decreases; thus, peptic ulcer disease is uncommon during pregnancy, and preexisting ulcers often become less severe.

Incidence of gallbladder disorders increases somewhat. Pregnancy subtly affects hepatic function, especially bile transport. Routine liver function test values are normal, except for alkaline phosphatase levels, which increase progressively during the 3rd trimester and may be 2 to 3 times normal at term; the increase is due to placental production of this enzyme rather than hepatic dysfunction.

**Endocrine**

Pregnancy alters the function of most endocrine glands, partly because the placenta produces hormones and partly because most hormones circulate in protein-bound forms and protein binding increases during pregnancy.

The placenta produces the beta subunit of human chorionic gonadotropin (beta-hCG), a trophic hormone that, like follicle-stimulating and luteinizing hormones, maintains the corpus luteum and thereby prevents ovulation. Levels of estrogen and progesterone increase early during pregnancy because beta-hCG stimulates the ovaries to continuously produce them. After 9 to 10 weeks of pregnancy, the placenta itself produces large amounts of estrogen and progesterone to help maintain the pregnancy.

The placenta produces a hormone (similar to thyroid-stimulating hormone) that stimulates the thyroid, causing hyperplasia, increased vascularity, and moderate enlargement. Estrogen stimulates hepatocytes, causing increased thyroid-binding globulin levels; thus, although total thyroxine levels may increase, levels of free thyroid hormones remain normal. Effects of thyroid hormone tend to increase and may resemble hyperthyroidism, with tachycardia, palpitations, excessive perspiration, and emotional instability. However, true hyperthyroidism occurs in only 0.08% of pregnancies.

The placenta produces corticotropin-releasing hormone (CRH), which stimulates maternal adrenocorticotropic hormone (ACTH) production. Increased ACTH levels increase levels of adrenal hormones, especially aldosterone and cortisol, and thus contribute to edema.

Increased production of corticosteroids and increased placental production of progesterone lead to insulin resistance and an increased need for insulin, as does the stress of pregnancy and possibly the increased level of human placental lactogen. Insulinase, produced by the placenta, may also increase insulin requirements, so that many women with gestational diabetes develop more overt forms of diabetes.

The placenta produces melanocyte-stimulating hormone (MSH), which increases skin pigmentation late in pregnancy.

The pituitary gland enlarges by about 135% during pregnancy. The maternal plasma prolactin level increases by 10-fold. Increased prolactin is related to an increase in thyrotropin-releasing hormone production, stimulated by estrogen. The primary function of increased prolactin is to ensure lactation. The level returns to normal postpartum, even in women who breastfeed.

**Dermatologic**

Increased levels of estrogens, progesterone, and MSH contribute to pigmentary changes, although exact pathogenesis is unknown. These changes include

i) Melasma (mask of pregnancy), which is a blotchy, brownish pigment over the forehead and malar eminences

ii) Darkening of the mammary areolae, axilla, and genital.

iii) Linea nigra, a dark line that appears down the midabdomen

Incidence of spider angiomas, usually only above the waist, and of thin-walled, dilated capillaries, especially in the lower legs, increases.