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Questions

Discuss lactation and gestation period in a normal female

Answers

Pregnancy and lactation are anabolic states that are orchestrated via hormones to produce a redirection of nutrients to highly specialized maternal tissues characteristic of reproduction (i.e., placenta and mammary gland) and their transfer to the developing fetus or infant.

Hormonal changes during pregnancy.

Plasma levels of human chorionic gonadotropin increase immediately upon implantation of the ovum; the hormone is detectable in urine within 2 wk of implantation. It reaches a peak at ≈8 wk of gestation and then declines to a stable plateau until birth. Human chorionic gonadotropin maintains corpus luteum function for 8–10 wk. Human placental lactogen (also called human chorionic somatomammotropin) has a structure that closely resembles growth hormone, and its rate of secretion appears to parallel placental growth and may be used as a measure of placental function. At its peak, the rate of secretion of placental lactogen is 1–2 g/d, far in excess of the production of any other hormones. Placental lactogen stimulates lipolysis, antagonizes insulin actions and may be important in maintaining a flow of energy-yielding substrates to the fetus. Placental lactogen along with prolactin from the pituitary may promote mammary gland growth. After delivery, placental lactogen rapidly disappears from the circulation.

The placenta becomes the main source of steroid hormones at weeks 8–10 of gestation. Before then, progesterone and estrogens are synthesized in the maternal corpus luteum. These hormones play essential roles in maintaining the early uterine environment and development of the placenta. The placenta takes over progesterone production, which increases throughout pregnancy. Progesterone, known as the hormone of pregnancy, stimulates maternal respiration; relaxes smooth muscle, notably in the uterus and gastrointestinal tract; and may act as an immunosuppressant in the placenta, where its concentration can be 50 times greater than in plasma. Progesterone may promote lobular development in the breast and is responsible for the inhibition of milk secretion during

pregnancy.
The secretion of estrogens from the placenta is complex (5). Estradiol and estrone are synthesized from the precursor dehydroepiandrosterone sulfate (DHEA-S), which is derived from both maternal and fetal blood. The synthesis of estriol is from fetal 16-α-hydroxy-dehydroepiandrosterone sulfate (16-OH-DHEA-S). The fetus is unable to synthesize pregnenolone, the precursor of DHEA-S and 16-OH-DHEA-S, and must get the precursor from the placenta. The placental secretion of estrogens also increases manyfold with the progression of pregnancy. The functions of high estrogen levels in pregnancy include stimulation of uterine growth, enhancement of uterine blood flow and possibly promotion of breast development. Because estrogen precursors originate in the fetus, maternal estrogen levels can be used as a measure of fetal viability.
The increased amount of estrogens during pregnancy also stimulates a population of cells (somatotrophs) in the maternal pituitary to become mammotrophs, or prolactin-secreting cells. The increased prolactin secretion probably helps promote mammary development. In addition, the increased number of pituitary mammotrophs at the end of pregnancy provides the large amounts of prolactin necessary to initiate and maintain lactation.

During pregnancy there is an increase in blood volume of ≈35–40%, expressed as a percentage of the nonpregnant value, that results principally from the expansion of plasma volume by ≈45–50% and of red cell mass by ≈15–20% as measured in the third trimester. Because the expansion of red cell mass is proportionally less than the expansion of plasma, hemoglobin concentration and hematocrit values fall in parallel with red cell volume. Hemoglobin and hematocrit values are at their lowest in the second trimester of pregnancy and rise again in the third trimester. For these reasons, trimester-specific values for hemoglobin and hematocrit are proposed for screening for anemia in pregnant women (6). Total plasma protein concentration falls from ≈70 to 60 g/L largely because of a fall in albumin concentration from ≈4 to 2.5 g/l00 mL near term. Plasma concentrations of α1-, α2- and β-globulins increase by ≈60%, 50% and 35%, respectively, whereas the γ-globulin fraction decreases by 13% (7). Estrogens are responsible for these changes in plasma proteins, which can be reproduced by administration of estradiol to nonpregnant women. Plasma levels of most lipid fractions, including triacylglycerol, VLDL, LDL and HDL, increase during pregnancy.

The average weight gained by healthy primigravidae eating without restriction is 12.5 kg (27.5 lb) (5). This weight gain represents two major components: 1) the products of conception: fetus, amniotic fluid and the placenta and 2) maternal accretion of tissues: expansion of blood and extracellular fluid, enlargement of uterus and mammary glands and maternal stores (adipose tissue).
Low weight gain is associated with increased risk of intrauterine growth retardation and

perinatal mortality. High weight gain is associated with high birth weight and secondarily with increased risk of complications related to fetopelvic disproportion. A large body of epidemiologic evidence now shows convincingly that maternal prepregnancy weight-for-height is a determinant of fetal growth above and beyond gestational weight gain. At the same gestational weight gain, thin women give birth to infants smaller than those born to heavier women. Because higher birth weights present lower risk for infants, current recommendations for weight gain during pregnancy are higher for thin women than for women of normal weight and lower for short overweight and obese women.

Recommendations for weight gain during pregnancy were formulated in recognition of the need to balance the benefits of increased fetal growth against the risks of labor and delivery complications and of postpartum maternal weight retention. The target range for desirable weight gain in each prepregnancy weight-for-height category is that associated with delivery of a full-term infant weighing between 3 and 4 kg. Recent evidence indicates that <50% of 622 women sampled in upstate New York gained weight within the ranges recommended and that weight gain greater than these recommended amounts placed them at risk for major weight gain postdelivery.

Lactation

Lactation, secretion and yielding of milk by females after giving birth. The milk is produced by the mammary glands, which are contained within the breasts.The establishment and maintenance of human lactation are under the influence of complex neuroendocrine control mechanisms . After parturition, elevated levels of prolactin and withdrawal of estrogens and progesterone results in the onset of milk secretion (lactogenesis). The breasts must have undergone appropriate growth and development beginning in puberty and completed during pregnancy for milk secretion to occur. The initiation of lactogenesis does not require infant sucking but lactation cannot be maintained unless the infant is put to the breast by 3 or 4 d postpartum. For the first 3–5 d postpartum the mammary secretion is termed “colostrum.” This early milk is thick and straw-colored, rich in minerals and immune factors (i.e., lactoferrin and secretory immunoglobulin A) and low in lactose and total protein. The concentration of lactose increases and that of sodium and chloride decrease as milk secretion is enhanced. The characteristics of mature milk are evident by day 10 of lactation.
With established lactation, prolactin is required for maintenance of milk production. Prolactin release into the circulation from mammotrophs in the anterior pituitary is in response to sucking. Prolactin secretion is mediated by a transient decline in the secretion of dopamine from the hypothalamus, which normally inhibits its secretion. Milk secretion continues as long as the infant continues to nurse more than once a day. The daily milk volume transferred to the infant increases from ≈50 mL on day 1 to 500 mL by day 5, ≈650 mL by 1 mo and 750 mL at 3 mo of lactation. Most women can secrete considerably more milk than needed by a

single infant. Milk secretion is continuous and the quantity produced is principally regulated by infant demand. Oxytocin release from the posterior pituitary results from neural impulses reaching the hypothalamus caused by sucking of the nursing infant. Circulating oxytocin causes contraction of myoepithelial cells that surround mammary alveoli and ducts, forcing milk into ducts of the nipple so that it can be removed by the infant. This response is termed “milk ejection” or “let-down” and can be initiated by the mere sight of the infant or by hearing the infant cry. Continuation of lactation and associated hyperprolactinemia inhibit ovarian activity by suppressing the pulsatile release of luteinizing hormone and by interfering with the secretion of gonadotropin-releasing hormone. This provides 98% protection from pregnancy during the first 6 mo of lactation if the nursing mother continues to be amenorrheic . Milk secretion ceases in 1 or 2 d when infant sucking or milk removal is terminated.