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ASSIGNMENT

Adaptations to pregnancy refers to the change in gait, postural parameters, as well as sensory feedback, due to the numerous anatomical, physiological and hormonal changes women experience during pregnancy.

The pregnant woman's body goes through some profound anatomical, physiologic, and biochemical changes to adapt to and support the entire pregnancy, which ultimately support the growing fetus. Although these physiologic changes are normal, often they can be misinterpreted as disease. These changes may also unmask or worsen a preexisting condition or disease, ultimately because the pregnant woman's body cannot adequately adapt to the changes of pregnancy. This includes the basic adaptations related to pregnancy, placental physiology and action, uterine activity physiology, and fetal heart rate regulation.

CHANGES IN THE GASTROINTESTINAL SYSTEM

Nausea and vomiting are the most frequent complaints involving the gastrointestinal system and usually happen in early pregnancy while heartburn happen primarily in late pregnancy. The gums become hyperemic and edematous during pregnancy and tend to bleed. The muscular wall of the esophagus is relaxed and this may cause reflux, which in turn can lead to esophagitis and
heartburn. The stomach and the intestines have decreased motility presumably due to the effect of progesterone on smooth muscle contractility. This causes an increase in the time that
it takes for the stomach to empty. Reduced gastric secretion has also been documented and it could account for the improvement of peptic ulcers sometimes observed in pregnancy. Decreased motility of the large intestine may lead to constipation. The liver is affected significantly by pregnancy. Cholestatic jaundice is considered to be the result of estrogen effect on elimination of bilirubin by the liver. The effect of estrogens also, is to increase protein synthesis in the liver, which leads to

increased production of fibrinogen and binding proteins. The liver enzymes are usually unaffected with the exception of alkaline phosphatase, which is increased at approximately two fold to four fold that is a result of a placental production. Pregnancy increases the size and decreases the motility of the gall bladder. The decreasing motility and increase in volume, combined with changes in the bile's composition, explain the
correlation between the incidence of cholelithiasis and pregnancy. CARDIOVASCULAR CHANGES Of all changes that happen in pregnancy, the single most
important is the one involving the cardiovascular system. Adequate cardiovascular adaptation secures good placental development and thus appropriate fetal growth. In brief, the cardiovascular changes involve a substantial change in the blood volume, cardiac output, heart rate, systemic arterial blood pressure, systemic vascular resistance, oxygen consumption and alterations in regional blood flow of various organ systems.

Blood Volume

Significant increases in the blood volume start taking place in the first trimester and continue until the mid third trimester, at approximately the 32nd to the 34th week. Beyond this point in
gestation, the blood volume plateaus. This pattern was established with studies that kept the patients in the left-lateral position to avoid vena cava compression. However, studies that kept the patient in the supine position had controversial results indicating a decline in the blood volume after 34 to 36 weeks. The average absolute increase in blood volume during pregnancy is about 1600 ml and in terms of percent change one should expect a 40 to 50 percent increase above pre-pregnancy levels. The increase in the blood volume is achieved by a combination of increases in the plasma volume and the RBC mass. The calculated plasma volume expansion is approximately 1300 ml and the volume of the RBC increases about 400 ml. This discordance in the change between the cellular elements of the blood and the liquid portion leads to the so called "physiologic anemia of pregnancy". The mechanisms leading to hypervolemia in pregnancy are still not entirely understood and seem to be multifactorial. Increased estrogen levels in pregnancy cause increased production of renin from the kidneys, the uterus and the liver and thus cause elevated renin plasma levels. The increase in renin, which stimulates aldosterone secretion, is associated with sodium retention and an increase in total body water. The roll of atrial natriuretic factor (ANF) in mediating

The increase in blood volume with pregnancy appears to serve the essential physiologic needs of both the mother and fetus. It ensures adequate supplies required for normal fetal growth and oxygenation even under circumstances that affect the maternal cardiac output (inferior vena cava compression). This increased blood volume also helps normal pregnant women to withstand hemorrhage equal to the volume of blood added to the circulation during the course of the normal pregnancy without any signs of decompensation.

Cardiac Output

It has been well established since the beginning of this century that the cardiac output increases an average of 50 percent during pregnancy. It is generally accepted that cardiac output begins to rise during the first trimester, probably around the tenth week of pregnancy and continues to rise up until the 24th week of gestation. Once it reaches the peak it stays rather stable. That was the case in most if not all of the studies that evaluated women in a left-lateral tilt while studies that placed women in the supine position have shown a rather false reduction in cardiac output which was primarily mediated by inferior vena cava compression. Cardiac output is a product of stroke volume and pulse rate. The rise in cardiac output early in pregnancy is disproportionately
greater than the increase in heart rate, and therefore is attributable to augmentation in stroke volume. As pregnancy advances, heart rate increases and becomes a more predominant factor in increasing cardiac output. At the late stages of pregnancy, the stroke volume declines to normal, non-pregnant values. The effect of maternal posture on cardiac output was demonstrated by a number of studies. A significant decrease (25 to 30 percent) in cardiac output, measured by dye dilution technique, was demonstrated in the supine position between the 38th and 40th weeks of pregnancy but not before the 24th week. These findings were confirmed recently by echocardiographic studies. Since heart rate was not affected significantly, positional decline in cardiac output was due to decreased stroke volume. The fall in cardiac output was also not associated with a significant change in blood pressure. This is probably due to an increase in peripheral vascular resistance. As many as 11 percent of women when placed in the supine position, will develop symptomatic hypotension and drop in the cardiac output which may lead to a loss of consciousness. These symptoms are relieved promptly with left-lateral positioning. In these particular patients who develop the symptoms, the cardiac output is not maintained despite the fact that they develop a significant increase in their heart rate. It is believed that the patients who become symptomatic are those who lack sufficient paravertebral collateral circulation to permit blood from the legs and the pelvic organs to bypass the occluded inferior vena cava.

Heart Rate during Normal Pregnancy

The baseline heart rate increases by about 10 to 20 beats per minute. This increase starts early in pregnancy and gradually continues to go upward with the highest values achieved at term. Some investigators, however, suggested that the total increase happens early in pregnancy and remains so throughout the remainder of gestation. In twin gestations, the rise of the heart rate is more pronounced and it can reach as much as 40 percent above the non-pregnant state. A change also from the supine position to the lateral position may cause the heart rate to drop slightly. Blood Flow Changes in Various Organ Systems During Pregnancy The most profound changes in regional blood flow occur in the uterus with a 5 to 10 fold increase. This change starts early in pregnancy and continues until almost term. Approximately 20% of the maternal cardiac output perfuses the uterine vessels (placental and nonplacental). The kidneys also demonstrate substantial increase of the regional blood flow as much as 30 to 80

percent and at the same time a 50 percent increase in glomerular filtration rate is noted. The regional blood flow in the extremities also increases and more so in the hands than the legs. As it was mentioned previously, there is a significant dilatation in the skin vessels which leads to an increase in the
regional blood flow. These changes in the skin vessels may cause warm skin, clammy hands, vascular spiders, and palm erythema. The liver circulation is not affected very much and the same is true for the brain blood flow which is autoregulated. The blood flow to the breast is increased during pregnancy to prepare the breast for lactation. The effect of pregnancy on coronary blood flow is still unknown. It is safe, however, to speculate that an increase may happen since augmentation of cardiac function is present during pregnancy.
Cardiocirculatory Changes During Labor and Delivery During labor significant hemodynamic changes take place. These changes can in part be explained by the effect of the uterine contractions, which may cause a significant increase of 300 to 500 ml in central blood volume, and in part by the effect of pain and anxiety on the cardiovascular system. It is important to note here that in the lateral position, cardiac output between contractions is higher than in the supine position and the increase during contractions is smaller. The effect of uterine contractions during labor on the heart rate is variable. Some investigators have reported an increase in the heart rate and others have reported a decline in the heart rate. The differences may have to do with different position of the patient during the labor process and certainly different hemodynamic changes that can lead to the variability in the heart rate.

Fetal-placental unit

Levels of progesterone and estrogen rise continually throughout pregnancy, suppressing the hypothalamic axis and subsequently the menstrual cycle. The progesterone is first produced by the corpus luteum and then by the placenta in the second trimester. Women also experience increased human chorionic gonadotropin (β-hCG), which is produced by the placenta.

Pancreatic Insulin

The placenta also produces human placental lactogen (hPL), which stimulates maternal lipolysis and fatty acid metabolism. As a result, this conserves blood glucose for use by the fetus. It can also decrease maternal tissue sensitivity to insulin, resulting in gestational diabetes.

Pituitary gland

The pituitary gland grows by about onethird as a result of hyperplasia of the lactrotrophs in response to the high plasma estrogen. Prolactin, which is produced by the lactrotrophs increases progressively throughout pregnancy. Prolactin mediates a change in the structure of the breast mammary glands from ductal to lobular-alveolar

and stimulates milk production.

Parathyroid

Fetal skeletal formation and then later lactation challenges the maternal body to maintain their calcium levels. The fetal skeleton requires approximately 30 grams of calcium by the end of pregnancy. The mother's body adapts by increasing parathyroid hormone, leading to an increase in calcium uptake within the gut as well as increased calcium reabsorption by the kidneys. Maternal total serum calcium decreases due to maternal hypoalbuminemia, but the ionized calcium levels are maintained.

Adrenal glands

Total cortisol increases to three times of non-pregnant levels by the third trimester. The increased estrogen in pregnancy leads to increase corticosteroid-binding globulin production and in response the adrenal gland produces more cortisol. The net effect is an increase of free cortisol. This contributes to insulin resistance of pregnancy and possibly striae. Despite the increase in cortisol, the pregnant mom does not exhibit Cushing syndrome or symptoms of high cortisol. One theory is that high progesterone levels act as an antagonist to the cortisol.

The adrenal gland also produces more aldosterone, leading to an eight-fold increase in aldosterone.[4] Women do not show signs of hyperaldosterone, such as hypokalemia, hypernatremia, or high blood pressure.

The adrenal gland also produces more androgens, such as testosterone, but this is buffered by estrogen's increase in sexhormone binding globulin (SHBG). SHBG binds avidly to testosterone and to a lesser degree DHEA.

Thyroid

The thyroid enlarges and may be more easily felt during the first trimester. The increase in kidney clearance during pregnancy causes more iodide to be excreted and causes relative iodine deficiency and as a result an increase in thyroid size. Estrogen-stimulated increase in thyroid-binding globulin (TBG) leads to an increase in total thyroxine (T4), but free thyroxine (T4) and triiodothyronine (T3) remain normal.

Musculoskeletal

Immune tolerance

Neuromechanical adaptations to pregnancy refers to the change in gait, postural parameters, as well as sensory feedback, due to the numerous anatomical, physiological, and hormonal changes women experience during pregnancy. Such changes increase their risk for musculoskeletal disorders and fall injuries. Musculoskeletal disorders include lower-back pain, leg cramps, and hip pain. Pregnant women fall at a similar rate (27%) to women over age of 70 years (28%). Most of the falls (64%) occur during the second trimester. Additionally, twothirds of falls are associated with walking on slippery floors, rushing, or carrying an object. The root causes for these falls are not well known. However, some factors that may contribute to these injuries include deviations from normal posture, balance, and gait.

The body's posture changes as the pregnancy progresses. The pelvis tilts and the back arches to help keep balance. Poor posture occurs naturally from the stretching of the woman's abdominal muscles as the fetus grows. These muscles are less able to contract and keep the lower back in proper alignment. The pregnant woman has a different pattern of gait. The step lengthens as the pregnancy progresses, due to weight gain and changes in posture. On average, a woman's foot can grow by a half size or more during pregnancy. In addition, the increased body weight of pregnancy, fluid retention, and weight gain lowers thearches of the foot, further adding to the foot's length and width. The influences of increased hormones such as estrogen and relaxin initiate the remodeling of soft tissues, cartilage and ligaments. Certain skeletal joints such as the pubic symphysis and sacroiliac widen or have increased laxity.The addition of mass, particularly around the torso, naturally changes a pregnant mother's center of mass (COM). The change in COM requires pregnant mothers to adjust their bodies to maintain balance.

Breast size

A woman's breasts grow during pregnancy, usually 1 to 2 cup sizes and potentially several cup sizes. A woman who wore a C cup bra prior to her pregnancy may need to buy an F cup or larger bra while nursing. A woman's torso also grows and her bra Breast sizeband size may increase one or two sizes. An average of 80% of women wear the wrong bra size, and mothers who are preparing to nurse can benefit from a professional bra fitting from a lactation consultant. Once the baby is born up to about 50–73 hours after birth, the mother will experience her breasts filling with milk (sometimes referred to as “the milk coming in”). Once lactation begins, the woman's breasts swell significantly and can feel achy, lumpy and heavy (which is referred to as engorgement). Her breasts may increase in size again by an additional 1 or 2 cup sizes, but individual breast size may varydepending on how much the infant nurses from each breast. A regular pattern of nursing is generally established after 8–12 weeks, and a woman's breasts will usually reduce in size, but may remain about 1 cup size larger than prior to

her pregnancy. Changes in breast size during pregnancy may be related to the sex of the infant, as mothers of female infants have greater changes in breast size than mothers of male infants.

Many people and even medical professionals mistakenly think that breastfeeding causes the breasts to sag (referred to as ptosis). As a result,some new parents are reluctant to nurse their infants. In February 2009, Cheryl Cole told British Vogue that she hesitated to breastfeed because of the effect it might have on her breasts. "I want to breastfeed," she said, "but I've seen what it can do, so I may have to reconsider." In actuality, breastfeeding is not considered to be a major contributor to ptosis of the breasts.

Changes in renal anatomy and function

As a consequence of renal vasodilatation, renal plasma flow and glomerular filtration rate (GFR) both increase, compared to non-pregnant levels, by 40–65 and 50–85%, respectively. In addition, the increase in plasma volume causes decreased oncotic pressure in the glomeruli, with a subsequent rise in GFR. Vascular resistance decreases in both the renal afferent and efferent arterioles and therefore, despite the massive increase in renal plasma flow, glomerular hydrostatic pressure remains stable, avoiding the development of glomerular hypertension. As the GFR rises, both serum creatinine and urea concentrations decrease to mean values of about 44.2 μmol/l and 3.2 mmol/l, respectively.

The increased renal blood flow leads to an increase in renal size of 1–1.5 cm, reaching the maximal size by mid-pregnancy. The kidney, pelvis and calyceal systems dilate due to mechanical compressive forces on the ureters. Progesterone, which reduces ureteral tone, peristalsis and contraction pressure, mediates these anatomical changes. The increase in renal size is associated with an increase in renal vasculature, interstitial volume and urinary dead space. There is also dilation of the ureters, renal pelvis and calyces, leading to physiological hydronephrosis in over 80% of women. There is often a right-sided predominance of hydronephrosis due to the anatomical circumstances of the right ureter crossing the iliac and ovarian vessels at an angle before entering the pelvis. Urinary stasis in the dilated collecting system predisposes pregnant women with asymptomatic bacteriuria to pyelonephritis.

There are also alterations in the tubular handling of wastes and nutrients. As in the non-pregnant state, glucose is freely filtered in the glomerulus. During pregnancy, the reabsorption of glucose in the proximal and collecting tubule is less effective, with variable excretion. About 90% of pregnant women with normal blood glucose levels excrete 1–10 g of glucose per day. Due to the increases in both GFR and glomerular capillary permeability to albumin, the fractional excretion of protein may increase up to

300 mg/day and protein excretion also increases. In normal pregnancies the total protein concentration in urine does not increase above the upper normal limit. Uric acid excretion also increases due to increased GFR and/or decreased tubular reabsorption.

Body water metabolism

Arterial under-filling in pregnancy leads to the stimulation of arterial baroreceptors, activating the RAA and the sympathetic nervous systems. This results in a non-osmotic release of AVP from the hypothalamus. These changes lead to sodium and water retention in the kidneys and create a hypervolaemic, hypoosmolar state characteristic of pregnancy. Extracellular volume increases by 30– 50% and plasma volume by 30–40%. Maternal blood volume increases by 45% to approximately 1 200 to 1 600 ml above non-pregnant values. By the late third trimester the plasma volume increases by more than 50–60%, with a lower increase in red blood cell mass, and therefore plasma osmolality falls by 10

mosmol/kg. The increase in plasma volume plays a critical role in maintaining circulating blood volume, blood pressure and uteroplacental perfusion during pregnancy.

Activation of the RAA system leads to increased plasma levels of aldosterone and subsequent salt and water retention in the distal tubule and collecting duct. In addition to the increased renin production by the kidneys, ovaries and uteroplacental unit produce an inactive precursor protein of renin in early pregnancy. The placenta also produces oestrogens that stimulate the synthesis of angiotensinogen by the liver, resulting in proportionally increased levels of aldosterone compared to renin. Plasma levels of aldosterone correlate well with those of oestrogens and rise progressively during pregnancy. The increase in aldosterone is responsible for the increase in plasma volume during pregnancy. Progesterone, which is a potent aldosterone antagonist, allows natriuresis despite the sodium-retaining properties of aldosterone. The rise in GFR also increases distal sodium delivery, allowing excretion of excess sodium. Progesterone has antikaliuretic effects and therefore excretion of potassium is kept constant throughout pregnancy due to changes in tubular reabsorption, and total body potassium increases during pregnancy.

Hypothalamic AVP release increases early in pregnancy as a result of increased relaxin levels. AVP mediates an increase in water reabsorption via aquaporin 2 channels in the collecting duct. The threshold for hypothalamic secretion of AVP and the threshold for thirst is reset to a lower plasma osmolality level, creating the hypo-osmolar state characteristic of pregnancy. These changes are mediated by human chorionic gonadotropin (hCG) and relaxin.

In middle and late pregnancy there is a four-fold increase in vasopressinase, an

aminopeptidase produced by the placenta. These changes enhance the metabolic clearance of vasopressin and regulate the levels of active AVP. In conditions of increased placental production of vasopressinase, such as preeclampsia or twin pregnancies, a transient diabetes insipidus may develop. As a consequence of this volume expansion, the secretion of atrial natriuretic peptides increases by 40% in the third trimester, and rises further during the first week postpartum. The levels of natriuretic peptides are higher in pregnant women with chronic hypertension and pre-eclampsia.