كلية : كلية الطب العام

القسم او الفرع : النسائية والتوليد

المرحلة: الرابعة

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اسم المادة باللغة العربية :التوليد

اسم المادة باللغة الإنكليزية : Obstetrics

اسم المحاضرة الثامنة باللغة العربية: التغييرات الفسيولوجية في جسم المراة الحامل

اسم المحاضرة الثامنة باللغة الإنكليزية: MATERNAL PHYSIOLOGY

محتوى المحاضرة الثامنة

MATERNAL PHYSIOLOGY

The anatomical, physiological, and biochemical adaptations to pregnancy are profound. Many of these remarkable changes begin soon after fertilization and continue throughout gestation, and most occur in response to physiological stimuli provided by the fetus and placenta. Equally astounding is that the woman who was pregnant is returned almost completely to her pregnancy state after delivery and lactation.

Many of these physiological adaptations could be perceived as abnormal in the nonpregnant woman. For example, cardiovascular changes during pregnancy normally include substantive increases in blood volume and cardiac output, which may mimic thyrotoxicosis. On the other hand, these same adaptations may lead to ventricular failure if there is underlying heart disease. Thus, physiological adaptations of normal pregnancy can be misinterpreted as pathological but can also unmask or worsen preexisting disease.

During normal pregnancy, virtually every organ system undergoes anatomical and functional changes that can alter criteria for diagnosis and treatment of diseases. Thus, the understanding of these adaptations to pregnancy remains a major goal of obstetrics, and without such knowledge, it is almost impossible to understand the disease processes that can threaten women during pregnancy.

- In early pregnancy, the developing fetus, corpus luteum and placenta produce and release increasing quantities of hormones, growth factors and other substances into the maternal circulation. This triggers a cascade of events that transform the mother's cardiovascular, respiratory and renal systems.
- The first trimester of pregnancy is therefore a transition period between the pregnant and non-pregnant state, during which changes in all these systems take place to prepare the mother to support fetal growth. Most pregnant women report symptoms of pregnancy by the end of the sixth week after the last menstrual period.
- It is assumed that most physiological adaptations are completed during the first trimester, although studies examining early pregnancy physiological changes are limited.

Following implantation, the maternal adaptation to pregnancy can be categorized based on the following functions:

1.Increased availability of precursors for hormone production and fetal–placental metabolism.

2. Improved transport capacity.

3. Maternal–fetal exchange.

4. Removal of additional waste products.

Increased availability of metabolic substrates and hormones is achieved by increases in dietary intake, as well as endocrine changes that increase the availability of substrates like glucose.

Transport capacity is enhanced by increases in cardiac output, facilitating both the transport of substrates to the placenta, and fetal waste products to maternal organs for disposal.

The placenta regulates maternal–fetal exchange by 10–12 weeks gestation, but transfer occurs through other mechanisms before this. Disposal of waste products (heat, carbon dioxide and metabolic byproducts) occurs through peripheral vasodilatation and by increases in ventilation and renal filtration.

Volume homeostasis

Maternal blood volume expands during pregnancy to allow adequate perfusion of vital organs, including the placenta and fetus, and to anticipate blood loss associated with delivery.

The rapid expansion of blood volume begins at 6–8 weeks gestation and plateaus at 32–34 weeks gestation. While there is some increase in intracellular water, the most marked expansion occurs in extracellular fluid volume, especially circulating plasma volume.

This expanded extracellular fluid volume accounts for between 8 and 10 kg of the average maternal weight gain during pregnancy. Overall, total body water increases from 6.5 to 8.5 L by the end of pregnancy.

Changes in blood volume are key to other physiological adaptations; predominantly increases in cardiac output and in renal blood flow. The interpretation of haematological indices in normal pregnancy is also affected, for example the larger increase of plasma volume relative to erythrocyte volume results in haemodilution and a physiologic anaemia.

The mechanisms responsible for fluid retention and changes in blood volume are unclear:

In pregnancy, changes in osmoregulation and the renin-angiotensin system result in active sodium reabsorption in renal tubules and water retention.

There is a net retention of sodium during normal pregnancy (3–4 mmol per day) and concentrations of anti-natriuretic hormones increase, opposing natriuretic factors, such as atrial natriuretic peptide and progesterone, also increase during pregnancy.

As maternal plasma sodium concentration decreases slightly during pregnancy it is possible that other factors, such as changes in intracellular metabolism, may contribute to fluid retention.

Plasma osmolality decreases by about 10 mOsmol/kg.

There is also a decrease in the thirst threshold so that pregnant women feel the urge to drink at a lower level of plasma osmolality than non-pregnant women.

Plasma osmotic pressure decreases during pregnancy, while oncotic pressure (colloid osmotic pressure) is reduced. Plasma oncotic pressure is mainly determined by albumin concentration, and this decreases by about 20 per cent during normal pregnancy.

As plasma oncotic pressure partly determines the degree to which fluid passes into and out of capillaries, its decrease is one of the factors responsible for the increase in glomerular filtration rate (GFR) during pregnancy and probably contributes to the development of peripheral oedema, a feature of normal pregnancy.

- Factors contributing to fluid retention

1. Sodium retention.
2. Resetting of osmostat.
3. ↓ Thirst threshold.
4. ↓ Plasma oncotic pressure.

- Consequences of fluid retention

1. ↓ Haemoglobin concentration.
2. ↓ Haematocrit.
3. ↓ Serum albumin concentration.
4. ↑ Stroke volume.
5. ↑ Renal blood flow.

Haematology

The circulating red cell mass increases by 20–30% during pregnancy, with rises in both cell number and size. It rises more in women with multiple pregnancies, and substantially more with iron supplementation (∼29% compared with 17%). Serum iron concentration falls, the absorption of iron from the gut rises and iron-binding capacity rises in a normal pregnancy. Plasma folate concentration halves by term, because of greater renal clearance, although red cell folate concentrations fall less. Even now, only ∼20% of fertile women have adequate iron reserves for a pregnancy and ∼40% have virtually no iron stores. Even relatively mild maternal anaemia is associated with increased placental:birthweight ratios and decreased birth weight. However, inappropriate supplementation can itself be associated with pregnancy problems. Erythropoietin rises in pregnancy, more if iron supplementation is not taken (55% compared with 25%) but the changes in red cell mass antedate this; human placental lactogen may stimulate haematopoiesis.

Maternal haemoglobin levels are decreased because of:

. The discrepancy between the 1000 to 1500 mL increases in plasma volume and the increase in erythrocyte mass, which is around 280 mL.

. Transfer of iron stores to the fetus contributes further to this physiological anaemia.

The mean haemoglobin concentration falls from 13.3 g/dL in the non-pregnant state to 10.9 g/dL at the 36th week of normal pregnancy. A normal pregnancy haematocrit is approximately 32–34 per cent, also lower than non-pregnant values.

These physiological changes may be mistaken for the development of pathological anaemia, most commonly due to iron deficiency. Pregnant women require increased amounts of iron, and absorption of dietary iron from the gut is increased as a result.

Despite this adaptation, women who do not take supplementary iron during pregnancy show a reduction in iron in the bone marrow as well as a progressive reduction in mean red cell volume and serum ferritin levels. The latter are still lower at six months after delivery than in early pregnancy, suggesting that pregnancy without iron supplementation leads to depletion of iron stores.

Renal clearance of folic acid increases substantially during normal pregnancy and plasma folate concentrations fall. However, red cell folate concentrations do not decrease to the same extent.

Folate supplementation for haematinic purposes in women eating an adequate diet and carrying a single fetus is therefore not routinely indicated.

Finally, the maternal platelet count usually remains stable throughout pregnancy, although may be lower than in the non-pregnant state due to increased aggregation.

Increases in the platelet count have been reported in the first week postpartum and this may contribute to the increased risk of thromboembolic complications in this period.

Coagulation

Continuing low-grade coagulopathy is a feature of normal pregnancy. Several of the potent procoagulatory factors rise from at least the end of the first trimester. For example, Factors VII, VIII and X all rise and absolute plasma fibrinogen doubles, while antithrombin III, an inhibitor of coagulation, falls. The erythrocyte sedimentation rate rises early in pregnancy due to the increase in fibrinogen and other physiological changes. Protein C, which inactivates Factors V and VIII, is probably unchanged in pregnancy, but concentrations of Protein S, one of its co-factors, fall during the first two trimesters. An estimated 5–10% of the total circulating fibrinogen is consumed during placental separation, and thromboembolism is the main cause of maternal death in the UK. Plasma fibrinolytic activity is decreased during pregnancy and labour, but returns to non-pregnant values within an hour of delivery of the placenta, suggesting strongly that the control of fibrinolysis during pregnancy is significantly affected by placentally derived mediators.

Biochemistry

Plasma protein concentrations, particularly albumin, are decreased during normal pregnancy, which not only affects the plasma oncotic pressure (as already discussed), but also affects the peak plasma concentrations of drugs that are highly protein bound.

Serum creatinine, uric acid and urea concentrations are reduced during normal pregnancy, although the renal handling of uric acid changes in late gestation, resulting in increased re-absorption.

Alkaline phosphatase levels increase throughout pregnancy, due to production of placental alkaline phosphatase. In contrast, levels of alanine transaminase and aspartate transaminase have been shown to be lower in uncomplicated pregnancy when compared to non-pregnant levels.

The lactate dehydrogenase (LDH) concentration in serum either remains unaltered or increases a small amount during normal pregnancy.The observed rise in serum LDH 1 week after delivery might originate from the involuting uterus and from damaged erythrocytes involved in the haemostatic process in the placental bed.

Normal parameters

It is now known that the reference values of most routine laboratory parameters change during pregnancy and the puerperium.

Therefore, clinicians treating pregnant women must be aware of the physiological changes that occur during pregnancy to avoid misinterpretation of laboratory results, which could lead to erroneous diagnoses or incorrect treatment.

White blood cells do not show a dilutional decrease during normal pregnancy, unlike red cells. In contrast, the total white cell count increases up to values of 14 109/L in the third trimester. This is mainly because of increases in the numbers of polymorph nuclear leukocytes, observed as early as 3 weeks gestation and especially marked postpartum.

The respiratory system

Tidal volume rises by ~30% in early pregnancy to 40–50% above non-pregnant values by term, with a fall in expiratory reserve and residual volume. Neither FEV 1 nor peak expiratory flow rate are affected by pregnancy, even in women with asthma. The rise in tidal volume is largely driven by progesterone, which appears to decrease the threshold and increase the sensitivity of the medulla oblongata to carbon dioxide. Respiratory rate does not change.

Carbon dioxide production rises sharply during the third trimester, as fetal metabolism increases. The fall in maternal Pco2 allows more efficient placental transfer of carbon dioxide from the fetus.

There is an increase of ~16% in oxygen consumption by term, due to increasing maternal and fetal demands. Pulmonary blood flow, of course, rises in parallel with cardiac output and enhances gas transfer. Pregnancy places greater demands on the cardiovascular than the respiratory system. This is shown in the response to moderate exercise.

The cardiovascular system

There is a significant fall in total peripheral resistance by 6 weeks gestation to a nadir of ~40% by mid-gestation, resulting in a fall in afterload. This is 'perceived' as circulatory underfilling, which activates the renin-angiotensin aldosterone system and allows the necessary expansion of the plasma volume (PV). By the late third trimester, the PV has increased from its baseline by about 50% in a first pregnancy and 60% in a second or subsequent pregnancy. The bigger the expansion is, the bigger, on average, the birth weight of the baby. The total extracellular fluid volume rises by about 16% by term. The plasma osmolality falls by ~10 mOsm/kg as water is retained.

The heart rate rises by 10–15 b.p.m., so the cardiac output begins to rise. There is probably a fall in baroreflex sensitivity as pregnancy progresses and heart rate variability falls. Stroke volume rises a little later in the first trimester. These two factors push the cardiac output up by 35–40% in a first pregnancy, and ~50% in later pregnancies; it can rise by further in third stage in labour.

Measuring systemic arterial blood pressure in pregnancy is difficult, but there is now broad consensus that Korotkoff 5 should be used with auscultatory techniques. However measured, there is a small fall in systolic and a greater fall in diastolic blood pressure during the first half of pregnancy resulting in an increased pulse pressure. The blood pressure then rises steadily and, even in normotensive women, there is some late overshoot of non-pregnant values. Supine hypotension occurs in ~8% of women in late gestation.

Pregnancy does not alter the response of intramyometrial arteries to a variety of vasoconstrictors. Nitric oxide may modulate myogenic tone and flow mediated responses in the resistance vasculature of the uterine circulation in normal pregnancy.

The venous pressure in the lower circulation rises for both mechanical and hydrodynamic reasons. The pulmonary circulation is able to absorb high rates of flow without an increase in pressure; so pressure in the right ventricle and the pulmonary arteries and capillaries does not change. Pulmonary resistance falls in early pregnancy and does not change thereafter. There is progressive venodilatation and rises in venous distensibility and capacitance throughout a normal pregnancy, possibly because of increased local nitric oxide synthesis.

Gastrointestinal Tract

The physiological changes of pregnancy include effects on mucous membranes, pigmentation and glandular function.

Pregnancy gingivitis is the term used for inflammation and hyperplasia of the gingival mucosa occurring during gestation and from 30 to 75 per cent of pregnant women develop erythema, oedema, hyperplasia and increased bleeding of the gingival tissue.

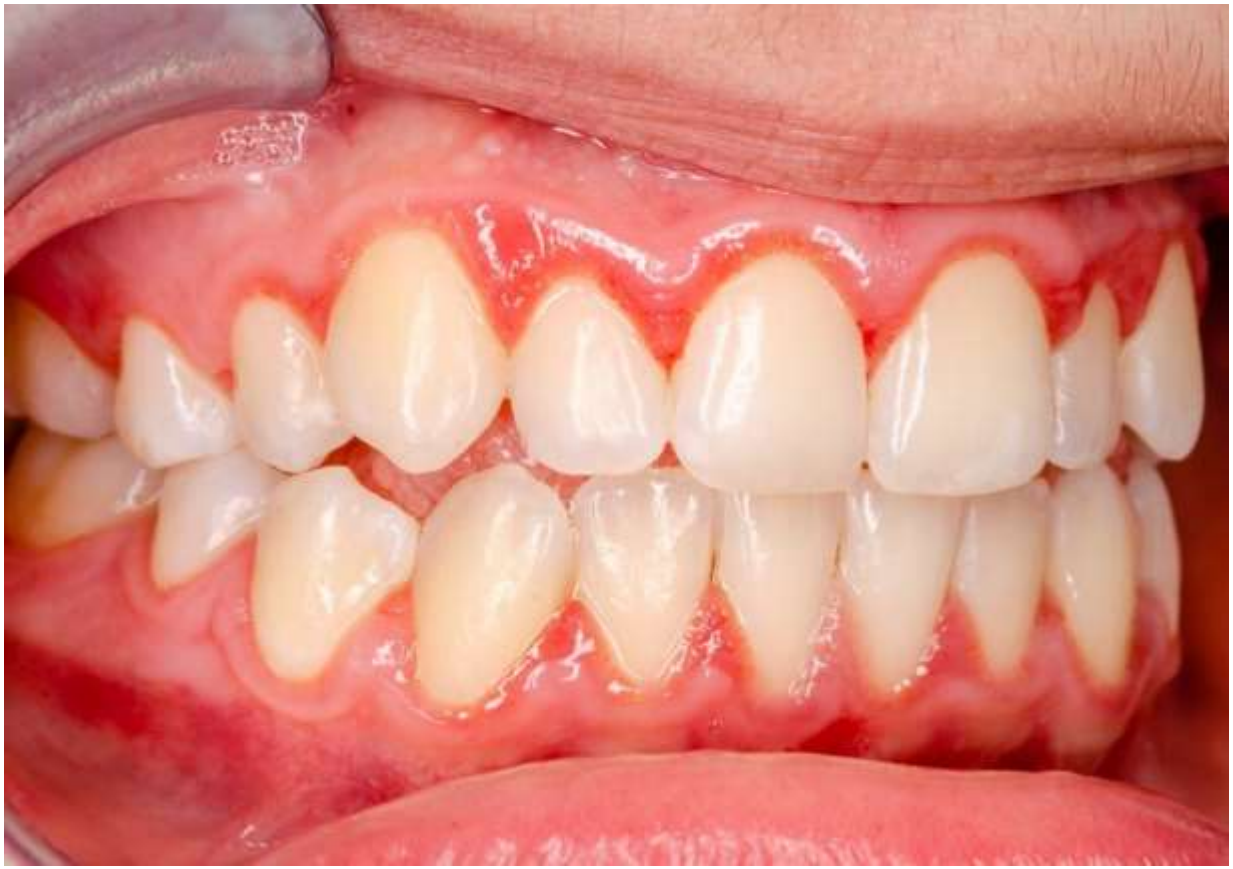
Elevated circulating oestrogen and progesterone levels are implicated in increasing vascular permeability and decreasing immune resistance, thereby increasing susceptibility to gingivitis.

The hormone levels of pregnancy also affect the response of the periodontal tissues to bacterial colonization, creating a more favourable environment for anaerobic infection.

The main salivary changes in pregnancy include variations in pH and composition, with a reduction of sodium concentration that leads to a decrease in pH and increased concentration of protein. Salivary oestrogen levels are increased, resulting in an increased proliferation and desquamation of the oral mucosa.

While teeth usually retain their structure, salivary changes late in pregnancy, as well as oestrogen enhanced changes in the mucosa, predispose to dental caries. Increased tooth mobility, especially of the upper incisors, has been detected in pregnant women, even those with normal periodontal tissues.

Pregnancy gingivitis



Gut:

As gestation advances, the uterus displaces the stomach and intestines upwards, which can hinder diagnosis of intra-abdominal surgical events as well as confound the routine abdominal examination.

Elevated progesterone levels reduce lower oesophageal sphincter tone and increase the placental production of gastrin, increasing gastric acidity.

These changes combine to increase the incidence of reflux oesophagitis and heartburn, which affect up to 80 per cent of pregnant women.

Mechanical factors, the enlarging uterus and progesterone levels all contribute to delayed gastric emptying and increased stomach volume.

Gastric motility decreases further during labour and emptying remains delayed during the puerperium. As a result of all of these changes, the pregnant woman is at increased risk of aspiration of gastric contents when sedated or anaesthetized after 16 weeks gestation.

Delayed gastric motility and prolonged gastrointestinal transit time may also lead to constipation and alter the bioavailability of medications.

Liver

The liver, normally palpated 2 cm below the right costal margin, may become more difficult to examine because of the expanding uterus within the abdominal cavity.

Physical findings such as telangiectasia and palmar erythema, otherwise suggestive of liver disease in non-pregnant women, appear in up to 60 per cent of normal pregnancies because of the hyperoestrogenic state of pregnancy, as the liver cannot easily metabolize the large quantity of placental oestrogen and progesterone.

Although hepatic protein production increases, serum albumin levels decline in pregnancy due to the increase in maternal plasma volume. In contrast, an increase in serum alkaline phosphatase secondary to fetal and placental production is observed in pregnancy and persists postpartum, rendering it unhelpful in diagnosing cholestasis during the third trimester.

Probably the most important hepatic changes in pregnancy are the increased production and plasma levels of fibrinogen and the clotting factors VII, VIII, X and XII.

Finally, increased cholesterol level is well described in pregnancy – as plasma cholesterol levels rise by around 50 per cent in the third trimester and triglycerides may rise to two or three times normal levels. Levels fall after delivery, returning to normal faster in lactating women.

Gallbladder

During normal pregnancy, the contractility of the gallbladder is reduced, leading to an increased residual volume. This may be because progesterone impairs gallbladder contraction. Impaired emptying leads to stasis, which associated with increased bile cholesterol saturation of pregnancy, contributes to the increased prevalence of cholesterol gallstones in multiparous women.

Intrahepatic cholestasis has been linked to high circulating levels of estrogen, which inhibit intraductal transport of bile acids. In addition, increased progesterone and genetic factors have been implicated in the pathogenesis.

The renal system

The kidneys increase in size in pregnancy mainly because renal parenchymal volume rises by about 70% with marked dilatation of the calyces, renal pelvis and ureters in most women. The effective renal plasma flow (RPF) is increased by at least 6 weeks gestation and rises to some 80% by mid-pregnancy falling thereafter to ~65% above non-pregnant values. This increase is proportionally greater than the increase in cardiac output, presumably reflecting specific vasodilatation, probably via the increased renal prostacyclin synthesis. The glomerular filtration rate (GFR) also increases, by ~45% by the 9th week, only rising thereafter by another 5–10%, but this is largely maintained to term.

These major increments do not, however, exhaust the renal reserve. This differential changes in ERPF and GFR in late pregnancy suggest a mechanism acting at the efferent arterioles, possibly angiotensin II.

The filtered load of metabolites therefore increases markedly, and reabsorptive mechanisms frequently do not keep up (e.g. glucose and aminoacids). These changes have profound effects on the concentration of certain plasma metabolites and electrolytes and 'Normal' laboratory reference ranges may thus be inappropriate in pregnancy. For example, plasma creatinine falls significantly by the 4th week of gestation and continues to fall to mid-pregnancy, to below 50 mmol/l.

Total body water rises by about 20% during pregnancy (~8.5 l) with a very sharp fall in plasma osmolality between weeks 4–6 after conception, possibly through the actions of hCG. As well as water present in the fetus, amniotic fluid, placenta and maternal tissues, there is also oedema fluid and increased hydration of the connective tissue ground substance with laxity and swelling of connective tissue.

Serum uric acid concentration falls by about a quarter in early pregnancy, with an increase in its fractional excretion secondary to a decrease in net tubular reabsorption. A similar pattern is seen in relation to urea, which is also partly reabsorbed in the nephron. Glucose excretion may rise 10-fold. If the urine of pregnant women is tested sufficiently often, glycosuria will be detected in 50% of them.

The excretion of most amino acids increases. Excretion of the water-soluble vitamins is also increased. The mechanism for all these is inadequate tubular reabsorption in the face of a 50% rise in GFR.

Urinary calcium excretion is also two to threefold higher in normal pregnancy than in the non-pregnant woman, even though tubular reabsorption is enhanced, presumably under the influence of the increased concentrations of 1,25-dihydroxyvitamin D. To counter this, intestinal absorption doubles by 24 weeks, after which it stabilizes. Renal bicarbonate reabsorption and hydrogen ion excretion appear to be unaltered during pregnancy.

Both total protein and albumin excretion rise during pregnancy, up to at least 36 weeks, due to the increased GFR and changes in both glomerular and tubular function. Thus in late pregnancy, an upper limit of normal of 200 mg total protein excretion/24 h collection is accepted. The assessment of proteinuria in pregnancy using dipsticks has been shown to give highly variable data.

Reproductive organs

Uterus:

Uterine blood flow increases 40-fold to approximately 700 mL/min at term, with 80 per cent of the blood distributed to the intervillous spaces of the placentae, and 20 per cent to the uterine myometrium.

Oestrogen mediates the adaptation of the uterine smooth muscle to pregnancy. High levels of maternal oestradiol and progesterone induce both hyperplasia and hypertrophy of the myometrium, increasing the weight of the uterus from 50–60 g prior to pregnancy to 1000 g by term. The growing size of the uterine contents is an important stimulus, with individual muscle fibers increasing in length by up to 15-fold.

As well as changes in the size and number of myometrial cells, specialized cellular connections also develop with increasing gestation. These intercellular gap junctions allow changes in membrane potential to spread rapidly from one cell to another, facilitating the spread of membrane depolarization, and subsequent myometrial contraction.

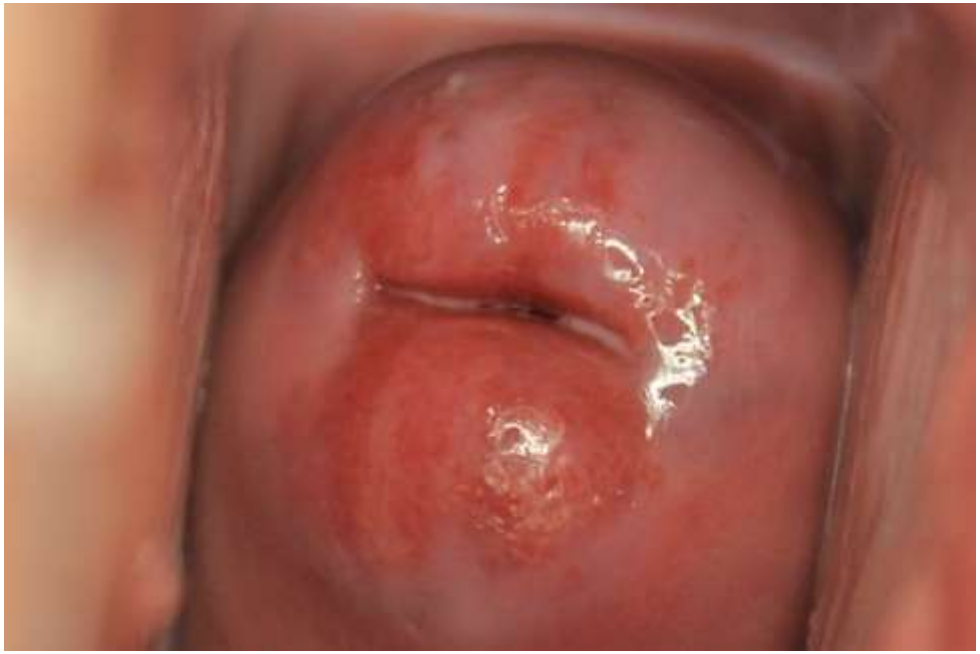
Steroid hormones also have an effect on signalling pathways. As these junctions mature, uterine contractions become more frequent. These are apparent initially as Braxton Hicks, painless contractions that are noticed in the second half of pregnancy. Subsequently, these allow the pacemaker activity of the uterine fundus to promote the coordinated, fundal-dominant contractions necessary for labour.

Cervix:

The cervix is described as looking bluer during pregnancy, which is due to its increased vascularity. It becomes swollen and softer during pregnancy under the influence of progesterone and oestradiol; the latter also stimulates growth of the columnar epithelium of the cervical canal. This becomes visible on the ectocervix and is called an ectropion, which is prone to contact bleeding.

In addition, the mucous glands of the cervix become distended and increase in complexity. Prostaglandins induce a remodelling of cervical collagen in late gestation, while collagenase released from leukocytes locally also aids in softening the cervix. Under the influence of oestrogens, the vaginal epithelium becomes more vascular during pregnancy, and there is increased desquamation resulting in increased vaginal discharge. This discharge has a more acid pH than non-pregnant vaginal secretions (4.5–5.0) and may protect against ascending infection.

Ectropion



Breasts and lactation:

The cyclical changes seen in breast tissue with the menstrual cycle are accentuated during pregnancy. Deposition of fat around glandular tissue occurs, and the number of glandular ducts is increased by oestrogen, while progesterone and human placental lactogen (hPL) increase the number of gland alveoli.

Prolactin is essential for the stimulation of milk secretion and during pregnancy prepares the alveoli for milk production. Although prolactin concentration increases throughout pregnancy, it does not then result in lactation since it is antagonized at an alveolar receptor level by oestrogen.

The rapid fall in oestrogen concentration over the first 48 hours after delivery removes this inhibition and allows lactation to begin. Towards the end of pregnancy, and in the early puerperium, the breasts produce colostrum, a thick yellow secretion rich in immunoglobulins.

Lactation is initiated by early suckling, which stimulates the anterior and posterior pituitary to release prolactin and oxytocin, respectively.

Endocrinology:

Pituitary gland:

The pituitary gland enlarges during normal pregnancy and concentrations of prolactin reach levels during pregnancy that are 15-fold higher than in the non-pregnant state. Oestrogen has a stimulatory role in this process.

The endocrinological mechanisms that regulate prolactin production in the non-pregnant state, such as sleep, which increases and dopamine agonists, which reduce prolactin concentration, remain effective during pregnancy. Therefore prolactin production by the anterior pituitary gland continues despite intrauterine production from cells within the decidua. Receptors for prolactin are also present on trophoblast cells and within the amniotic fluid.

Increased prolactin production is essential for lactation but also acts in the brain to reduce responses to stress.

Thyroid function:

Human chorionic gonadotrophin (hCG) has thyrotrophic activity owing to subunit homology with thyroid-stimulating hormone (TSH) and maternal TSH production is suppressed during the first trimester of pregnancy, when hCG levels are highest. Thyroid binding globulin increases in the first 2 weeks of pregnancy and reaches a plateau by 20 weeks. This leads to increased production of total T3 (tri-iodothyronine) and T4 (thyroxine). The increased GFR of pregnancy results in an increased renal loss of iodide, which is essential for thyroid hormone synthesis, so the thyroid compensates by increasing the proportion of iodide it takes up from the circulation. Where there is relative background iodide deficiency, these changes may result in enlargement of the thyroid gland during pregnancy.

The hypermetabolic state of normal pregnancy makes clinical assessment of thyroid function more difficult and therefore thyroid function often needs to be checked biochemically. However the physiological changes of pregnancy, including the 50 per cent plasma volume expansion, increased thyroid binding globulin production and relative iodine deficiency, mean that thyroid hormone reference ranges for non-pregnant women are not appropriate in pregnancy. Free T4 (fT4), free T3 and TSH should be analysed when assessing thyroid function in pregnancy, and total T3 and T4 not used. There is a fall in TSH and a rise in fT4 concentrations in the first trimester of normal pregnancy. This is followed by a fall in fT4 concentration with advancing gestation, with the most marked effect in the third trimester.

Uterus and placenta:

Many pregnancy-specific peptides are produced within the uterus, but not all have been shown to have definite endocrine roles. The best known is hCG, produced by trophoblast cells. The β-subunit is pregnancy specific and used as a sensitive pregnancy test, being detectable within the maternal circulation in small quantities within days of implantation.

Human chorionic gonadotrophin has a major role during early pregnancy in maintaining the function of the corpus luteum, which produces progesterone, but circulating hCG values fall off after 12 weeks, as the placental production of progesterone becomes dominant.

During normal pregnancy, hCG also suppresses the secretion of FSH and LH by the anterior pituitary gland, perhaps by interaction at the hypothalamic level.

Sex steroid hormones are produced in large quantities by the placenta and fetus. Concentrations of oestrogens and progesterone increase substantially from early pregnancy, and then plateau for the remainder of the pregnancy. Both oestrogen and progesterone have effects on the myometrium, where oestrogen encourages cellular hypertrophy while progesterone discourages contraction and, together with prolactin, on the tissues of the breast. They also have effects on many other tissues during pregnancy, such as the smooth muscle of the vascular tree and of the urinary and gastrointestinal tracts.

Skin

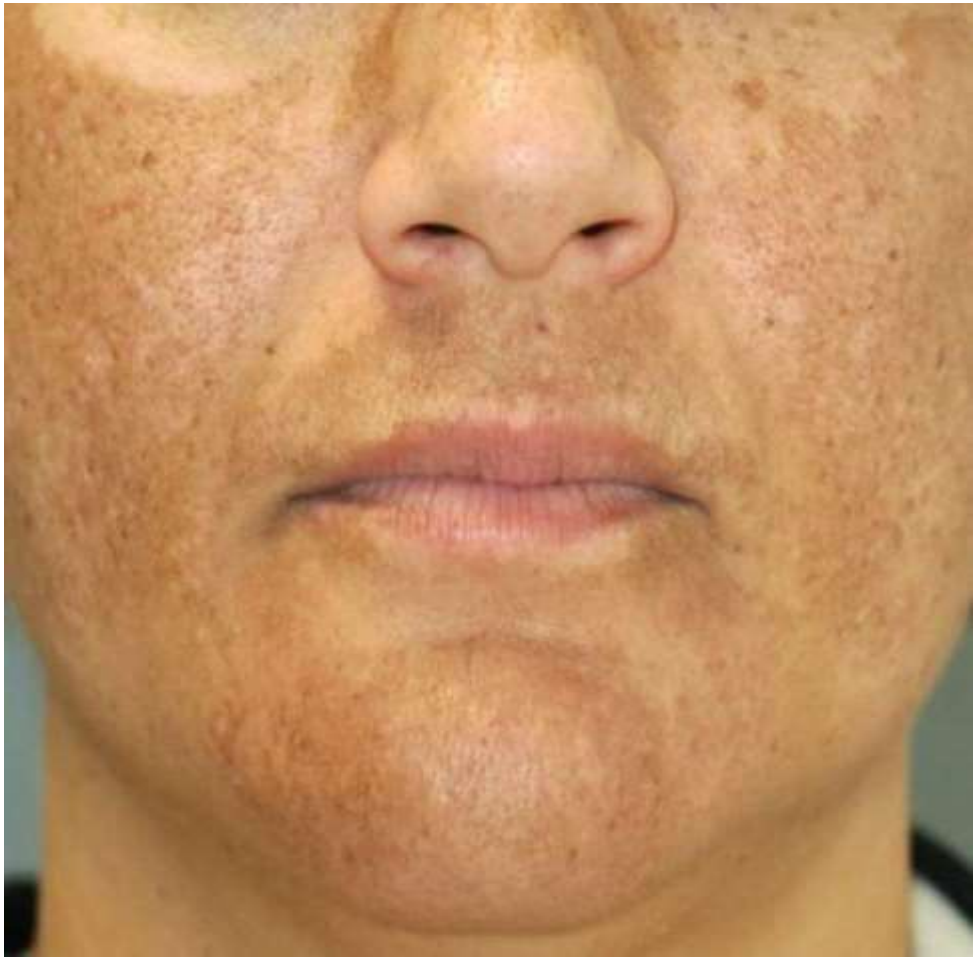
Hyperpigmentation can be localized or generalized and affects almost 90 per cent of pregnant women, being more obvious in women with darker skin. Pre-existing moles, freckles and recent scars also tend to become darker, as do areas of skin that are already normally pigmented – including the areolae, nipples, axillae and periumbilical skin.

The linea alba darkens to a brown line along the midline of the abdomen, which reaches the symphysis pubis, and is called the linea nigra. Growth and increase in the number of naevi have also been reported, but there is no evidence of an increased risk of malignant change.

All of these changes appear to regress after delivery, but may recur in subsequent pregnancies. Melasma, also called chloasma, is an acquired hypermelanosis characterized by symmetrical, irregular, macular brown-grey pigmentation of the face, reported in up to 75 per cent of pregnant women.

This hyperpigmentation results from the deposition of melanin in the epidermis, dermis, or both. The underlying cause is uncertain, but the hormonal influences of pregnancy are involved, as well as exposure to ultraviolet radiation, and the number of melanocytes in the skin is also increased. Pigmentation usually regresses after delivery but may persist in less than 10 per cent of those affected.

chloasma



Striae gravidarum (stretch marks) occur in most pregnant women, usually by the end of the second trimester, with a reported incidence of 90 per cent in Caucasians. Linear bands develop on the abdomen and sometimes on the thighs, arms, breasts, axillae and buttocks, then slowly progress into pale, skin-coloured, atrophic bands around the time of delivery.

Pruritus of the abdomen may be an accompanying feature.

The cause of striae is not fully understood but is probably related to destruction of elastic fibers. Several factors affect their development, including the degree of abdominal distension and maternal weight gain, genetic predisposition and hormonal changes (oestrogen, relaxin), which influence connective tissue formation. Striae persist postpartum but become less evident.

Sebaceous gland activity is increased during the second half of pregnancy with greasy skin, especially on the face, a common complaint.

Acne may also commence during pregnancy. Montgomery tubercles are small sebaceous glands on the areolae of the breasts that enlarge and hypertrophy during early pregnancy, and they present as multiple elevated brown papules, being one of the first signs of pregnancy.

Hirsuitism, defined as excessive growth of body hair, is seen in many pregnant women, especially those with dark or abundant hair. Women often notice thickening of scalp hair during pregnancy and a prolonged anagen phase has been demonstrated.

However, one to four months after delivery, a large proportion of hair enters the telogen phase, resulting in increased hair shedding known as telogen effluvium. This shedding may persist for several months postpartum and is most likely precipitated by the sudden hormonal changes at delivery as well as the stress of labour.

THANK YOU