D. 産科疾患の診断・治療・管理

Diagnosis, Therapy and Management of Obstetrics Disease

1. 妊娠の生理

Maternal Physiology

妊娠によって母体には著しい生理的変化が起こる．その多くは妊娠の終了と供に妊娠前の状態に戻る．これらの現象を正しく知ることは妊婦管理を行ううえで，極めて重要なことである．正常妊娠に伴う生理的変化について概説する．

1. 物質代謝

1）体重増加

妊娠期間中の体重増加の大部分は，子宮とその内容物，乳房，循環血液量と細胞外液の増加に起因する．そのほかに，細胞内液の増加と脂質，蛋白質の蓄積が挙げられる．妊娠中の体重増加は約20％である．

2）水代謝

正常な生理的変化として水分貯留が生じる．原因の一つに膠質浸透圧の低下が挙げられる．特に下肢では下大静脈の部分的な圧迫のため静脈圧が上昇し，浮腫を生じやすい．

3）蛋白質

妊娠末期には胎児と胎盤に約500g，ほかに，500g が子宮，乳房，ヘモグロビン，血漿蛋白として母体の血中に蓄積される．窒素平衡は妊娠週数に伴って増加する．

4）炭水化物

正常妊娠では軽度の空腹時の血糖低下と食後の血糖上昇および高インスリン血症が認められるが，これはグルコースを取り込みやすくする反応であろうと考えられている．また，同時にグルカゴン濃度の抑制も認められる[1]．胎児へのグルコース供給を円滑にするため末梢のインスリン抵抗性は上昇し，妊娠後期にはインスリン活性は非妊時の50～70％に低下する[2]．このインスリン抵抗性の原因として，プロゲステロン，エストロゲンが推定されている．また妊娠進行に伴い増加する胎盤ラクトーゲンは成長ホルモンとしての作用を持ち，血中の遊離脂肪酸を増加させる[3]．その結果，インスリン抵抗性が上昇する．空腹時の血中グルコース，アミノ酸濃度は低下し，遊離脂肪酸，トリグリセライド，コレステロール濃度は上昇し，母体のエネルギー源はグルコースから脂質へとシフトする．

5）脂質

血中脂質濃度は上昇する．妊娠中期から脂質の蓄積が行われるが，妊娠後期には胎児の栄養要求増加に従い母体の蓄積量は減少する．

6）電解質

妊娠期間中におよそ1,000mEq の Na と300mEq の K が蓄積される[4]．糸球体濾過率は上昇するが，尿細管での再吸収も増加するため，これらの電解質の排泄率は変化しない[5][6]．血中濃度は母体血液量の増加に伴いわずかに低下するが，非妊時正常域に極めて近い値を保つ[7]．血清 Ca 濃度は低下する．血中アルブミン濃度の低下に伴い結合型アルブミンが低下するが，血清イオン化 Ca 濃度は変化しない[8]．胎児骨格の発育に多量の Ca を必要とするため十分な摂取が求められる．血清 Mg 濃度も低下する．

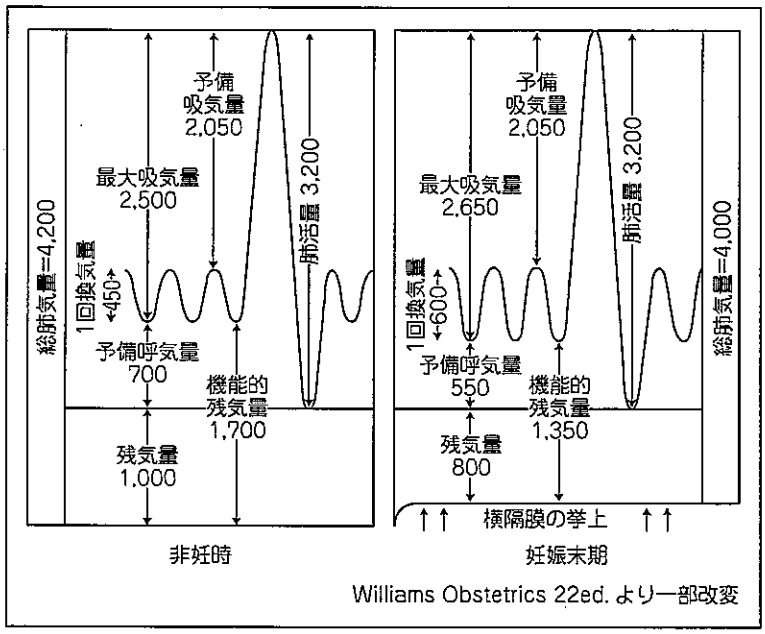
が，下肢では増大した子宮が骨盤内静脈や下大静脈を圧迫するため上昇する．静脈圧上昇や静脈うっ血のため下肢の浮腫，下肢や外陰部の静脈瘤，痔核を生ずることがある．妊娠後期では，仰臥位になると増大した子宮の圧迫により下半身からの静脈灌流が阻害され心拍出量が減少する．その結果動脈血圧は低下する．これは仰臥位低血圧症候群 Supine Hypotension Syndrome と呼ばれる[15]．さらに仰臥位が続けば動脈も圧迫されるため子宮動脈圧は上肢の動脈圧よりも低くなる．腰椎麻酔等で全身の血圧が下がると，子宮動脈圧は上肢の動脈圧よりもさらに低下する．

3. 呼吸器系

妊娠子宮の増大で横隔膜は挙上され，胸郭は横に拡大する．そのため，機能的残気量，残気量は減少する(図 D-1-3)．呼吸数はほとんど変化しないが，妊娠が進行するにつれ，一回換気量，分時換気量は増加する．一回換気量の増加，循環ヘモグロビン量の増加，心拍出量の増加で，妊娠によって増大した必要酸素量に見合う酸素を摂取する．妊婦では呼吸が増加し，血中 Pco2濃度は低下するが，これはプロゲステロンが呼吸中枢の Pco2反応閾値に作用する結果と考えられている．

4. 泌尿器系

腎臓は妊娠中わずかに拡大する．糸球体濾過率，腎血漿流量は妊娠初期から増加し，妊娠中期の初めには非妊娠時の50％まで増加する[17]．糸球体濾過率は妊娠末期まで高値を保つが，腎血漿流量は妊娠後期に減少する．妊娠末期には体位により腎機能検査値が変化する．正常妊婦の血清クレアチニン，尿素窒素の値は非妊娠時より低値を示す．クレアチニンクリアランスは30％増加する[18]．妊娠中の腎の特徴として，各種の栄養素の尿中排泄が挙げられる．アミノ酸や水溶性ビタミンは非妊娠時と比較し多量に排泄される[19][20]．



(図 D-1-3)

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〈道方香織*，池ノ上克*〉

*Kaori MICHIKATA, *Tsuyomu IKENOUE
**Department of Obstetrics Gynecology, University of Miyazaki, Miyazaki*
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Pulmonary Disease and Pregnancy

• Author: Tarun Madappa, MD, MPH; Chief Editor: Zab Mosenifar, MD more...

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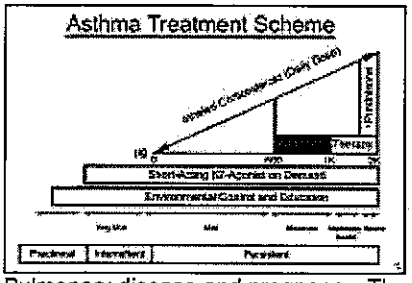
Alterations in Pulmonary Physiology During Pregnancy

Pregnancy induces profound changes in the mother, resulting in significant alterations in normal physiology. The anatomical and functional changes affect the respiratory and cardiovascular systems. Management of respiratory diseases in pregnancy requires an understanding of these changes for interpretation of clinical and laboratory manifestations of disease states.

An image depicting asthma consensus guidelines to manage chronic asthma can be seen below.



Pulmonary disease and pregnancy. The graph depicts asthma consensus guidelines to manage chronic asthma. These guidelin are also generally used to treat pregnant patients with asthma.

Respiratory physiology

Anatomical changes

Hormonal changes in pregnancy affect the upper respiratory tract and airway mucosa, producing hyperemia, mucosal edema, hypersecretion, and increased mucosal friability. Estrogen is probably responsible for producing tissue edema, capillary congestion, and hyperplasia of mucous glands.

The enlarging uterus and the hormonal effects produce anatomical changes to the thoracic cage. As the uterus expands, the diaphragm is displaced cephalad by as much as 4 cm; the anteroposterior and transverse diameter of the thorax increases, which enlarges chest wall circumference. Diaphragm function remains normal, and diaphragmatic excursion is not reduced.

Pulmonary function

Anatomical changes to the thorax produce a progressive decrease in functional residual capacity, which is reduced 10-20% by term. The residual volume can decrease slightly during pregnancy, but this finding is not consistent; decreased expiratory reserve volume definitely changes. The increased circumference of the thoracic cage allows the vital capacity to remain unchanged, and the total lung capacity decreases only minimally by term. Hormonal changes do not significantly affect airway function. Pregnancy does not appear to change lung compliance, but che wall and total respiratory compliance are reduced at term.

Ventilation

The minute ventilation increases significantly, beginning in the first trimester and reaching 20-40% above baseline a term. Alveolar ventilation increases by 50-70%. The increase in ventilation occurs because of increased metabolic carbon dioxide production and because of increased respiratory drive due to the high serum progesterone level. Th tidal volume increases by 30-35%. The respiratory rate remains relatively constant or increases slightly.

Arterial blood gases

Physiological hyperventilation results in respiratory alkalosis with compensatory renal excretion of bicarbonate. The arterial carbon dioxide pressure reaches a plasma level of 28-32 mm Hg, and bicarbonate is decreased to 18-21 mmol/L, maintaining an arterial pH in the range of 7.40-7.47. Mild hypoxemia might occur when the patient is in the

supine position. Oxygen consumption increases at the beginning of the first trimester and increases by 20-33% by term because of fetal demands and increased maternal metabolic processes.

In active labor, hyperventilation increases and tachypnea caused by pain and anxiety might result in marked hypocapnia and respiratory alkalosis, adversely affecting fetal oxygenation by reducing uterine blood flow. In some patients, severe pain and anxiety can lead to rapid shallow breathing with alveolar hypoventilation, atelectasis, and mild hypoxemia.

Alterations in Cardiac Physiology During Pregnancy

Hemodynamic changes

Cardiovascular changes begin in the first trimester of pregnancy and continue into the postpartum period. Maternal blood volume increases progressively, peaking at a value of approximately 40% above baseline by the third trimester. Plasma volume increases by 45-50%, and red cell mass increases by 20-30%, resulting in anemia of pregnancy.

The increased blood volume is associated with elevated cardiac output, which increases by 30-50% above baseline levels by 25 weeks. The heart rate increases and reaches a maximal value of 10-30% above baseline values by 32 weeks.

Systemic blood pressure decreases slightly during pregnancy, with the diastolic pressure falling approximately 10-20% and reaching a nadir at 28 weeks. Plasma colloid oncotic pressure decreases because of the dilution of plasm proteins; the critical pulmonary capillary pressure at which pulmonary edema forms also decreases. Systemic vascular resistance and pulmonary vascular resistance decrease by 20-30%.

Structural and functional changes in the heart

The left atrial size increases, correlating with the change in blood volume; left ventricular end-diastolic dimension increases; and left ventricular end-systolic dimension might decrease somewhat as a result of changes in cardiac contractility. The left ventricular wall thickness increases by 28%, and the left ventricular mass increases by 52%.

Supine hypotension syndrome

Near term, the enlarged uterus can compress surrounding vascular structures, resulting in a decrease in venous return and stroke volume and a decrease in cardiac output. Placing the patient in the left lateral position can reduce these effects.

Labor and puerperium

The hemodynamic changes are most striking during labor and immediately postpartum. Cardiac output normally increases during labor by 10-15% above third trimester levels. Uterine contractions result in increased preload due t autotransfusion of 300-500 mL of blood immediately postpartum. A further increase in blood volume occurs due to the contracted uterus.

Fetal oxygenation

Oxygen delivered to the placenta and fetus is dependent on maternal arterial oxygen content and uterine blood flow Any decrease in maternal cardiac output can adversely affect fetal oxygenation. Maternal hypotension and endogenous catecholamines may constrict the uterine artery, decreasing blood flow to the fetus. This can also be caused by maternal alkalosis.

The umbilical vein PaO_2 rarely exceeds 40 mm Hg, but the fetal oxygen content is relatively high. This is largely due to the marked left shift of the oxygen dissociation curve of fetal hemoglobin, which is 80-90% saturated at a PaO_2 o 30-35 mm Hg. The oxygenated umbilical vein blood mixes with deoxygenated blood in the fetal inferior vena cava, decreasing fetal arterial PaO_2 to 20-25 mm Hg. The fetus has a high hemoglobin concentration (150 g/L) and an increased systemic cardiac output, and both the left ventricle and the right ventricle (via ductus arteriosus) supply th systemic circulation. Small changes in maternal PaO_2 can cause large changes in fetal oxygen saturation because the fetus is operating on the steep portion of the oxygen dissociation curve.