# Normal Physiology of Pregnancy

Topic- based Uworld Questions

Block 1, 2, 7, 8





















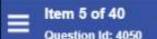


A 36-year-old woman, gravida 2 para 1, at 32 weeks gestation comes to the office with dull, low back pain radiating bilaterally to the buttocks and posterior thighs. The pain is minimal in the morning but increases with activity and at the end of the day. She also has ankle edema and numbness in her feet at the end of the day. The patient has urinary frequency and nocturia but no hematuria or dysuria. She did not have back pain in her first pregnancy. During that pregnancy, the patient received epidural anesthesia during labor and had a postlumbar puncture headache after removal that was treated with an epidural blood patch. Temperature is 36.7 C (98 F), blood pressure is 120/80 mm Hg, and pulse is 90/min. She has gained 20 kg (44.1 lb) during this pregnancy. On physical examination, the patient ambulates with a wide, waddling gait. Walking forward or backward causes no change in the pain level. There is no spinal or paravertebral tenderness. Deep tendon reflexes are 2+. The abdomen is soft, and the cervix is long, closed, and posterior. Which of the following is the most appropriate next step in management of this patient?

) A	Corticosteroid injection
) B.	MRI of the spine
) C.	Reassurance and conservative management
) D.	Strict bed rest
) E	Trial of nonsteroidal anti-inflammatory agent

Submit

















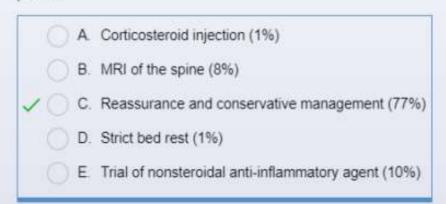








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C

77%
Answered correctly





Explanation























Low back pain during pregnancy		
Etiology	Enlarged uterus → exaggerated lordosis     Joint/ligament laxity from ↑ progesterone/relaxin     Weak abdominal muscles → decreased lumbar support	
Risk factors	Excessive weight gain     Chronic back pain     Back pain in prior pregnancy     Multiparity	
Imaging	Not indicated	
Management	Behavioral modifications     Heating pads     Analgesics	

Low back pain is common in pregnancy, especially in the third trimester. Contributing factors include exaggerated lordosis and altered posture due to the enlarging uterus, weakness of the abdominal muscles, and increased joint/ligamentous laxity. Additional risk factors include multiparity, back pain with prior pregnancy, and excessive weight gain. A history of neuraxial anesthesia does not increase the risk of long-term back pain.

Back pain during pregnancy typically manifests as **mechanical back pain** (ie, achy pain that is worse with activity and relieved with rest).

However, pregnant patients should be assessed for nonmechanical causes of pain (eg, preterm labor, pyelonephritis). Worrisome features include fever, neurologic deficits (eg, bowel or bladder incontinence), and recent epidural anesthesia. Constant, nonpositional, nocturnal pain suggests possible malignancy or epidural abscess; however, in pregnant women, nocturnal position-dependent pain is common and usually benign.

Patients with no concerning findings, such as this one, can be given **reassurance** that the back pain is normal and will resolve postpartum. **Conservative management** includes behavioral modifications (eg, wearing supportive shoes, using a firm mattress) and exercise; heating pads and massage are also beneficial.





















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(Choice A) An epidural corticosteroid injection can provide short-term relief for chronic radicular pain (ie, unilateral pain radiating below the knee along the sciatic nerve) related to a herniated disk. However, disk herniation with lumbosacral radiculopathy is uncommon in pregnancy. Epidural injections are not recommended for mechanical back pain, as in this patient.

(Choice B) MRI is the preferred imaging modality for pregnant women with back pain but is usually needed only when there are associated neurologic deficits or features of infection (eg, fever) or malignancy (eg, unexpected weight loss, history of malignancy). Minor pregnancy-related symptoms (eg, urinary frequency, edema) are common and do not require imaging.

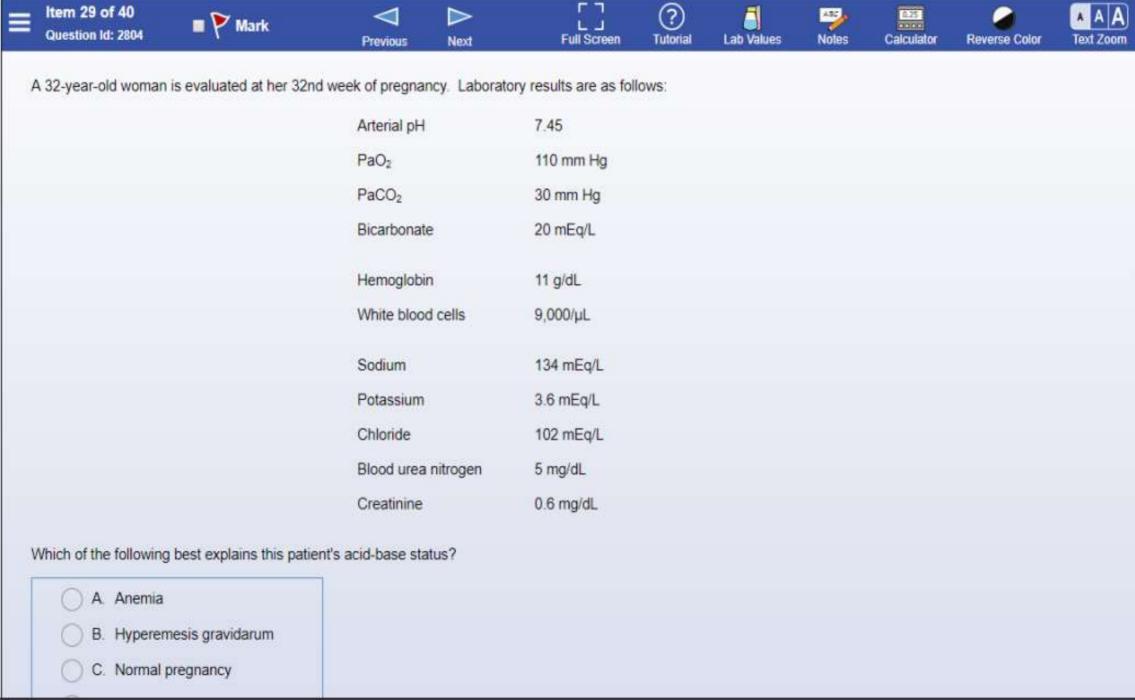
(Choice D) Strict bed rest is rarely recommended in pregnancy due to the increased risk of thromboembolism and decreased bone mineral density. In addition, prolonged bed rest can lead to physical deconditioning and exacerbate back pain.

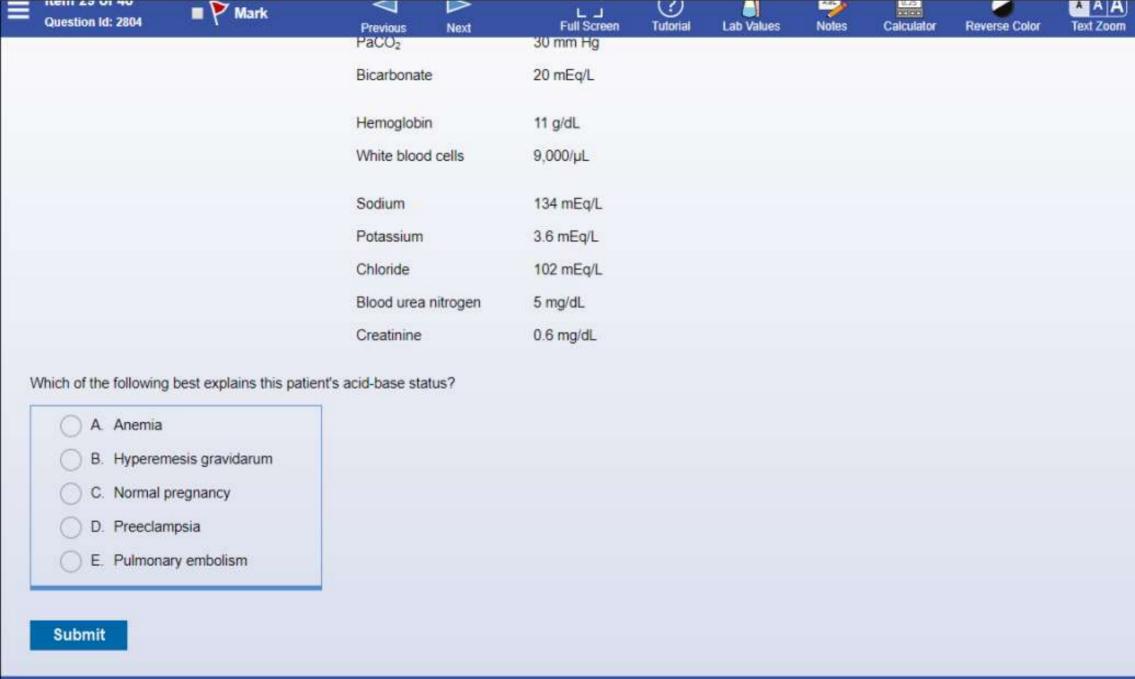
(Choice E) Acetaminophen is generally considered safe during pregnancy, although its effectiveness in low back pain is limited. However, nonsteroidal anti-inflammatory drugs are avoided in the first and third trimesters. Opioids are also generally avoided in pregnancy.

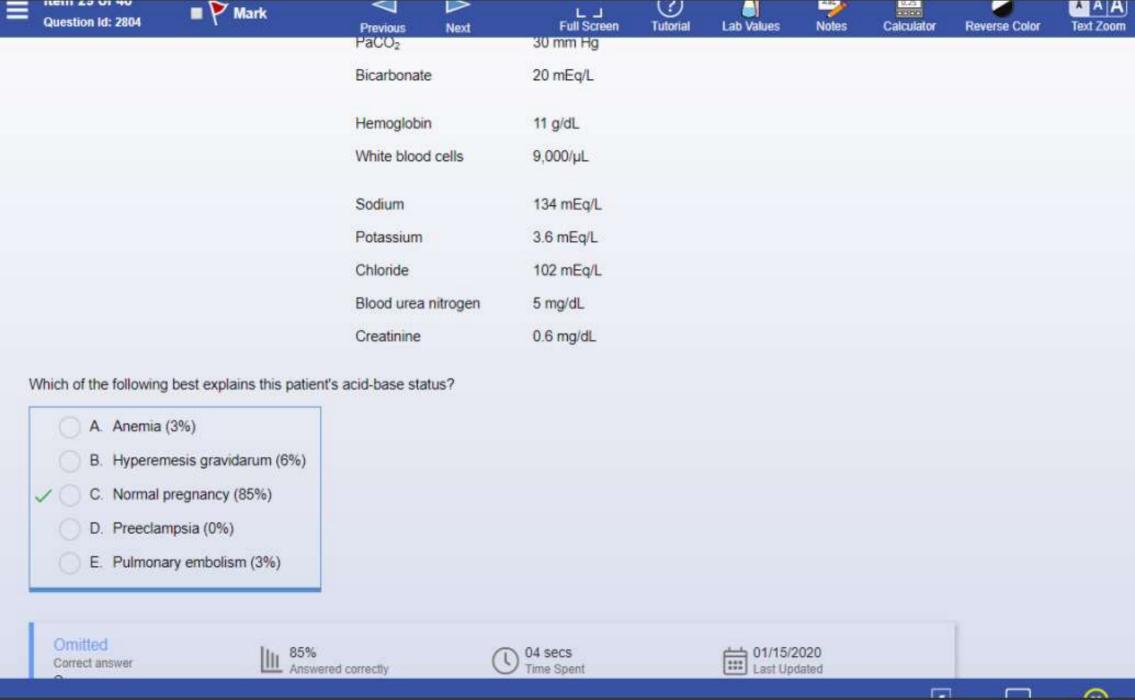
### Educational objective:

Low back pain is common in the third trimester of pregnancy due to postural changes, weakened abdominal muscles, and joint/ligament laxity. Conservative management includes behavioral modifications, exercise, heating pads, and massage. Pregnancy-related back pain usually resolves after delivery.









Physiologic changes of pregnancy		
Pulmonary	↑ central respiratory drive (hyperventilation)     ↓ PaCO₂ (respiratory alkalosis), ↑ PaO₂	
Renal	↑ renal blood flow & urine output     ↑ GFR, ↓ BUN & serum creatinine     ↑ HCO <sub>3</sub> <sup>-</sup> excretion (metabolic compensation)     ↓ serum Na <sup>+</sup> concentration (↑ ADH secretion)	
Hematologic	† prothrombotic coagulation factors     ↓ hemoglobin concentration (dilutional anemia)	

ADH = antidiuretic hormone; BUN = blood urea nitrogen; GFR = glomerular filtration rate; RBC = red blood cell.

Pregnancy causes many normal physiological changes (Table). Progesterone rises significantly during the first trimester and changes the homeostatic set points in the medullary respiratory centers of the brain. Progesterone also directly stimulates the respiratory centers to increase ventilation. The medulla is thought to become more sensitive to changes in PaCO<sub>2</sub> and responds with an exaggerated respiratory effort.

The normal hyperventilation of pregnancy is characterized by increased tidal volume, increased minute ventilation, and chronic respiratory alkalosis. The increased minute ventilation increases the PaO<sub>2</sub> (usually to 100-110 mm Hg) to meet the metabolic demands of pregnancy. The respiratory alkalosis usually lowers the PaCO<sub>2</sub> to 27-32 mm Hg. As the progesterone concentration increases during the later stages of pregnancy, there is increased pH to 7.40-7.45 with some metabolic compensation with decreased serum HCO<sub>3</sub>, as seen in this patient.

(Choice A) Anemia in pregnancy is defined by the Centers for Disease Control and Prevention as hemoglobin <11 g/dL in the 1st and 3rd trimesters and <10.5 g/dL in the second. Severe anemia can lead to poor tissue perfusion with lactic acidosis and an anion gap metabolic

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(Choice A) Anemia in pregnancy is defined by the Centers for Disease Control and Prevention as hemoglobin <11 g/dL in the 1st and 3rd trimesters and <10.5 g/dL in the second. Severe anemia can lead to poor tissue perfusion with lactic acidosis and an anion gap metabolic acidosis. This patient's hemoglobin of 11 g/dL and absence of an anion gap make this less likely.

(Choice B) Hyperemesis gravidarum usually occurs early in pregnancy (not in the 3rd trimester) and presents with significant vomiting leading to volume depletion and a hypochloremic metabolic alkalosis (increased bicarbonate).

(Choice D) Preeclampsia commonly presents after 20 weeks gestation with proteinuria or evidence of end-organ damage. Patients typically develop severe hypertension, headaches, nausea/vomiting, and vision problems (eg, blurry vision). Laboratory studies usually show thrombocytopenia, elevated liver enzymes, microangiopathic hemolytic anemia, and elevated creatinine >1.1 mg/dL.

(Choice E) Pregnancy is a prothrombotic state that can increase risk of deep venous thrombosis and/or pulmonary embolism (PE). PE usually presents with hypoxia, tachypnea, and respiratory alkalosis. This patient's absence of hypoxia makes PE unlikely.

# Educational objective:

The elevated progesterone during pregnancy stimulates the respiratory centers in the brain to cause increased tidal volume, increased minute ventilation, increased PaO<sub>2</sub>, and a physiological chronic compensated respiratory alkalosis.

### References

- · Renal physiology of pregnancy.
- Respiratory physiologic changes in pregnancy.





















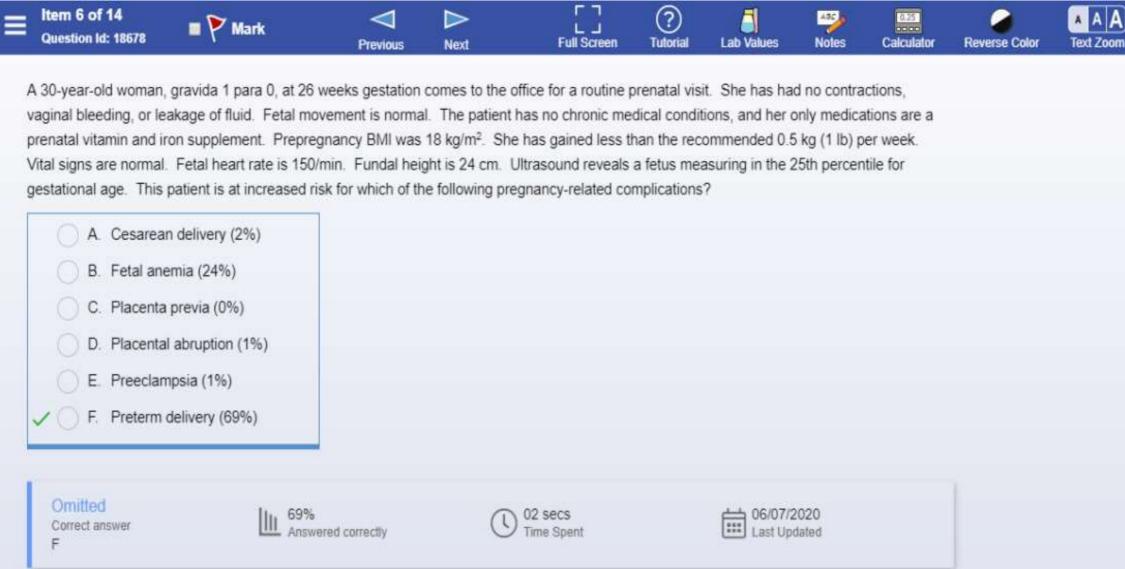




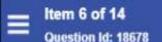
A 30-year-old woman, gravida 1 para 0, at 26 weeks gestation comes to the office for a routine prenatal visit. She has had no contractions, vaginal bleeding, or leakage of fluid. Fetal movement is normal. The patient has no chronic medical conditions, and her only medications are a prenatal vitamin and iron supplement. Prepregnancy BMI was 18 kg/m². She has gained less than the recommended 0.5 kg (1 lb) per week. Vital signs are normal. Fetal heart rate is 150/min. Fundal height is 24 cm. Ultrasound reveals a fetus measuring in the 25th percentile for gestational age. This patient is at increased risk for which of the following pregnancy-related complications?

( A.	Cesarean delivery
○ B.	Fetal anemia
○ C.	Placenta previa
O D.	Placental abruption
○ E.	Preeclampsia
○ F.	Preterm delivery

Submit



























Weight gain in pregnancy			
Prepregnancy BMI (kg/m²)	ldeal weight gain	Complications	
<18.5	28-40 lb (12.7-18 kg)	Inadequate weight gain  • Low birth weight	
18.5-24.9	25-35 lb (11.4-15.9 kg)	Preterm delivery     Excessive weight gain	
25-29.9	15-25 lb (6.8-11.4 kg)	Gestational diabetes mellitus	
≥30	11-20 lb (5-9 kg)	Fetal macrosomia     Cesarean delivery	

During pregnancy, women have increased nutritional requirements because both calories and nutrients (eg, folate, calcium, iron) are preferentially shunted to the developing fetus. To meet these metabolic demands, average women should increase their intake by approximately 350-450 kcal/day in the second and third trimesters.

The target goals for gestational weight gain depend on prepregnancy BMI, which is an indicator of baseline maternal fat and nutrient stores.

Patients with an **underweight prepregnancy BMI** (ie, <18.5 kg/m²) have low baseline stores and therefore require greater weight gain (~1 lb [0.5 kg]/week) to maintain a healthy pregnancy.

This patient is both underweight and has had **inadequate gestational weight gain**, which along with the lagging fundal height (eg, 24 cm at 26 weeks gestation) and fetal growth in the 25th percentile suggest that this patient's nutrition may become insufficient for appropriate fetal development. This depleted state increases the risk for **pregnancy-related complications**, including **preterm delivery** (which occurs when patients can no longer shunt enough nutrients to support the fetus) and **low birth weight** (ie, small for gestational age infant).

(Choices A and E) Women with a high prepregnancy BMI (eg, ≥30 kg/m²) and excessive gestational weight gain are at increased risk of cesarean delivery due to higher rates of gestational diabetes mellitus, fetal macrosomia, and labor dystocia. Obesity also increases the risk of hypertension and preeclampsia due to increased systemic vascular resistance.

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(Choice B) Unlike adult anemia, which is commonly due to iron deficiency (eg, inadequate nutrition, blood loss), fetal anemia typically occurs due to fetal red blood cell destruction (ie, hemolysis). Common causes include Rh alloimmunization, which is caused by maternal autoantibodies against fetal red blood cells, and parvovirus B19 infection, which is cytotoxic to fetal red blood cell precursors.

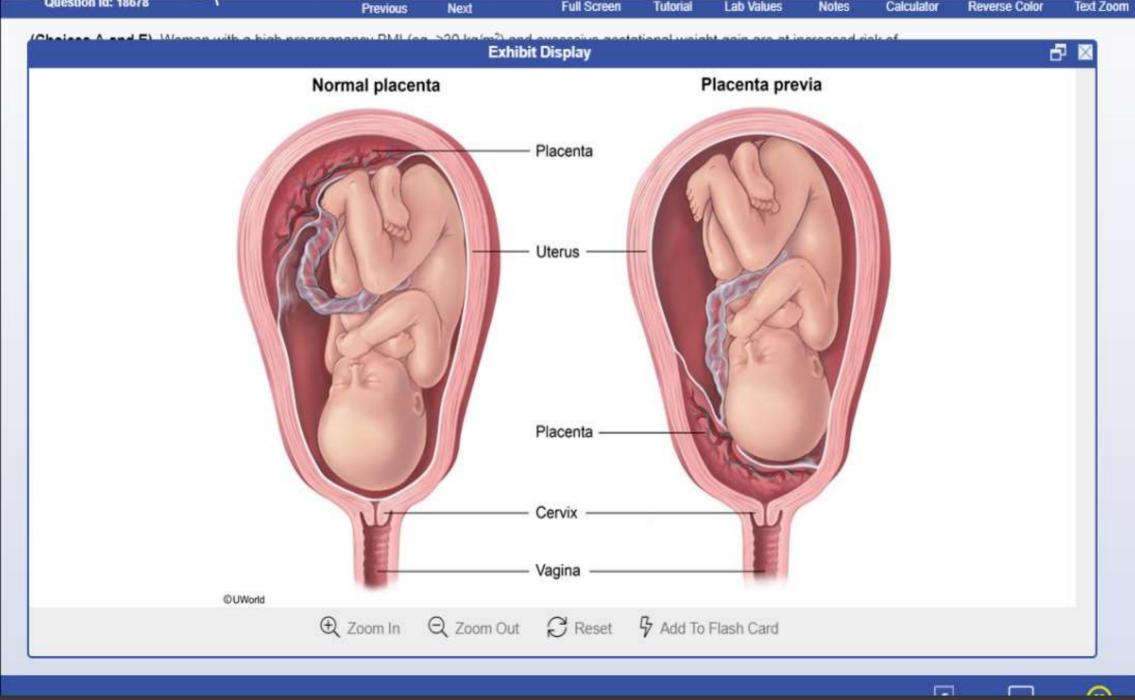
(Choice C) Risk factors for placenta previa include prior cesarean delivery (due to uterine scarring) and multiple gestation (due to increased placental surface area).

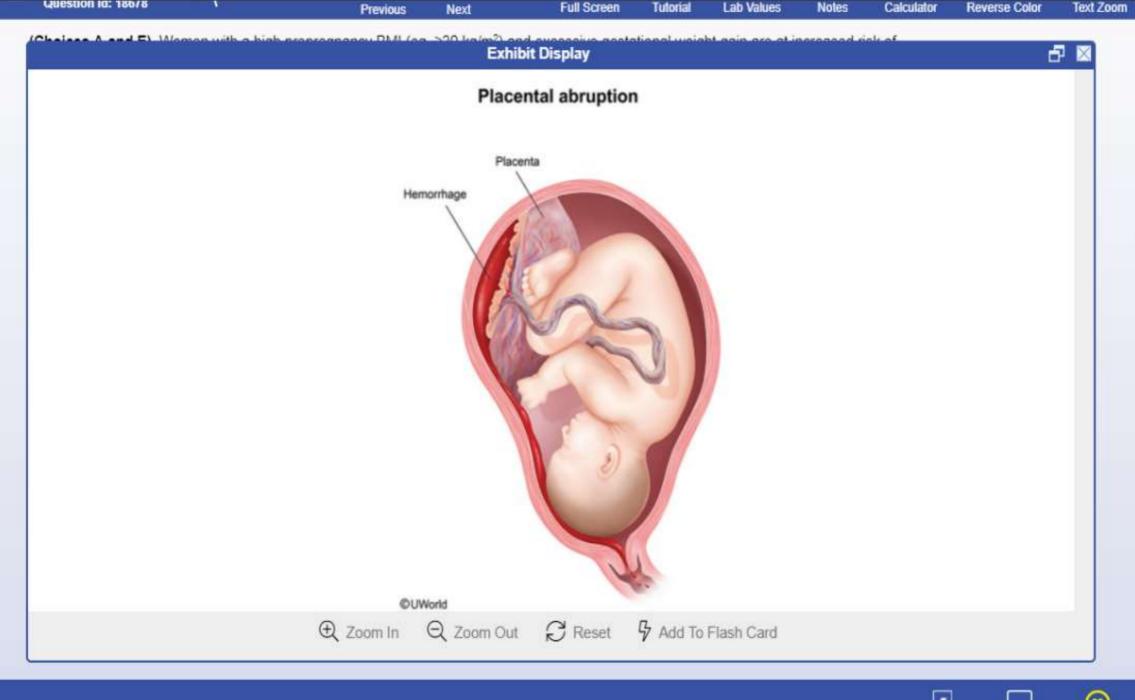
(Choice D) Risk factors for placental abruption include abdominal trauma and conditions that disrupt the vascular integrity of the placenta (eg, maternal hypertension, smoking, cocaine use). Inadequate gestational weight gain is not associated with placental abruption.

# Educational objective:

Patients with an underweight prepregnancy BMI and inadequate gestational weight gain are at increased risk of pregnancy-related complications (eg, low birth weight, preterm delivery).



























A 24-year-old primigravida comes to the office for her first prenatal visit. Her last menstrual period was 8 weeks ago and 2 home pregnancy tests have been positive. The patient feels generally well, although she has experienced mild intermittent nausea and anxiety during the pregnancy. Medical history is unremarkable, and her only medication is an over-the-counter prenatal vitamin. Thyroid function tests were normal on evaluation for fatigue and mild palpitations 6 months ago. The patient stopped drinking alcohol after finding out she was pregnant, and she does not use tobacco or illicit drugs. Blood pressure is 120/80 mm Hg and pulse is 82/min. Physical examination findings are normal. Pelvic ultrasound shows a gestational sac containing a fetal pole and a yolk sac. Thyroid function study results are as follows:

	Today	Six months ago
TSH	0.3 µU/mL	2.0 µU/mL
Total thyroxine (T4)	15 μg/dL	8 µg/dL
Total triiodothyronine (T3)	220 ng/dL	130 ng/dL

Which of the following is the most likely cause of this patient's current laboratory findings?

A. Euthyroid sick syndrome	
B. Graves disease	
C. Hashimoto disease	
D. lodine deficiency	
E. Normal physiologic changes	
F. Subacute thyroiditis	





















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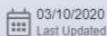
B. Graves disease (13%)

Euthyroid sick syndrome (2%)

- C. Hashimoto disease (2%)
- D. lodine deficiency (0%)
- E. Normal physiologic changes (74%)
- F. Subacute thyroiditis (6%)





























Hormone	Change	Mechanism
Total T4	Increased	β-hCG stimulates thyroid hormone production in first trimester
Free T4	Unchanged or mildly increased	<ul> <li>Estrogen stimulates TBG; thyroid increases hormone production to maintain steady free T4 levels</li> </ul>
тѕн	Decreased	<ul> <li>Increased β-hCG &amp; thyroid hormone suppress TSH secretion</li> </ul>

-numan chononic gonadotropin, 166 ©UWorld.

Thyroid hormone production increases during pregnancy to cope with metabolic demands. This is accomplished by 2 mechanisms:

- Estrogen stimulates synthesis of thyroxine-binding globulin (TBG) and decreases TBG clearance, leading to an increased pool of bound thyroid hormone. Patients with normal thyroid reserve subsequently increase thyroid hormone production to maintain free hormone levels.
- hCG (which shares a common alpha subunit with TSH and a very similar beta subunit) directly stimulates TSH receptors. This results in increased hormone production with feedback suppression of pituitary TSH release.

In patients with normal thyroid function, the net effect (as seen in this patient) can resemble subclinical hyperthyroidism with increased circulating total T3 and T4, normal (or mildly elevated) free T4, and suppressed TSH, although the patient remains clinically euthyroid. However, patients with impaired thyroid function (eg. chronic lymphocytic [Hashimoto] thyroiditis) cannot increase thyroid hormone production, resulting in a relative hypothyroid state (Choice C). For this reason, patients with baseline hypothyroidism who become pregnant should have their replacement thyroxine dose increased.





















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- (Choice A) Euthyroid sick syndrome is an alteration in biochemical thyroid function tests in the setting of severe nonthyroid illness. Common findings include a low total and free T3 with a normal T4 and TSH.
- (Choice B) Graves disease causes an increase in total and free thyroid hormone levels. However, most patients will have additional manifestations, such as proptosis, diffuse goiter, hypertension, and tachycardia.
- (Choice D) Iodine requirements increase during pregnancy, and inadequate iodine intake can cause maternal and fetal hypothyroidism. Such patients would have an elevated TSH with decreased thyroid hormone levels.
- (Choice F) Subacute (granulomatous or De Quervain) thyroiditis can cause hyperthyroidism with a suppressed TSH. However, it most commonly follows an acute viral illness and is associated with a painful, tender goiter.

## Educational objective:

Thyroid hormone production increases during pregnancy to cope with metabolic demands. Estrogen causes an increase in thyroxine-binding globulin, leading to increased total (but not free) thyroid hormone levels. hCG directly stimulates TSH receptors, causing increased production of thyroid hormones.

### References