THYROID AND *PULMONARY* DISORDERS IN PREGNANCY

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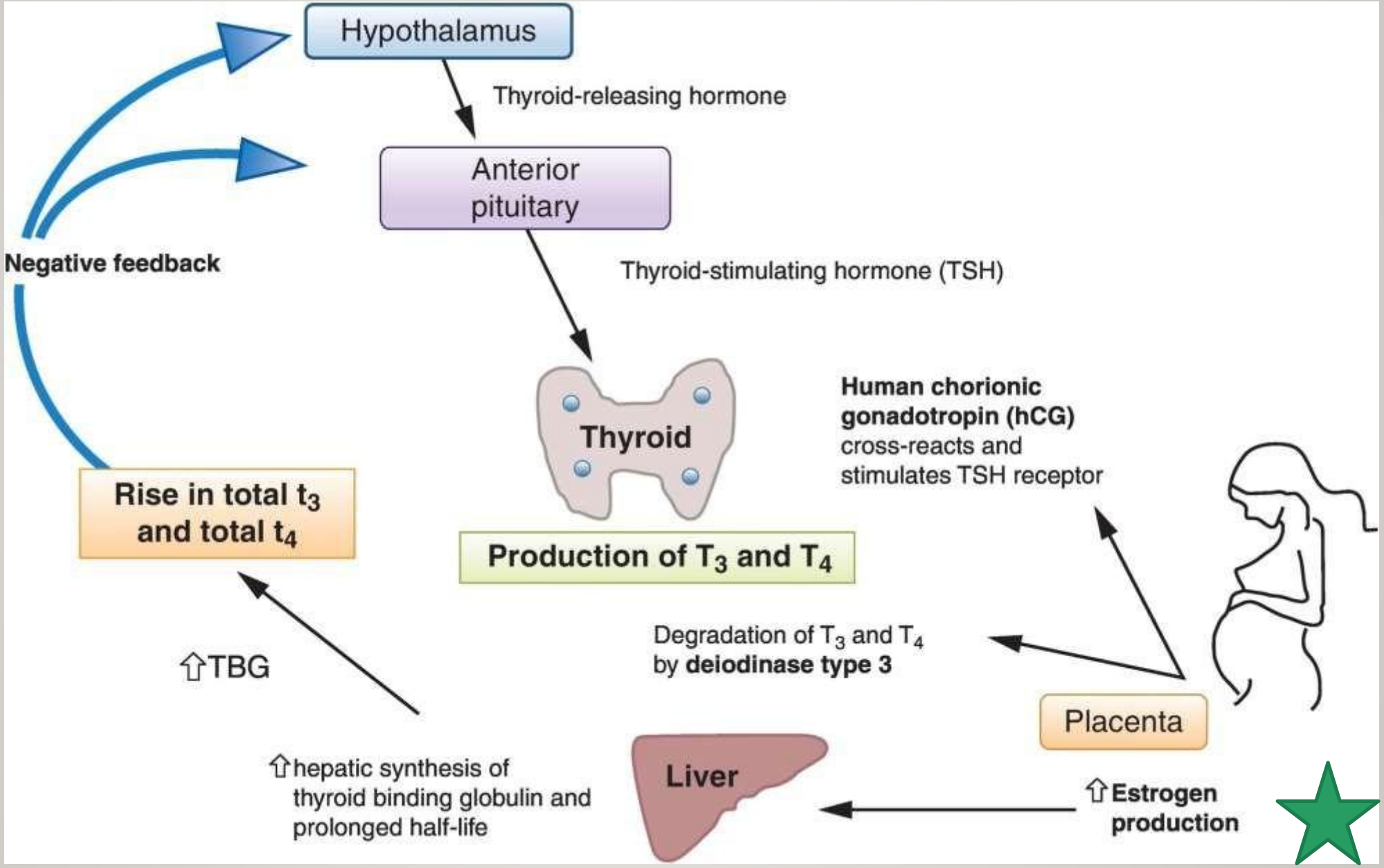
THYROID DISEASE AND PREGNANCY

References: (UpToDate Articles)

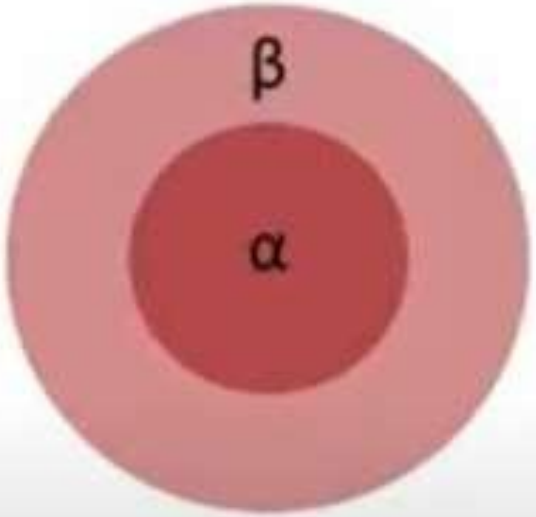
1. Overview of thyroid disease and pregnancy
2. Hyperthyroidism during pregnancy: Clinical manifestations, diagnosis, and causes
3. Hyperthyroidism during pregnancy: Treatment
4. Hypothyroidism during pregnancy: Clinical manifestations, diagnosis, and treatment

THYROID ADAPTATION DURING NORMAL PREGNANCY

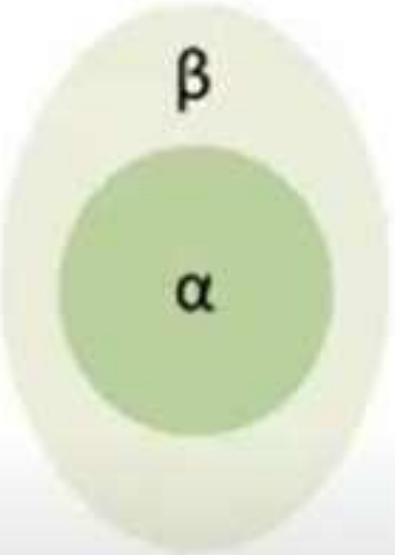
The major changes in thyroid function during pregnancy are	
Estrogen increases TBG production and decreases TBG clearance	**Stimulation of the TSH receptor by hCG**
↓	↓
Increased T4 and T3 production to maintain the free T4 and T3 concentrations	**hCG and TSH are members of glycoprotein hormones family with a common alpha subunit and a unique beta subunit**
↓	↓
The TBG excess leads to an increase in both serum total, but not free, T4 and T3 concentrations	**As a result, hCG has weak thyroid-stimulating activity**


Hypothalamus
Thyroid-releasing hormone
Anterior pituitary
Negative feedback
Thyroid-stimulating hormone (TSH)
Thyroid
Human chorionic gonadotropin (hCG) cross-reacts and stimulates TSH receptor
Rise in total t_3 and total t_4
Production of T_3 and T_4
⇧TBG
Degradation of T_3 and T_4 by deiodinase type 3
Placenta
⇧hepatic synthesis of thyroid binding globulin and prolonged half-life
Liver
⇧Estrogen production

TSH and hCG



TSH



hCG

THYROID ADAPTATION DURING NORMAL PREGNANCY

Thyroid gland size	Increases
TBG	Increases
Total T3 & T4	Increases
Free T3 & T4	No change
TSH & TRH	No change

THYROID ADAPTATION DURING NORMAL PREGNANCY

Serum hCG: concentrations increase soon after fertilization and peak at 10 to 12 weeks

- During this peak: total T3 and T4 are increased
- Serum free T4 and T3 concentrations increase slightly, usually within the normal range
- Serum TSH concentrations are appropriately reduce

This transient, usually subclinical, hyperthyroidism should be considered a normal physiologic finding.

As hCG secretion declines, serum free T4 and T3 concentrations decline and serum TSH concentrations rise slightly to or within the normal range.

THYROID FUNCTION IN THE FETUS

During first trimester the fetus depend on maternal thyroid hormones, which are critical for growth and development

During 10th – 12th week of gestation fetal TSH appears, and the fetal thyroid is capable of concentrating iodine and synthesizing iodothyronines. However, little hormone synthesis occurs until the 18th to 20th week. Thereafter, fetal thyroid secretion increases gradually

At term, fetal serum T4, T3, and TSH concentrations differ substantially from those in the mothers. Serum TSH concentrations are higher, serum free T4

concentrations are lower, and serum T3 concentrations are one-half those of the mothers.

Soon after birth, serum TSH concentrations rapidly increase to 50 to 80 mU/L and then fall to 10 to 15 mU/L within 48 hours.

Serum T3 and T4 concentrations rapidly increase to values slightly higher than those in normal adults.

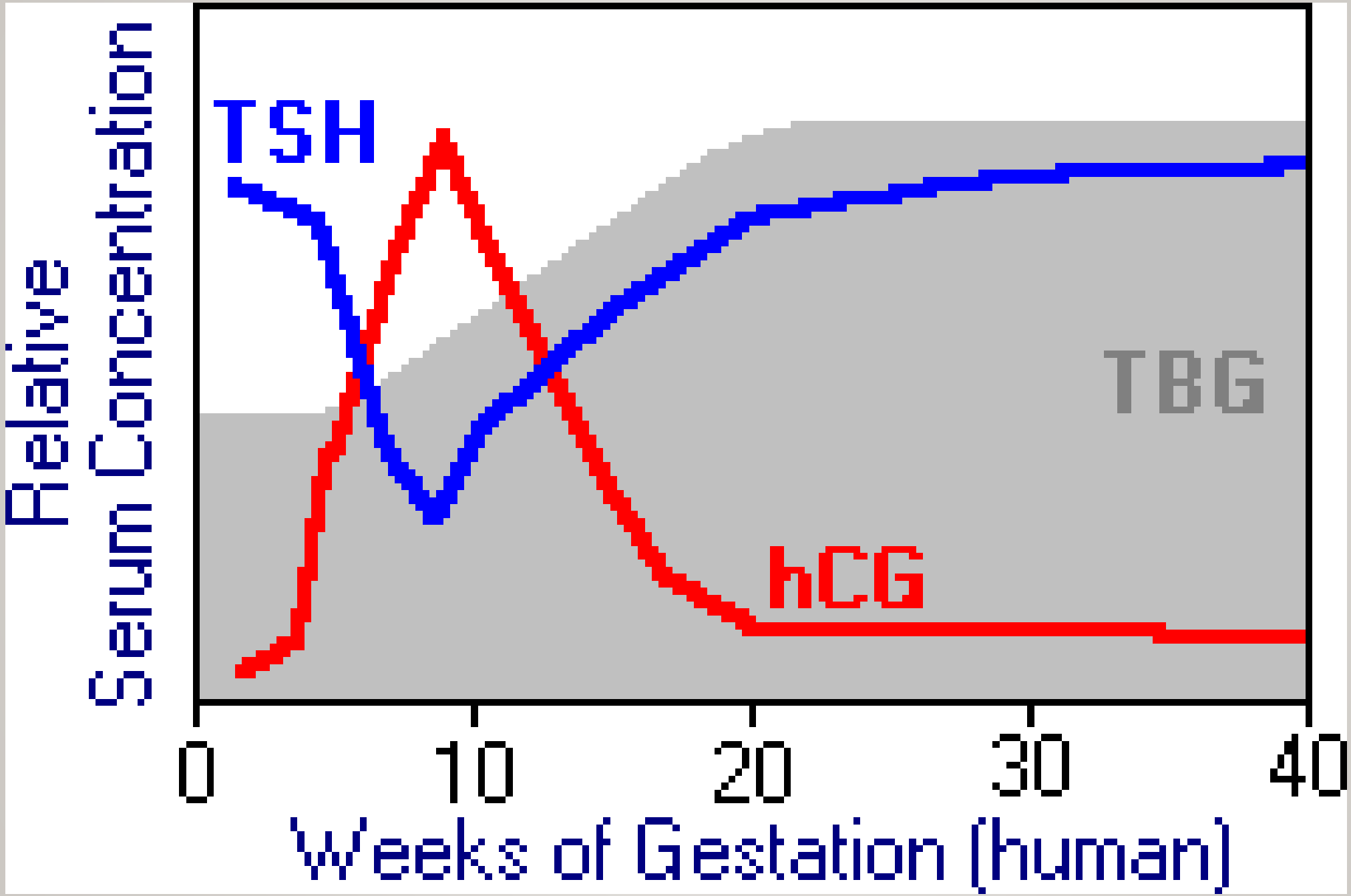
ASSESSMENT OF THYROID FUNCTION

When evaluating thyroid tests during pregnancy, we typically measure:

- **TSH + Free T4** (if there is a trimester-specific reference range) **+/- Total T4**

In the absence of population and trimester-specific normal ranges, ATA guidelines suggest the following for interpretation of thyroid function tests:

- **Weeks 7 to 12** – Reduce the lower limit of the reference range of TSH by approximately 0.4 mU/L and the upper limit by 0.5 mU/L
- **Second and third trimester** – There should be a gradual return of TSH towards the nonpregnant normal range
- The upper reference range for total T4 increases by approximately 5 percent per week, beginning at week 7. At approximately **16 weeks**, total T4 (and T3) levels during pregnancy are 1.5-fold higher than in nonpregnant women (due to TBG excess).


Relative
Serum Concentration
TSH
TBG
hCG
0
10
20
30
40
Weeks of Gestation (human)

HYPOTHYROIDISM DURING PREGNANCY

Etiology

- Hashimoto's thyroiditis (TPO AB)
- Previous radioiodine therapy/thyroid surgery
- Previous postpartum thyroiditis
- Hypopituitarism
- Iodine deficiency

Clinical features

- The range of clinical symptoms of hypothyroidism during pregnancy is similar to those that occur in nonpregnant patients and may include fatigue, cold intolerance, constipation, and weight gain.
- Symptoms may be overlooked or attributed to the pregnancy itself as some of the symptoms of hypothyroidism are similar to those of pregnancy (although cold intolerance is not a normal clinical manifestation of pregnancy)

DIAGNOSIS

Steps

- The diagnosis of primary hypothyroidism during pregnancy is based upon the finding of an elevated serum TSH concentration, defined using population and trimester-specific TSH reference ranges for pregnant women.
- For women with a TSH above the population and trimester-specific upper limit of normal (or **above 4.0** mU/L when local reference ranges are not available), we also measure a free T4 (or total T4, if trimester-specific reference range for free T4 is not provided or if free T4 measurements appear discordant with TSH measurements).
- In addition, measure **thyroid peroxidase (TPO) antibodies in pregnant women with TSH >2.5 mU**/L to inform treatment considerations

Overt primary hypothyroidism: elevated trimester-specific TSH concentration in conjunction with a decreased free T4 concentration

Subclinical hypothyroidism is defined as an elevated trimester-specific serum TSH concentration and a normal free T4 concentration.

PREGNANCY COMPLICATIONS

- Pregnancy complications in Overt hypothyroidism
  - Preeclampsia and gestational hypertension
  - Placental abruption
  - Non-reassuring fetal heart rate tracing
  - Preterm delivery, including very preterm delivery (before 32 weeks)
  - Low birth weight
  - Increased rate of cesarean section
  - Postpartum hemorrhage
  - Perinatal morbidity and mortality
  - Neuropsychological and cognitive impairment in the child

- The risk of complications during pregnancy is lower in women with subclinical, rather than overt and higher if TPO is positive

SCREENING

Because overt and subclinical hypothyroidism are associated with pregnancy complications, including pregnancy loss, and thyroid tests are widely available and easy to perform

Whom to screen ?

- Living in an area of moderate to severe iodine insufficiency
- Symptoms of hypothyroidism
- Family or personal history of thyroid disease
- Personal history of: Thyroid peroxidase (TPO) antibodies, G oiter, Age >30 years, Type 1 diabetes, Head and neck irradiation, Recurrent miscarriage or preterm delivery, Multiple prior pregnancies (two or more), Class 3 obesity (body mass index [BMI] ≥40 kg/m2), Infertility, Prior thyroid surgery, Use of amiodarone, lithium, or recent administration of iodinated radiologic contrast agents

The results of observational studies suggest that assessment of thyroid function only in women at high risk for thyroid or other autoimmune disease (targeted screening) will miss up to one-third of women with subclinical or overt hypothyroidism. However, in prospective trials, universal screening compared

with a targeted approach or with no screening did not improve pregnancy outcomes

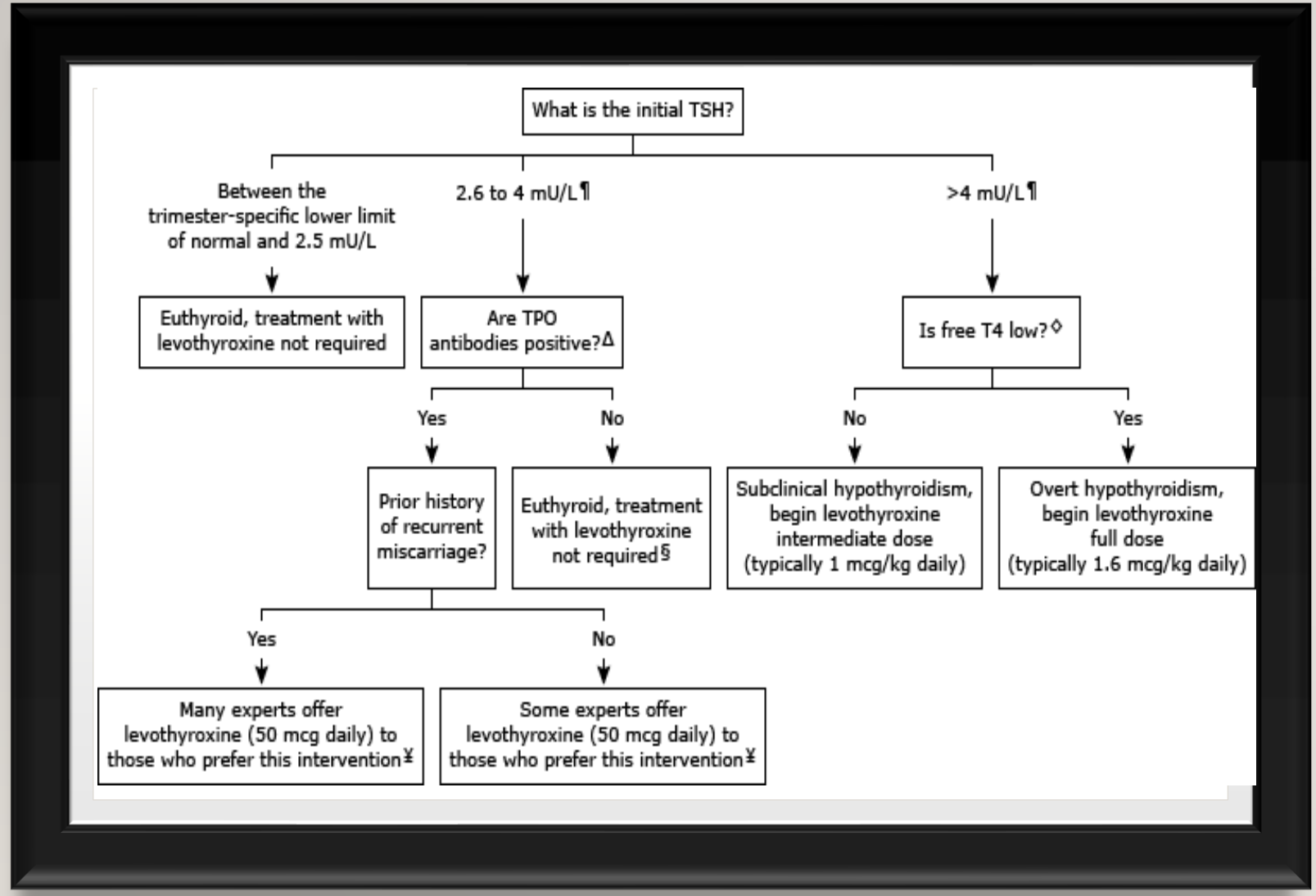
(NO guidlines – screen all women)

APPROACH TO SCREENING

In women who meet the case-finding criteria, we suggest measurement of serum TSH during the first trimester as the screening test for hypothyroidism:

- If the serum TSH is between the trimester-specific lower limit of normal and 2.5 mU/L, most women require no further testing.
- In women at particularly high risk for developing hypothyroidism during pregnancy (post-radioiodine treatment, post-hemithyroidectomy, history of exposure to high-dose irradiation of the head or neck region), we reassess TSH during pregnancy (eg, approximately every four weeks during the first trimester, and then once during each of the second and third trimesters).
- If the serum TSH is >2.5 mU/L, we measure TPO antibodies.
- Women with positive TPO antibodies have a worse outcome. Therefore, the presence of TPO antibodies may be useful for making treatment decisions in women with borderline thyroid function tests

TREATMENT


What is the initial TSH?
Between the trimester-specific lower limit of normal and 2.5 mU/L
2.6 to 4 mU/L¶
>4 mU/L¶
Euthyroid, treatment with levothyroxine not required
Are TPO antibodies positive?Δ
Is free T4 low?◊
Yes
No
No
Yes
Prior history of recurrent miscarriage?
Euthyroid, treatment with levothyroxine not required§
Subclinical hypothyroidism, begin levothyroxine intermediate dose (typically 1 mcg/kg daily)
Overt hypothyroidism, begin levothyroxine full dose (typically 1.6 mcg/kg daily)
Yes
No
Many experts offer levothyroxine (50 mcg daily) to those who prefer this intervention¥
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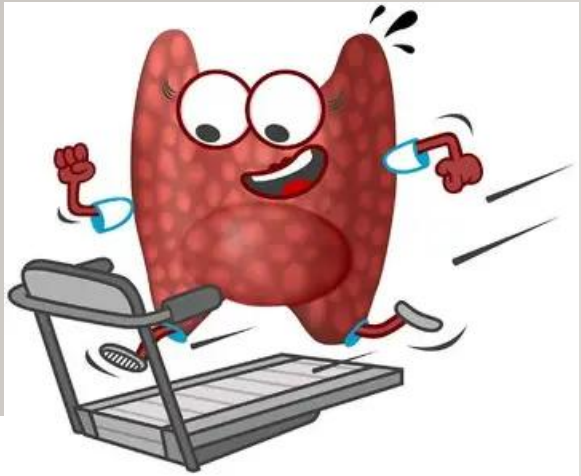


HYPERTHYROIDISM DURING PREGNANCY

HYPERTHYROIDISM DURING PREGNANCY

Epidemiology: Hyperthyroidism is rare in pregnancy (< 0.5%)

Etiology: (the most common causes)

- **Graves disease** usually becomes less severe during the later stages of pregnancy

β-hCG-mediated hyperthyroidism usually occurs transiently in the first half of gestation and is typically less severe than Graves disease(repeat TSH after 1 month– normal)

Clinical features

- Many of the nonspecific symptoms associated with pregnancy are similar to those associated with hyperthyroidism, including tachycardia, heat intolerance, and increased perspiration.
- Additional symptoms include anxiety, hand tremor, and weight loss despite a normal or increased appetite.
- Specific findings such as **goiter** and **ophthalmopathy** suggest Graves' hyperthyroidism

HCG-MEDIATED HYPERTHYROIDISM

Gestational transient thyrotoxicosis (GTT)

- During the time of peak hCG concentrations (10 to 12 weeks), total serum T4 and T3 concentrations increase. Serum free T4 and T3 concentrations increase slightly, usually within the normal range, and serum TSH concentrations are appropriately reduced. Thus, in some women, the high serum concentration of hCG during early pregnancy can lead to subclinical or mild overt hyperthyroidism characterized by slightly low serum TSH concentrations and high-normal or mildly elevated serum free T4 concentrations

Hyperemesis gravidarum

- Hyperemesis gravidarum is a **syndrome of nausea and vomiting associated with weight loss of 5 percent or more during early pregnancy** that occurs in 0.1 to 0.2 percent of pregnancies. Women who develop hyperemesis gravidarum have **higher serum hCG and estradiol** concentrations than normal pregnant women; in addition, their hCG has more thyroid-stimulating activity (mortality rate is higher due to dehydration, renal failure and electrolyte imbalance)

HCG-MEDIATED HYPERTHYROIDISM

Trophoblastic hyperthyroidism

- Hyperthyroidism can also occur with gestational trophoblastic disease. A hydatidiform mole (molar pregnancy) is benign but may give rise to choriocarcinoma. Both are associated with high serum hCG concentrations and abnormal hCG isoforms
- Thus suspect a molar pregnancy or choriocarcinoma if severe hyperthyroidism manifests during pregnancy

Familial gestational hyperthyroidism

- Recurrent gestational hyperthyroidism has been described in one family due to a mutant thyrotropin receptor that is hypersensitive to physiologic concentrations of hCG

TYPES OF HYPERTHYROIDISM IN PREGNANCY

- **Clinical hyperthyroidism**
- **Subclinical hyperthyroidism**
- **Free T4 in the upper normal quintile**

Table 2. Trimester-Specific Reference Ranges for Common Thyroid Tests

Test	*Nonpregnant*	*First trimester*	*Second trimester*	*Third trimester*
Thyroid-stimulating hormone (mIU per L)	0.3 to 4.3	0.1 to 2.5	0.2 to 3.0	0.3 to 3.0
Thyroxine-binding globulin (mg per dL)	1.3 to 3.0	1.8 to 3.2	2.8 to 4.0	2.6 to 4.2
Thyroxine, free (ng per dL)	0.8 to 1.7	0.8 to 1.2	0.6 to 1.0	0.5 to 0.8
Thyroxine, total (mcg per dL)	5.4 to 11.7	6.5 to 10.1	7.5 to 10.3	6.3 to 9.7
Triiodothyronine, free (pg per mL)	2.4 to 4.2	4.1 to 4.4	4.0 to 4.2	Not reported
Triiodothyronine, total (ng per dL)	77 to 135	97 to 149	117 to 169	123 to 162

Information from references 3 and 7.

TYPES OF HYPERTHYROIDISM IN PREGNANCY

Overt/clinical hyperthyroidism

- **Definition**: Low TSH, with free T4 and/or T3 levels that exceed trimester-specific normal reference ranges or total T4 and T3 that exceed 1.5 times the nonpregnant range
- **Causes**: Graves disease, β-hCG-mediated hyperthyroidism in cases of hydatidiform moles/choriocarcinoma
- **Pregnancy complications**:
  - Spontaneous abortion
  - Premature labor
  - Low birth weight
  - Stillbirth
  - Preeclampsia
  - Heart failure
  - Neonatal hyperthyroidism

TYPES OF HYPERTHYROIDISM IN PREGNANCY

Subclinical hyperthyroidism

- **Definition**: Low TSH, with normal free T4 and T3 using trimester-specific normal reference ranges or total T4 and T3 that are less than 1.5 times the nonpregnant range
- **Causes**: β-hCG-mediated hyperthyroidism in cases of hyperemesis gravidarum
- **Pregnancy complications**: you should treat the patient to avoid clinical hyperthyroidism .

Free T4 in the upper-normal quintile

- **Definition**: A normal free T4 in the upper quintile with a normal TSH
- **Causes**:
- **Pregnancy complications**: Associated with lower birth weight, and maternal hypertension

TREATMENT

Goal of treatment:

maintain persistent but mild hyperthyroidism in the mother to prevent fetal hypothyroidism since the fetal thyroid is more sensitive to the action of antithyroid drugs

Indications for treatment:

Women with symptomatic, moderate to severe, overt hyperthyroidism

Not all patients with biochemical, overt hyperthyroidism require treatment if the hyperthyroidism is mild, since the goal of treatment is to maintain mild maternal hyperthyroidism

In women who are being monitored without therapy, we measure TSH, free T4 (if there is a trimester-specific reference range), and/or total T4 or total T3 every four to six weeks.

TREATMENT

Control of symptoms

- **Agents**: β-blockers such as metoprolol or propranolol (2-6 w)
- **Indication**: Symptomatic, moderate to severe hyperthyroidism
- **Notes**: should be weaned as soon as the hyperthyroidism is controlled by thionamides because it can result in neonatal growth restriction, hypoglycemia, respiratory depression, and bradycardia

Decrease thyroid hormone synthesis:

- **Agents**: Thionamides
- **Notes**: Propylthiouracil (1st trimester), methimazole (2nd trimester)/category c

Surgery

- **Indication**: Thionamide intolerance
- **Notes**: Safest in second trimester

CHOICE OF THIONAMIDE

Diagnosed prior to pregnancy (pt. takes methimazole)

- Elect to have definitive therapy with surgery or radioiodine prior to pregnancy
- Switch to PTU before trying to conceive. (for younger women with normal periods)
- Switch to PTU as soon as the pregnancy test is confirmed. (for older individuals having difficulty conceiving)

Diagnosed during the first trimester

- Women diagnosed with symptomatic, moderate to severe hyperthyroidism during the first trimester of pregnancy should take PTU
- After the 1st trimester patient can be kept on PTU or changed to methimazole

Diagnosed after the first trimester

- Women diagnosed with symptomatic, moderate to severe hyperthyroidism after the first trimester should take methimazole

NEONATAL HYPERTHYROIDISM

Occurs in ~ 5% of babies born to mothers with Graves disease

Etiology: transplacental passage of maternal TRAbs

Clinical features

- Hyperthyroidism: irritability, restlessness, tachycardia, diaphoresis, hyperphagia, poor weight gain, diffuse goiter (can cause tracheal compression), microcephaly (due to craniosynostosis)
- May arise directly after birth or delayed up to 10 days later as a result of transplacental maternal antithyroid medication (including propylthiouracil or carbimazole)

Treatment

- Neonatal Graves disease resolves within 1–3 months
- Infants with symptomatic hyperthyroidism: methimazole and propranolol

Complications

- Untreated symptomatic hyperthyroidism in infants can cause cardiac failure and intellectual disability.

PULMONARY DISORDERS IN PREGNANCY

RESPIRATORY CHANGES DURING PREGNANCY

Increase in Oxygen demand in Pregnancy and **increase Pulmonary Blood Flow**.

↑ Intraabdominal pressure through uterine growth → dyspnea

The enlarging uterus produce anatomical changes to the thoracic cage, the diaphragm is displaced upward by as much as 4 cm.

The anteroposterior and transverse diameter of the thorax increases, which enlarges chest wall circumference.

Progesterone stimulates the respiratory centers in the brain → hyperventilation (to eliminate fetal CO_2 more efficiently) → physiological, chronic compensated respiratory alkalosis

↑ Tidal volume → ↑ minute ventilation

↓ PCO_2 (~ 30 mm Hg)

VENOUS THROMBOEMBOLISM

VTE can manifest during pregnancy as an isolated lower extremity deep vein thrombosis (DVT) or clot can break off from the lower extremities and travel to the lung to present as a pulmonary embolus.

PE is the second leading cause of maternal mortality, responsible for 9 percent of maternal deaths . Thus, the detection of DVT during pregnancy is critical to preventing deaths from PE.

VENOUS THROMBOEMBOLISM

Antepartum – Most studies report equal distribution of VTE across the trimesters of pregnancy .However, two large conflicting retrospective studies reported a first trimester predominance (50 % before 15 weeks) and third trimester predominance (60 %)

Postpartum – Compared with the antepartum period, VTE is two to five times more common postpartum .

The risk is highest in the first six weeks postpartum and declines to rates that approximate that of the general population by about 13 to 18 weeks.

ANTEPARTUM RISK FACTORS:

Varicose veins

Diabetes

Hospitalization for non-delivery reasons (particularly those >3 days)

Increased maternal age ≥35 years

Body mass index (BMI) ≥30 kg/m2

Multiple births

Urinary tract infection

POSTPARTUM RISK FACTORS

- **Cesarean section** (CS), especially emergent CS
- **Medical comorbidities** (eg, varicose veins, cardiac disease, inflammatory bowel disease)
- **BMI ≥25 kg/m2**

•**Inherited thrombophilias** — The risk of VTE is further magnified in pregnant women who have inherited thrombophilias (eg, such as factor V Leiden; antithrombin III, protein S, or protein C deficiency; or antiphospholipid syndrome)

DVT

Signs and symptoms :

1. Swelling of the leg/calf
2. Warmth and redness of the Leg
3. Pain that may worsen when standing/walking

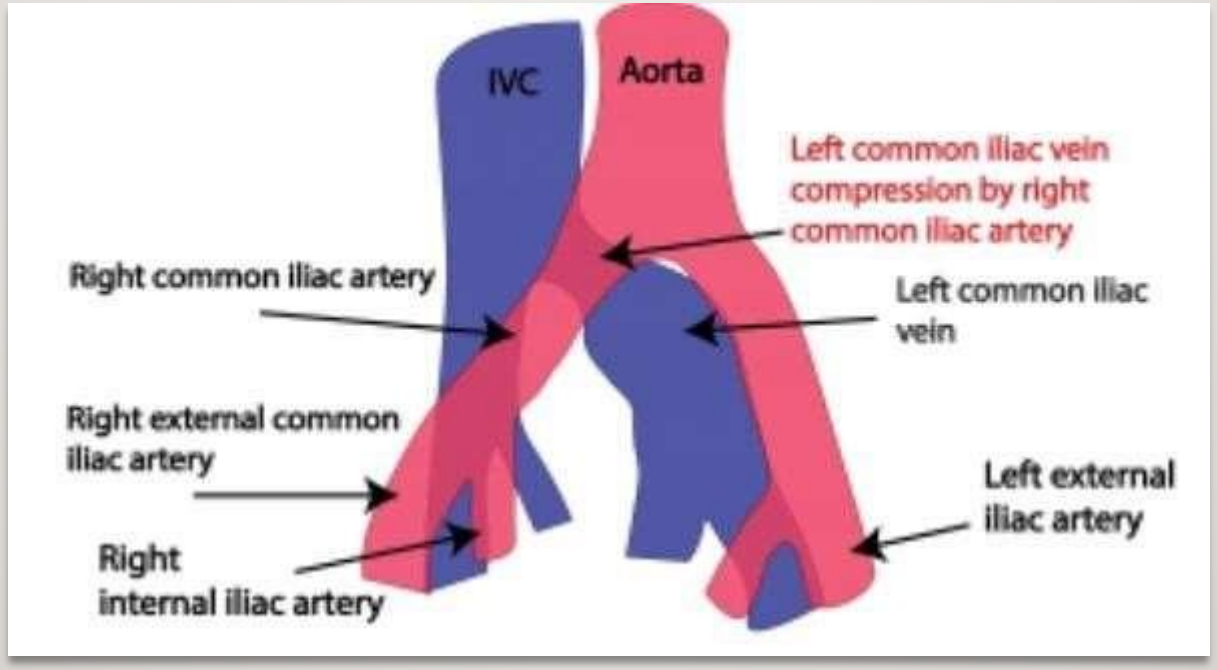
Types of DVT according to anatomical location :

1 Pelvic DVT :develops in one of the deep veins in the pelvis, which may result in pelvic pain.

2 Left lower extrimety DVT(MOST COMMON)

DVT

left leg related to compression of the left iliac vein by the right iliac artery, coupled with compression of the inferior vena cava by the gravid uterus



DIAGNOSIS

D-Dimer Test : Has a very high sensitivity (95%), but low specificity (50%); can be used to rule out DVT when combined with Doppler and clinical suspicion.

Wells score : 2 points or more

Proximal vein CUS is a highly sensitive and specific tool for the diagnosis of DVT in both pregnant and nonpregnant patients. However, it is less sensitive for pelvic vein thrombosis

In cases where CUS is negative, poor Doppler flow in the iliac vein has reasonable accuracy for the diagnosis of suspected pelvic vein DVT.

Table 1 Two-level DVT Wells score

Clinical feature	**Points**
Active cancer (treatment ongoing, within 6 months, or palliative)	1
Paralysis, paresis or recent plaster immobilisation of the lower extremities	1
Recently bedridden for 3 days or more, or major surgery within 12 weeks requiring general or regional anaesthesia	1
Localised tenderness along the distribution of the deep venous system	1
Entire leg swollen	1
Calf swelling at least 3 cm larger than asymptomatic side	1
Pitting oedema confined to the symptomatic leg	1
Collateral superficial veins (non-varicose)	1
Previously documented DVT	1
An alternative diagnosis is at least as likely as DVT	-2
Clinical probability simplified score	**Points**
DVT likely	2 points or more
DVT unlikely	1 point or less

PULMONARY EMBOLISM

PE during pregnancy are more problematic because dyspnea and tachypnea are common in pregnancy.

Symptoms : a. Dyspnea b. Pleuritic chest pain c. Cough d.Hemoptysis

Diagnosis :

CT Angiogram : sensitivity (>90%) and specificity +clinical suspicion of PE

Lower extremity Compression ultra sonography.

Chest x ray(usually normal)

TREATMENT

1. Low molecular weight heparin.

2. Unfractionated heparin (renal failure patient, high risk of bleeding, risk of emergent surgery).

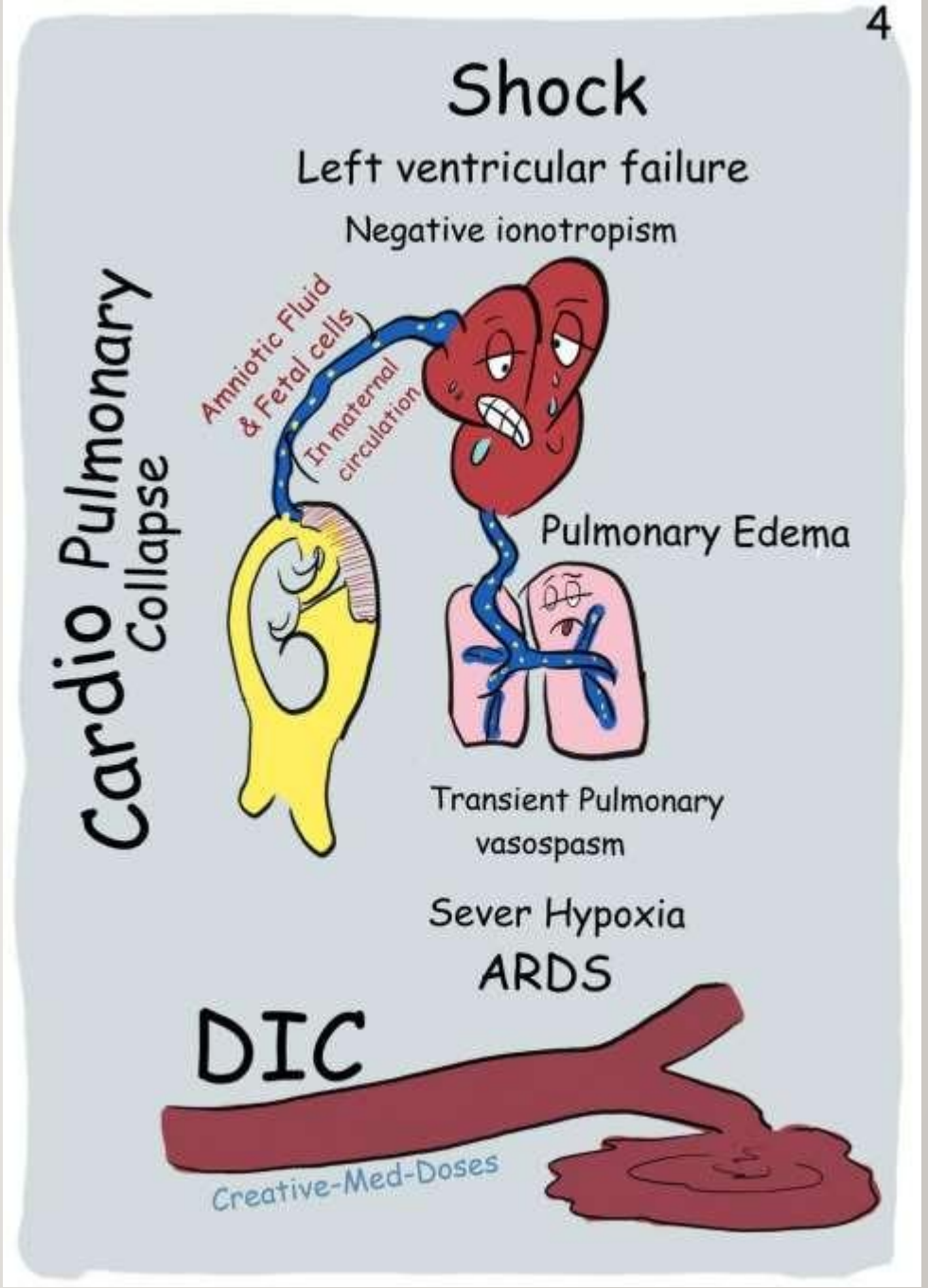
3. Thrombolytic (streptokinase 100 mg over 2 hrs)in hemodynamic unstable patient

AMNIOTIC FLUID EMBOLISM

occurs During labor or shortly after (~ 30 min)/ sudden collapse

It contains Amniotic fluid, fetal cells, fetal debris Enter maternal circulation by endocervical veins or uterine tears, obstructs the pulmonary vessels, and causes vascular spasms, resulting in pulmonary hypertension.

Often fatal



AMNIOTIC FLUID EMBOLISM

Phase I (respiratory/shock)

1. Respiratory distress
2. Hypoxemia
3. Hypotension

Phase II (hemorrhagic phase)

1. Massive hemorrhage
2. DIC

Seizures also often occur

Treatment: supportive care with prompt attention to adequate oxygenation, ventilation, and inotropic support

ASTHMA

➢ **Asthma** is a chronic inflammatory disorder characterized by bronchial hyperreactivity, and it is the most common pulmonary disease in pregnancy, affecting between 3% and 9% of pregnant women.

Can pregnancy make asthma worse or better?

Research suggests that asthma severity during pregnancy is related to asthma severity before pregnancy.
Symptoms are more likely to worsen in people with severe asthma.
If asthma gets better, the improvement is generally gradual as the pregnancy progresses.
If asthma worsens, the increase in symptoms is most noticeable during the **first** and **third** trimesters of pregnancy.
Some women might experience worse asthma signs and symptoms early in pregnancy because they stop taking their medications after becoming pregnant. Any changes you make to your medication routine might also influence the severity of your asthma.

ANY INVESTIGATION TO FOLLOW UP DURING PREGNANCY?

- Hypoxia may contribute to low birth weight, pre-eclampsia, congenital malformations, spontaneous abortions and placenta previa in asthmatic females . Reduced partial pressure of oxygen (PO2) is a feature of acute severe asthma or status asthmaticus and a small decrease in maternal PO2 can have serious effects on the fetus

- Ultrasounds starting at week 32 of your pregnancy to monitor baby growth and activity.

FEATURES OF SEVERE LIFETHREATENING ASTHMA

1. Peak expiratory flow rate <35% of predicted
2. pO2 <8 KPa (60mmHg)
3. pCO2 >4.6 Kpa
4. Silent chest.
5. Cyanosis.
6. Bradycardia.
7. Arrhythmia.
8. Hypotension.
9. Exhaustion.
10. Confusion.

IS IT SAFE TO TAKE ASTHMA MEDICATION DURING PREGNANCY?

Most asthma medications can be safely used during pregnancy.

Also, it's safer to take asthma medications during pregnancy than it is to experience asthma symptoms or an asthma attack. If you're having trouble breathing, your baby might not get enough oxygen.

WHAT ABOUT DELIVERY AND BREAST FEEDING?

Most women don't experience major asthma symptoms during labor and delivery.

If the patient takes asthma medication, continue doing so during labor and delivery.

Breast-feeding is encouraged for women who have asthma — even if they take medication.

TREATMENT GUIDELINES

- For those with **mild intermittent asthma**, a short-acting inhaled β2- agonist (albuterol) can be used as needed.
- Patients with **mild persistent asthma** should be treated with a daily low-dose inhaled glucocorticoid (budesonide).
- The preferred treatment for **moderate persistent asthma** is either a daily medium-dose inhaled glucocorticoid or a combination of a daily low-dose inhaled glucocorticoid and a long-acting β2-agonist (salmeterol).
- Those with **severe persistent asthma** should be treated with a daily high-dose inhaled glucocorticoid combined with a long-acting β2-agonist. They may require the addition of a systemic glucocorticoid.

- **Acute severe exacerbations** must be treated aggressively with oxygen therapy, intravenous fluids, systemic glucocorticoids, administration of short-acting β2-agonists and ipratropium by nebulized aerosol , and antibiotics if there is evidence of bacterial infection. Intravenous magnesium sulfate or subcutaneous terbutaline can be added if needed.

- To prevent fetal hypoxia, pulse oximetry should be used and oxygen saturation should be maintained at 95% or greater

- Because of the hyperventilation and compensated respiratory alkalosis present in normal pregnancy, **an arterial blood gas with a PaO_2 less than 70 mm Hg and/or a $PaCO_2$ greater than 35 mm Hg are indicative of severe respiratory compromise**

- Serial fetal monitoring and ultrasonic assessment of fetal growth should be implemented

WHAT CAN I DO TO PREVENT COMPLICATIONS?

- **Routine follow up**
- **Take medication as prescribed.**
- **Don't smoke.**
- **Avoid and control triggers.** Avoid exposure to secondhand smoke and other potential
- irritants, such as dust and animal dander.
- **Control gastroesophageal reflux disease (GERD).** GERD — a chronic digestive disease that
- causes acid reflux and heartburn — can worsen asthma
- **Recognize warning signs.** such as coughing, chest tightness, shortness of breath or
- wheezing.

- The timing of delivery is dependent on the status of both the mother and the fetus.
- When pregnancy is progressing well, there is no need for early delivery, and it is advisable to await the spontaneous onset of labor
- Early delivery can be considered for fetal growth restriction or maternal deterioration.

INFLUENZA

- It is the most common viral infection in pregnancy
- Resulting in increased morbidity and mortality
- Risk of hospitalization for an acute cardiopulmonary illness is three to four times more likely in 3rd trimester
- Influenza (H1N1) should be suspected in patients not responding to routine antibiotics and in pneumonia or respiratory failure.
- Increased risk of preterm delivery or low birth weight infant, severe pneumonia

INFLUENZA

- Headache.
- Runny nose.
- Sore throat.
- Fatigue.
- Shortness of breath/Cough.
- Loss of appetite.
- Diarrhea or vomiting.
- Sudden chills or fever.

INFLUENZA

Prevention and supportive care

Anti-pyretics for fever (reduces fetal tachycardia, protective against congenital abnormalities)

Neuraminidase inhibitors (zanamivir, oseltamivir) can be used in the treatment

THANK YOU