



ADDRESSES:

515 N. SAM HOUSTON PARKWAY EAST
SUITE 610
HOUSTON, TEXAS 77060

Healthy CONNECTIONS
"Stay Connected to Your Patient at Home"

Healthy CONNECTIONS

"Stay Connected to Your Patient at Home"

DYSPNEA IN PREGNANCY & OTHER MEDICAL CONDITIONS

JOANIE HARE, MD, FACOG—PERINATOLOGY



Dyspnea is defined as a subjective experience of breathing discomfort, caused by various physiologic, psychological, social and environmental conditions.

There are diverse etiologies of dyspnea caused by various medical conditions. Some examples include the following: pulmonary genetic disorder, such as cystic fibrosis; airway obstruction, such as in epiglottitis; immobilization of the

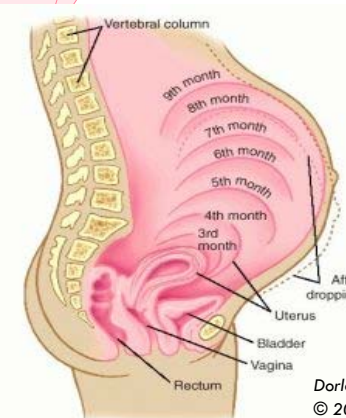
diaphragm, as in patients with spinal cord injuries; or restriction of chest volume, such as with emphysema. Congestive heart failure and valvular lesions may result in shortness of breath.

Other medical conditions that may result in dyspnea include: hemoglobinopathies such as sickle cell disease and metabolic disorders like diabetic ketoacidosis; muscle and nerve disorders, such as in muscular dystrophy; and claustrophobia, a psychological condition which may cause shortness of breath due to small environment confinement. Also, the use of medications, such as narcotics, can produce shortness of breath. Finally, the gravid uterus is a condition that causes shortness of breath and is experienced by most women during their pregnancy.

MATERNAL ADAPTATIONS IN PREGNANCY

There are mechanical, biochemical and physiologic adaptations during pregnancy that result in shortness of breath. First of all there are hormonal changes resulting in the relaxation of the chest wall that produce mechanical transformations of pregnancy. This includes a 2 cm enlargement in the transverse and anterior-posterior diameters of the thorax which results in an 8% increase in the overall thoracic circumference.

The enlarging uterus also generates the sensation of shortness of breath: this produces a 4 -5 cm elevation of the diaphragm. For example, during the first trimester 15% of pregnant women experience dyspnea, 50% by 20 weeks, and 71% by 32 weeks gestation. Thus, dyspnea in pregnancy is enhanced as gestational age increases due to the enlarging uterus.

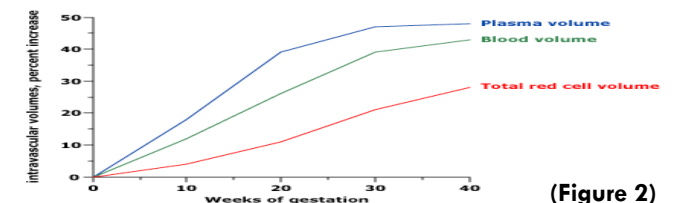


(Figure 1)

Dorland's Medical Dictionary for Health Consumers. © 2007 by Saunders, an Imprint of Elsevier, Inc. All rights reserved.

PHYSIOLOGY OF ANEMIA

There are various other causes of physiologic changes of pregnancy that may affect respiratory rate. One such cause is physiologic anemia during pregnancy. Anemia is defined as hemoglobin of less than 11 g/dl. The estrogenic effect of pregnancy produces an increase in red blood cells and blood volume. The red blood cell volume increases by 20-30% and begins about 10 weeks gestation and continues to term. For example, total blood volume in a singleton gestation increases by 50%. In multiple gestation, this may increase may be greater than 70%. The plasma volume increases by 45- 50% and plateaus by 32 weeks gestation. This results in a dilutional physiologic anemia despite the escalating red blood cell mass (Figure 2). This increase in plasma volume is a protective mechanism for potential blood loss during delivery: delivery may result in significant blood volume loss, but not necessarily entire red cell mass.



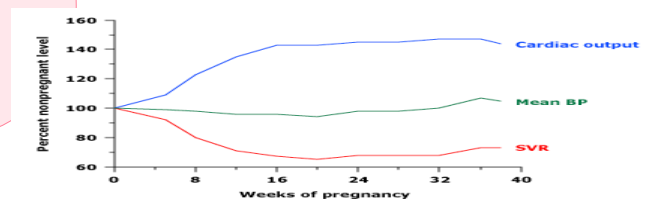
(Figure 2)

THE CARDIOVASCULAR SYSTEM IN PREGNANCY

Another important physiologic change in pregnancy is the cardiovascular system. Cardiac output is defined as heart rate times stroke volume. There is a 30-50% increase in the cardiac output that begins in the first trimester with a 20% increase in heart rate. Stroke volume is dependent upon systemic vascular resistance, which decreases in pregnancy due to the effect of progesterone on vascular resistance; this produces vasodilation. By eight weeks gestation, blood pressure has decreased by 10%. Systolic blood pressure decreases by more than 20 mmHg to 30 mmHg and diastolic blood pressure decreases by 10 to 15 mmHg (Figure 3).

This physiologic change may mask a patient with chronic hypertension who presents late in pregnancy and may initially appear normotensive. Suddenly in the third trimester this patient may now be hypertensive. However, this sudden rise in blood pressure is attributed to the return of normal, non-pregnant systemic vascular resistance by 28 weeks; this change in blood pressure continues until term.

Hemodynamic changes in normal pregnancy



UpToDate Michael R. Foley, January 2009 Figure 1. (Figure 3)

THE RESPIRATORY SYSTEM DURING PREGNANCY

The respiratory changes in pregnancy begins with mucosal edema that results in nasal stuffiness, increased

Gaylynn Thomas, RN, BSN COO

Timothy B. Waterhouse, MD, FACOG Medical Director

John Gee, RPh Pharmacy Director

OUR NURSES

- | | |
|----------------------------|-----------------------------|
| Lisa Blanchard, RN, BSN | Osia Moore, RN, BSN |
| Cheryl Bryant, RN | Cathy Olliff, RN |
| Laurie Cunningham, RN, BSN | Michelle Podhorsky, RN, BSN |
| Holly Dutton, RNC | Jennifer Price, RN, BSN |
| Pilar Erstling, RN, BSN | Jennifer Raffery, RN, BSN |
| Shquetta Flanigan, RN, BSN | Stacey Rainwater, RN, BSN |
| Katy Gerritt, RN, CCM | Cynthia Ramirez, RN |
| Geri Gigliotti, RN | Monique Rhodes, RN, BSN |
| Melodie Green, RN, MSN | Holly Rorick, RN, BSN |
| Megan Johnson, RN, BSN | Margaret Sissons, RN, BSN |
| Lydia Marsh, RN | Sue Thompson, RN, BSN |
| Melanie McClure, RN, BSN | Brenda Ventura, RN |
| Angela Miller, RN, BSN | Abbey Young, RN, BSN |
| April Moore, RN, BSN | |

OUR CLINICAL LIAISONS

- Patrick Clayton — Dallas/Fort Worth
 Lisa Hunter — Houston
 Daniel Schwartz — Houston
 Laura Vance—Dallas/Fort Worth

OUR PROGRAMS

- Pre-Term Labor Program
- HUAM & Terbutaline Pumps
- Hyperemesis Program – Reglan®/Zofran® Pumps
- In-Home IV Hydration
- Total Parenteral Nutrition (TPN)
- Infusion/Antibiotic Therapy
- In-Home Non-Stress Tests
- Pregnancy-Induced Hypertension Program
- Gestational Diabetes Teaching Program
- 17-OH Progesterone Injection Program

REFERRALS:

1-888-304-1800

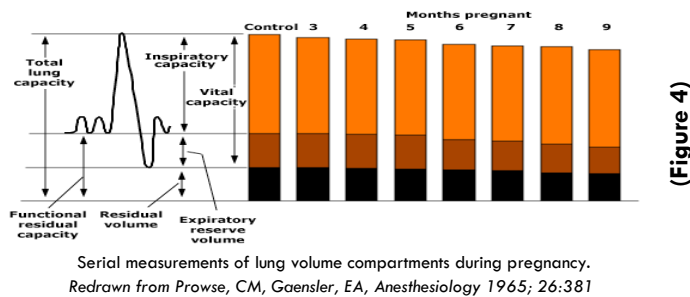
Visit Our New Website for
On-Line Referrals:

www.HCHCS.com

secretions, nosebleeds and even a chronic cough.

The pulmonary physiological changes that occur include an increase in the minute ventilation, an increase in the oxygen uptake, and an increase in the basal metabolic rate. Overall, these pregnancy changes are characterized by an increase in the tidal volume by 40% and increase in the minute volume by 40 to 50%. There is a decrease in the functional residual capacity by 20% and a decrease in the expiratory reserve and the residual volume by 15 to 20% (Figure 4).

CHANGES IN PULMONARY FUNCTION TESTS DURING PREGNANCY



(Figure 4)

MEDICAL CONDITIONS CAUSING DYSPNEA IN PREGNANCY

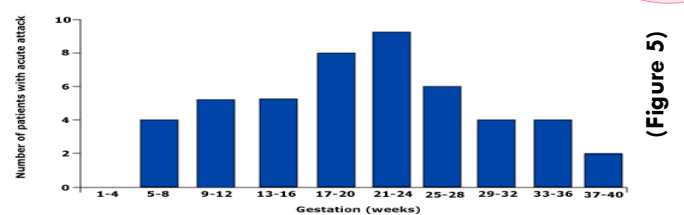
ASTHMA

Asthma is defined as a chronic long-term lung disease that results from inflammation and narrowing of the airway. Common asthma triggers or allergens include: dust, animal hair, cockroaches, mold and pollen. Of course, irritants such as cigarettes smoke and air pollution can precipitate an asthmatic reaction as well as upper airway infections, such as cold or bronchitis.

Allergens stimulate airways causing inflammation, which may lead muscles in the alveolar airway to tighten, producing more shortness of breath and increase in wheezing. This decreases the flow of air to the lungs and increases production of mucus.

Signs and symptoms of asthma include coughing (that may be worse at night), wheezing, and chest tightness (described as something sitting on the chest), and, of course, shortness of breath. Frequency of asthma attacks increase as gestational age increases and may peak in the mid-second trimester. In pregnant women with asthma, it is reported that one-third of patients improve, one-third remain the same, and one-third get worse (Figure 5).

Frequency distribution Of acute attacks during pregnancy



Asthma attacks during pregnancy were seen most frequently between weeks 17 & 24 of gestation. Data from Stenius-Aarniala, BSM, Hedman, J, Teramo, KS, Thorax 1996; 51:411.

(Figure 5)

Management of asthma includes medical therapy such as anti-inflammatory agents and bronchodilators. Anti-inflammatory agents reduce swelling and mucous production in the airways.

Bronchodilators relax the muscles that tighten around airways.

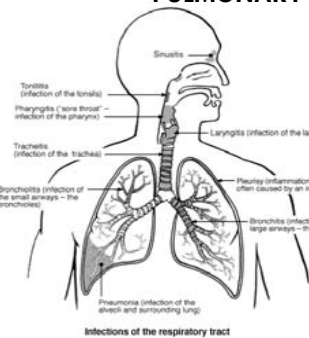
Patients should also check their peak flows once or twice daily. If there more than a 15% decrease in the peak flow, the patient should immediately contact her primary care physician or pulmonologist for medication changes or to be evaluated for other extraneous causes of her exacerbating asthma.

Many asthmatics are admitted to the emergency room and aggressive care with bronchodilators, steroids, oxygen, hydration and antibiotics may be initiated until the underlying etiology of the asthmatic flare can be determined. The NAEPP has published stepwise algorithms for the treatment of asthma and the various categories: mild, moderate and severe are addressed specifically. See Up-To-Date for specific details of the treatment of mild, moderate, and severe asthma.

PNEUMONIA

PULMONARY TREE INFECTIONS

(Figure 6)



The respiratory tree begins at the sinuses and the pathway travels to the lungs. Anyone can develop an infection anywhere along the course of the respiratory tract. The progression of infection may begin as sinusitis progress to tonsillitis or pharyngitis, next to bronchitis and ultimately

result in shortness of breath.

Pneumonia is an infection of one or both lungs caused by a bacterial, viral or fungal infection. Risk factors for pneumonia include smoking, asthma, influenza, drug abuse, and co-morbid medical conditions. Pneumonia is usually a result of the following pathogens: *Streptococcus pneumoniae* (45%), *Haemophilus influenzae* (14.3%), viral (12.6%), *Mycoplasma* (6.7%). Other causes may be due to *Chlamydia*, *Staphylococcus aureus*, *Legionella*, and fungus. In patients who are HIV positive, *Pneumocystis carinii* should be ruled out with a silver stain.

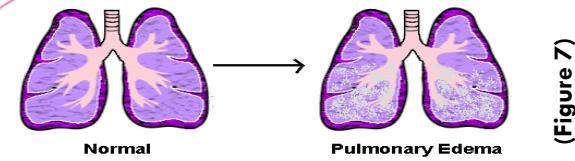
Prevention of pneumonia can start with a pneumococcal vaccine, particularly in patients who have chronic pulmonary disease, chronic renal failure, nephrotic syndrome, asplenia, sickle cell disease, diabetes and HIV infection. Influenza vaccines in these same patients, especially pregnant patients, may prevent the development of pneumonia.

Patients with pneumonia may present with fever, cough, dyspnea and abnormal breath sounds. Diagnosis of pneumonia involves a chest x-ray and sputum gram stain. An arterial blood gas may be warranted if low oxygen saturation is detected on pulse oximetry. Also, a chest x-ray may demonstrate an infiltrate in the lungs.

The American Thoracic Society has published guidelines for treatment of pneumonia. In a community-acquired pneumonia without fever or any significant respiratory compromise, a macrolide (azithromax) is the initial drug choice. However, if a coexisting illness and/or fever are present with significant cough and sputum production, then hospitalization may be required for additional IV antibiotics (usually a cephalosporin), oxygen supplementation and observation.

PULMONARY EDEMA

Pulmonary edema is an abnormal fluid accumulation or swelling in the lungs (Figure 7). Causes of pulmonary edema are cardiogenic or non-cardiogenic in nature. The cardiogenic causes in-



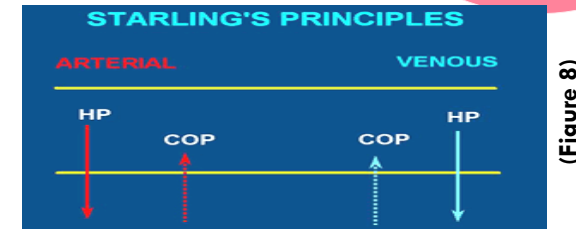
(Figure 7)

clude systolic dysfunction, diastolic dysfunction, or valvular disease.

Systolic Dysfunction is caused by decreased myocardial squeezing, such as peripartum cardiomyopathy. Diastolic dysfunction may be defined as impaired ventricular relaxation resulting in high filling pressures. An obese patient with chronic hypertension and left ventricular hypertrophy is a common clinical scenario where diastolic dysfunction may occur. Rheumatic mitral stenosis is a common example of valvular disease heart disease that produces cardiogenic pulmonary edema.

The non-cardiogenic causes of pulmonary edema are due to the changes in permeability at the capillary level; examples include: ARDS, pyelonephritis, chorioamnionitis, appendicitis, aspiration pneumonia, placental abruption, and sepsis. Iatrogenic volume overload, multiple gestation, tocolytic use, and preeclampsia can also result in pulmonary edema.

The complicated pathophysiology of pulmonary edema involves capillary hydrostatic pressure, colloid oncotic pressure, and Starling forces (Figure 8). The opposing effect of two forces, Hydrostatic Pressure (HP) and plasma Colloid Osmotic Pressure (COP) control the exchange of fluid between blood and the interstitial spaces at the capillary level. HP tends to force fluid out of the capillaries and COP tends to draw fluid into the capillaries. The Starling Equation gives net fluid flow across capillary walls as a result of the excess of capillary hydrostatic pressure over interstitial fluid hydrostatic pressure, and capillary oncotic pressure over interstitial fluid oncotic pressure -- modified by the water permeability of the capillary.



(Figure 8)

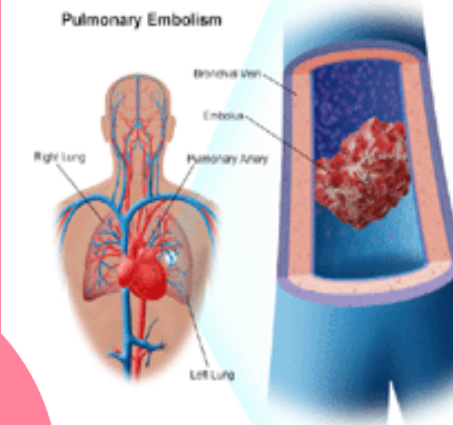
Phase I results an increase in capillary hydrostatic pressure, the pressures inside the blood vessels. Increases in capillary hydrostatic pressure cause fluid to be pushed out of the vessels into the alveolar air spaces. This fluid change interrupts the normal oxygen movement in the lungs and may result in shortness of breath. Phase II is a result of decreased colloid oncotic pressure, the force that holds fluid inside the vessels. This may be seen in a patient with preeclampsia with proteinuria, resulting in fluid being driven out of the capillaries.

Phase III is due to an increase in the interstitial colloid oncotic pressure. With a combination of these three: increased pulmonary capillary pressure, decreased intravascular colloid oncotic pressure and increased interstitial colloid oncotic pressure, there is a synergistic loss of fluid into the lungs resulting in pulmonary edema.

Symptoms of pulmonary edema include anxiety, cough, difficulty breathing, excessive sweating, feeling of "air hunger" or drowning, grunting or gurgling sounds, restlessness, wheezing, decreased level of awareness, inability to speak in full sentences, nasal flaring and orthopnea. A physical exam reveals crackles in the lungs, abnormal heart sounds, tachycardia, skin pallor (secondary to hypoxia) and tachypnea. The diagnosis of pulmonary edema is made by evaluating oxygen saturation on pulse oximetry, or

detecting hypoxia on an arterial blood gas, a chest x-ray showing pleural effusions and Kerly B signs, and an abnormal EKG or echocardiogram. Initial treatment of the pregnant patient with pulmonary edema may include positioning the patient upright to prevent vena caval compression, supplemental oxygenation, and fluid restriction. Medical treatment includes furosemide, morphine, anti-hypertensive therapy (such as a beta-blocker) and inotropes may be used as indicated.

PULMONARY EMBOLISM



Deep venous thrombosis is a medical condition where clots form in the large veins of the body, such as in the calves, thighs, pelvis, chest and arms, the right side of the heart or from the tip of a catheter. Signs and symptoms of deep venous thrombosis include leg pain, tenderness, increased warmth, swelling of a leg, cyanosis, paleness and a positive Homan's sign. A deep venous thrombosis impedes the circulation of nutrients in various organs and becomes very dangerous if it travels to the lungs. Thus, a pulmonary embolism remains a major cause of significant maternal morbidity and mortality and immediate attention is warranted if suspicion arises.

Symptoms of pulmonary embolism include acute shortness of breath, chest pain, hemoptysis, tachycardia, wheezing, leg swelling, clammy skin, skin pallor, excessive sweating, anxiety, weak pulse, light-headedness, fainting, syncope and/or fever. Risk factors for pulmonary embolism include: prolonged bedrest, surgery, obesity, pacemaker use, in-dwelling venous catheters, pregnancy, exogenous estrogen (e.g. birth control pills), family history (e.g. thrombophilia), and smoking. Interestingly, bedrest is a common prescription for various obstetrical conditions such as preterm labor and preeclampsia.

Pulmonary embolisms are diagnosed by arterial blood gas, chest x-ray, VQ scan, spiral CT using pulmonary angiogram, a D-Dimer test, ultrasound, venogram, and possibly MRI. A thrombophilia panel may help in deciphering a heritable cause for thrombosis. Pulmonary embolism is treated with anticoagulation, such as heparin, low molecular weight heparin, and warfarin (usually not used in pregnancy). Occasionally, a Greenfield filter, thrombolytic therapy, or embolectomy may be indicated in the treatment of massive pulmonary embolism. Pulmonary embolisms are usually prevented with anticoagulation, compression stockings, leg compression devices and increasing physical activity.

In summary, dyspnea or shortness of breath, has a myriad of etiologies in pregnancy. From the physiological adaptations of pregnancy to the multiple medical complications -- asthma, pneumonia, pulmonary edema, and pulmonary embolism--any could be present at any time during pregnancy. If a pregnant patient presents with dyspnea, first evaluate if this is an acute or progressive event. Begin with the basics: history and physical exam and ordering appropriate diagnostic tests. Making the correct diagnosis and ultimately prescribing the appropriate treatment should lead to a successful outcome.

REFERENCES

Creasy and Resnik's Maternal-Fetal Medicine: Principles and Practice. Robert Creasy, Robert Resnik, Jay D. Iams, et al. Sixth Edition. Saunders Elsevier 2009.
Critical Care Obstetrics. Fourth Edition. Gary A. Dildy III, Michael A. Belfort, George R. Saade, et al. Blackwell Science 2004.