Case Presentation

Dr. South-Paul: We have chosen a patient for this management conference who presented initially at another institution and was transferred for further treatment with a common complication of pregnancy. We will use this patient as an example to discuss some of the management principles that apply to a woman who presents with dyspnea during pregnancy.

A 27-year-old, gravida 2, para 1, woman was at 31 weeks' gestation and had an estimated date of confinement of Jan 1, 1988. She presented to the emergency room with increasing shortness of breath, diaphoresis, and cough productive of clear sputum that had lasted two days. She had similar symptoms and wheezing at 22 weeks' gestation. She was diagnosed as probably having Mycoplasma pneumonia and was treated with erythromycin. When evaluated on Oct 5, she was still symptomatic. A chest roentgenogram was obtained and antibiotic therapy changed to amoxicillin. One week later, a terbutaline inhaler was added for persistent wheezing after pulmonary function tests showed reactive airways disease. The patient returned to the emergency room on October 29 and 30. Therapy with amoxicillin and the terbutaline inhaler was continued and theophylline (300 mg twice daily) was added. She was admitted the next day with dyspnea, cough, wheezing, and fatigue. She had been using her inhalation bronchodilator on a frequent basis for 24 h.

Her previous pregnancy (1985) was uncomplicated, with a spontaneous vaginal delivery of a normal male infant at 38 weeks' gestation. She had had no prior surgical history and no known drug allergies or allergic history. She was a nonsmoker and denied exposure to passive smoke or other inhalational irritants.

The physical examination on admission included the following findings: she was afebrile; had tachycardia (rate, 124 bpm); her blood pressure was 118/50 mm Hg and respiratory rate 22 breaths/min at rest. She was alert and oriented with tripod posturing. The mucous membranes were slightly dry with no sinus tenderness, and the neck was supple without adenopathy. There were diffuse bilateral wheezes and retractions with dullness to percussion in both bases, but no rales were detected. Tachycardia was present with only an occasional ectopic beat and no murmurs. There was no jugular venous distention or bruits. The abdomen was soft and nontender, with a 31-cm fundus and active bowel sounds. Good capillary refill was present, with no cyanosis or clubbing of the extremities.

Initial laboratory tests revealed a hemoglobin of 9.3, hematocrit reading, 28.9; WBC count, 10,300/cu mm, with 10 percent eosinophils, and 300,000 platelets. The serum sodium level was 135 mEq/L; potassium, 4 mEq/L; chloride, 104 mEq/L; and bicarbonate, 19 mEq/L. Serum theophylline concentration was 10.6 μg/ml. Arterial blood gas (4 L oxygen by nasal cannula) analysis showed PaO₂ 60.6 mm Hg; PaCO₂ 29.5 mm Hg; pH, 7.4; and saturation, 91 percent. Urinalysis was normal, and the ECG showed sinus tachycardia.

Introduction

Dr. Tenholder: We selected this case for discussion because asthma and pneumonia are both common problems that internists and chest physicians see in evaluating patients who are pregnant and dyspneic. Internal medicine house staff are also very likely to be consulted by the obstetric service when pregnancy is complicated by many situations in which dyspnea is the chief complaint. We will discuss this patient first and follow with a review of the general topic of dyspnea in pregnancy.

The internist must ask himself three very important questions when he sees any pregnant patient with pulmonary disease. How will the pregnancy affect the disease itself? How will the disease affect the course of the pregnancy and the health of the fetus? How does management differ from that of the nonpregnant patient?

We realize that medical management in pregnancy is complicated because we must consider those drugs that cross the placenta and the effect they will have on the fetus. Substances with molecular weight >1,000 daltons do not cross the placenta. Most of the medicines we use in our general practice have a molecular weight between 250 and 400 daltons, and so they will cross the placenta. One must also consider how protein binding or the ionized fraction of a particular medicine will contribute to placental transfer. The dose and duration of the chemical is also critical in determining its teratogenic effect. If we
can employ nonpharmacologic management, this option should be considered.

The fetus is at greatest risk from most medications during the first trimester. Unfortunately, most drugs have been tested only in animal models; so the Physicians' Desk Reference will mention the safety of these drugs as suggested by these animal models. It is very important to remind ourselves of the mother that alcohol, caffeine, and tobacco are drugs. The fetus is probably actively involved by the products of cigarette smoking as opposed to passive smoking, which is receiving appropriate attention in the medical literature today.

CASE DISCUSSION

Let us now review the chest roentgenograms of this patient. On Nov 2 (Fig 1), she had bilateral infiltrates with blunting of both costophrenic angles. There was also silhouetting of the mediastinal half of the left hemidiaphragm and increased retrocardiac density. One month later (Fig 2), both infiltrates had improved.

The chest roentgenogram is necessary when the patient's symptoms are unexplained or not easily treated with safe medications. The radiation dose from a posteroanterior and lateral film is about 50 mrad to the chest and only 2 to 5 mrad to the gonads. The minimal dose for fetal teratogenic concern is much greater, at a level of 1 to 5 rad. It is standard practice to avoid x-ray examination in the first trimester, if possible, and to employ abdominal shielding during any period of gestation.

Another point of focus in this case is the derangement in the arterial blood gas values. She had a PaO₂ of 60 mm Hg (4 L oxygen by nasal cannula), PaCO₂ of 29 mm Hg, and pH of 7.41. In pregnancy, there is a mild increase in the (A-a)O₂ difference because of increased oxygen consumption and peripheral extrac-

Figure 1. PA chest film (Nov 2, 1987) shows bilateral parenchymal lower lobe infiltrates.

Figure 2. PA chest film of the same patient, one month later, shows resolution of the infiltrates.

by mother and fetus. An (A-a)O₂ gradient > 25 mm Hg is abnormal and should be investigated. This patient had an (A-a)O₂ gradient much greater than 25 mm Hg, with both pneumonia and bronchospasm contributing to the hypoxia. As early as four weeks' gestation, progesterone affects occur, resulting in maternal hyperventilation and respiratory alkalosis. The pH is usually stabilized by renal bicarbonate loss during pregnancy. This hyperventilation of pregnancy decreases fetal oxygen tension; so that any disease that causes the mother to increase her respiratory rate will have adverse effects. These include a decrease in uterine blood flow with vasoconstriction, a decrease in maternal venous return, and a decrease in umbilical blood flow. Hyperventilation and hypocapnia also shift the oxygen hemoglobin dissociation curve to the left for both mother and fetus, resulting in increased hemoglobin affinity for oxygen. Hypocapnia is accentuated by labor, and a dangerous situation exists if the pH exceeds 7.6 or the PaCO₂ falls below 15 mm Hg.

Before Dr. South-Paul reviews the patient's hospital course, I will briefly address the topic of asthma in pregnancy in a general way. Asthma is important to recognize and treat during pregnancy because the adverse effects of untreated or poorly treated disease may be life-threatening for the mother, fetus, or both. Asthma occurs in approximately 1 percent of pregnant women. About one-third of patients with a history of asthma may improve during gestation, another third tend to remain stable, while the other third have an exacerbation of asthma. Children born of asthmatic mothers have a slightly increased incidence of prematurity and low birth weight. With the large dilutional effect on blood volume during pregnancy, serum IgE levels are decreased. If you obtain an elevated

Dyspnea in Pregnancy (Tenholder; South-Paul)
level, that patient is probably having an exacerbation of the disease. Cyclic AMP peaks at weeks 14 and 34 of gestation. In this patient, the AMP peak was overshadowed by the ongoing lung infection.

HOSPITAL COURSE

Dr. South-Bulf: This patient had increasing dyspnea and was admitted to the ICU. She was treated with oxygen by face mask, parenteral aminophylline and Solu-Medrol, and terbutaline and atropine by nebulization. Within 48 h, her condition had significantly improved. Oral therapy was started, and a therapeutic theophylline concentration was maintained with theophylline (400 mg three times daily). Sinus films showed a left maxillary sinusitis which was treated with amoxicillin. Spirometric evaluation three days after admission indicated FVC, 2.36 L (59 percent predicted); FEV1, 1.94 L (61 percent predicted); and FEV1/FVC, 82 percent. Repeated spirometric study showed an increase of 300 ml in both FVC and FEV1 after two additional days of therapy. She improved steadily during her two-week hospitalization and fetal heart tones were maintained. Her medications on discharge were: prednisone (60 mg alternating with 20 mg every other day), theophylline (400 mg three times daily), amoxicillin (500 mg four times daily), terbutaline inhaler (2 puffs every 4 to 6 h), and inhaled ipratropium bromide (2 puffs every 8 h).

Dr. Tenholder: This case illustrates the use of many necessary medications for asthma in pregnancy when the patient has hypoxemia. This situation is high risk for the fetus and mother. Although these medications may have some side effects, they are needed for a patient in this kind of distress. Many reviews have been written discussing the safety of various classes of these drugs.

Since this patient had sinusitis, some would consider using antihistamines. Most are safe, except brompheniramine, which should not be used during pregnancy. Most bronchodilators are also safe, especially by inhalation. We should avoid giving epinephrine and encourage metered-dose inhaler use for effect. Since there have been some reports of fetal defects in animals with sodium cromolyn and it is more of a preventive than therapeutic drug, we should hold its use in reserve. Expectorants are rarely needed, and we must avoid any containing iodides owing to the danger of fetal goiter. Corticosteroids are controversial; however, in my opinion, this patient was so ill that their use was indicated even earlier in the course of her illness. There is a slightly increased incidence of cleft palate, but the use of corticosteroids at the lowest, symptom-free dose with inhaled steroids as a supplement is an acceptable risk when the dire consequences of refractory asthma are considered. Inhaled atropine has now proved efficacious in certain subsets of COPD and may have some beneficial effect in the treatment of refractory asthma. Atropine can cause fetal tachycardia, but inhaled ipratropium, with its decreased systemic absorption, may be a safe delivery route in pregnant patients. Antibiotics to be avoided include tetracyclines, sulfonamides, and aminoglycosides. Since the new class of quinolones blister cartilage in young children, they should be avoided pending further study. When one considers the potential risk of anaphylaxis, it is best not to attempt immunotherapy during pregnancy.

The spirometric findings recorded during the hospitalization revealed a decreased FVC and FEV1. This is not the classic obstructive pattern of severe asthma, but the pregnancy and inability to cooperate fully may have reduced the FVC out of proportion to the FEV1 impairment. Earlier outpatient spirometric study showed an obstructive pattern. Spirometric evaluation is useful in pregnancy and should have been performed during the emergency room visits. We should all have a low threshold to admit pregnant asthmatic patients, and decreasing spirometric values are an invaluable clue to deterioration.

NORMAL PHYSIOLOGY OF PREGNANCY

Before discussing the other dyspneic syndromes of pregnancy, it is helpful to review the normal physiologic changes in the mother and fetus. There are major effects on the cardiovascular system: increased intravascular volume in the mother, which dilutes laboratory values; increased cardiac output throughout gestation; and increased uterine blood flow. There are fetal responses associated with decreased PO2 gradient across the placenta: increased flow rate from fetus to placenta and the development of fetal shunts—ductus venosus, ductus arteriosus, and foramen ovale—to improve fetal oxygenation. Fetal hemoglobin (left-shifted dissociation curve) absorbs oxygen more fully and releases it more completely than adult hemoglobin. Despite all of these adaptations, the fetus has a narrow safety margin when the mother develops severe hypoxemia, since fetal vein PO2 is only about 28.5 mm Hg.

Several metabolic and hormonal changes may affect the sensation of dyspnea and affect a disease such as asthma. Human chorionic gonadotropin plays a prominent role in the first trimester and exerts a thyroid-stimulating activity. This thyroid gland stimulation may augment asthma to a minor degree. The most important factors to consider are the placenta-produced estrogens and progesterones. Progesterone is an active respiratory stimulant. Early in pregnancy, women begin to hyperventilate, and these effects continue to predominate in the second and third trimesters. Another critical factor is cortisol, with increased levels throughout pregnancy. Both transcor-

Tin and free cortisol are elevated in these patients.

Prostaglandin synthesis is an area for more investigation in all medical diseases. The F class of prostaglandins (potent bronchoconstrictors) are increased in all trimesters, especially during labor. The E class of prostaglandins (in vitro bronchodilators) are also increased during the third trimester. These two prostaglandin classes may cancel each other’s effects in most cases. Thromboxane A and histamine, another known bronchoconstrictor, are present in increased amounts during gestation. Extensive studies are being conducted in an attempt to verify a clinical utility for inhibition of these bronchoconstrictor pathways or augmentation of the coexistent bronchodilator pathways.

Although infections are not the main topic of today’s conference, several infections deserve attention when we are evaluating the dyspnea cough syndrome of pregnancy. The incidence of viral infections is increased in pregnancy.1 These patients can have tuberculosis or fungal infections,10 the most dangerous of which is coccidioidomycosis.11 We must consider the immunologic changes of the mother with decreased cellular immunity during pregnancy.13 There is an increase in the normal number of T suppressor cells formed to protect against the rejection of the fetus as a foreign body. Although there may be some decrease in delayed-type hypersensitivity, PPD skin testing is valid during pregnancy, and its sensitivity does not routinely decrease. The fetus also has a decreased immunologic defense, since only IgG can cross the placenta; so any infection in the mother could be disastrous for the fetus.

Oxygen consumption at rest in the pregnant patient is increased 15 to 20 percent over resting values in nonpregnant women. It increases throughout pregnancy, plateaus in the second and third trimesters, and increases an additional 200 ml/min during labor. Cardiac output increases by 40 percent during pregnancy, peaks in the third trimester, and declines near term. After delivery, the mother may experience dyspnea with increased venous return, causing the cardiac output to exceed baseline values by 60 percent. Figure 3 illustrates the distribution of oxygen consumption in milliliters per minute for the work of different organ systems as the pregnancy progresses through the 40-week gestation.13 It is apparent that oxygen consumed for the work of breathing has a proportionately small and smooth increase during gestation. If the mother has dyspnea and hypoxia from pulmonary disease, the cost from work of breathing may steal from other organ systems such as the fetus, placenta, uterus, and kidneys. The uteroplacental circulation is a low-pressure, highly vascular system, well developed by the 12th week. Its blood flow is 20 percent of the cardiac output by week 26. A healthy child depends on us to keep any maternal oxygen debt to a minimum by supplementing maternal oxygen whenever hypoxia becomes manifest.

Some amount of breathlessness is a normal physiologic response in pregnant patients.14 It is so common that 60 to 70 percent of the mothers will experience the sensation in the first two trimesters. Why does this dyspnea occur? Is it an increased mechanical load or an altered respiratory sensation secondary to increased progesterone? The mechanical load is increased by chest wall distortion from the gravid uterus, weight gain, and increased ventilatory rate. Alteration in lung compliance is not thought to be a contributing factor.1 At least one study showed no change in lung compliance between the third trimester and postpartum.15 It is probable that both altered sensation and changes in chest cage conformation play some role in this physiologic dyspnea.

Alterations in the mother’s pattern of breathing, changes in ribcage displacement, and increased laxity of abdominal musculature all affect ventilatory reserve.16 We all use two methods to increase minute ventilation, the bucket-handle forces generated by our chest cage muscles and the pump-handle forces of the diaphragm. The pregnant patient is able to increase her tidal volume by both of these mechanisms, because there is actually no diaphragmatic dysfunction incurred by a normal pregnancy. Abdominal volume displacement is not changed during pregnancy, and diaphragm contraction and movement is greater in pregnancy than postpartum.16 Even during labor with lightening (fetal head moving into the pelvis), the diaphragm assumes a more normal configuration and functions as well as it does in the nonpregnant state.
Another interesting area to discuss in relation to dyspnea are the alterations in pulmonary function that occur. Some obstetric texts suggest that exercise in pregnant patients is limited by a decreased ventilatory reserve. Actually, the ventilatory system has a remarkable reserve available for the mother which can be augmented by a well-planned exercise program. Although minute ventilation increases 40 to 50 percent during a normal gestation, the mother can increase her tidal volume by 450 to 600 ml to compensate. This results in a vital capacity and respiratory frequency that should remain virtually unchanged. I previously mentioned that a respiratory rate > 18 breaths/min at rest should be a warning sign for pathology superimposed on the normal "dyspnea of pregnancy."

Functional residual capacity is decreased in the last half of gestation by the increasing abdominal contents. Both the expiratory reserve and residual volume are decreased, but the total lung capacity remains essentially unchanged. Diffusion is likewise unchanged or increases with the increase in circulating blood volume. The closing volume has a linear increase with advancing stages of gestation and can actually exceed the functional residual capacity near term. This may result in some basal ventilation perfusion imbalance and mild hypoxemia late in pregnancy. With the marked bronchodiatory effect of progesterone, there is a decreased airway resistance and increased conductance during a normal pregnancy.

**Specific Dyspneic Syndromes**

With that background, we can now discuss some specific disease states in which dyspnea and hypoxia may be the predominant symptoms and signs. Fortunately, many pulmonary diseases are uncommon in the child-bearing years; but this may change as many women are having children later in life. We may see more pregnancies complicated by COPD. A 37-year old patient with α1-antitrypsin deficiency and an FEV1 of .96 L has been reported. She had an uncomplicated pregnancy and a normal term infant. In contrast, a 40-year-old patient with a long smoking history, FEV1 of .67 L, hypoxemia (PaO2 54 mm Hg), and hypercarbia (PaCO2 49 mm Hg) delivered a premature 32-week normal infant, but the mother required mechanical ventilation postpartum. Even though COPD is a rare problem, it might be prudent to consider cesarean section as an alternative for delivery when the mother's condition is optimized by appropriate medical treatment.

Management of the pregnant patient with cystic fibrosis presents even more of a challenge. There is an 11 percent perinatal mortality and a 12 percent maternal mortality in the first six months postpartum. However, 70 percent of pregnant cystic fibrosis patients do quite well and have good fetal outcome. The others have at least some decrease in pulmonary function that fails to return to baseline postpartum. Of course, all infants born of cystic fibrosis parents are obligate heterozygotes. It is critical to counsel the mother about these factors and to provide a complete quantitative nutritional and pulmonary function status before you undertake such counseling.

I will use sarcoidosis as a prototype of the restrictive lung diseases that can complicate pregnancy with added dyspnea. It is interesting that most sarcoid patients have some amelioration of symptoms during pregnancy. This may occur from the increased free cortisol circulating during gestation. However, postpartum exacerbations of sarcoidosis will occur. With all the restrictive lung diseases and the neuromuscular diseases, a vital capacity less than 1 L is probably a critical point for consideration of therapeutic abortion and or planned cesarean section.

Primary pulmonary hypertension is a disease of young women, usually in their child-bearing years. It carries a very high mortality rate. Patients present with severe dyspnea and syncope, and it has occurred during pregnancy. It is unknown if this is just an association or if pregnancy exacerbates the disease. If one suspects primary pulmonary hypertension, it is critical to eliminate other catastrophic events in the differential diagnosis that can complicate pregnancy such as amniotic fluid embolism, trophoblastic or air embolism, pulmonary thromboembolism, pulmonary veno-occlusive disease, and Eisenmenger's syndrome.

Fortunately, pulmonary thromboembolism is not common during pregnancy, since it is such a major management problem when it does occur. Deep venous thrombosis occurs in about 2/1,000 pregnancies and superficial thrombophlebitis in 12/1,000. The risk for pulmonary embolism is greatest in the first month postpartum, especially one to three days after delivery. The coagulation factor concentrations, especially 7, 9, and 10, are elevated during the entire pregnancy. We must consider all the potential risk benefit decisions involved in performing perfusion scans or angiography in these patients. Perfusion scans can be done with one-quarter to one-half the routine dose to reduce radiation risk. Ventilation scanning can be reserved only for the evaluation of positive perfusion scans. Iodine-labeled fibrinogen studies are contraindicated (fetal goiter effect).

The use of heparin and warfarin (Coumadin) is hazardous, and anticoagulation is a difficult task. Coumadin should not be used at all during the first trimester because it causes fetal embryopathy. It also can cause a fetal hemorrhage syndrome at birth, with an 18 percent perinatal mortality. Coumadin should be avoided in nursing mothers. Heparin, on the other hand, does not cross the placenta and does not affect
the fetus. However, there is an increase in maternal hemorrhage at birth with heparinization.

How, then, can we anticoagulate when pulmonary embolism has been well documented? I will outline a course, but this is subject to change as we improve our methods or develop newer anticoagulants. Always begin with IV heparin and you can switch to maintenance Coumadin as long as the first trimester has passed. Discontinue Coumadin at week 37 and give the patient IV heparin until the time of delivery. Discontinue heparin at the onset of labor until after delivery. Oral Coumadin, minimum six weeks, can be used for the duration of treatment. If the patient has premature labor while receiving Coumadin, you must give fresh frozen plasma for reversal. An attractive alternative plan is to use long-term, self-administered, subcutaneous heparin (7,500 to 10,000 units every 12 h).

Amniotic fluid embolism is another catastrophic event that internists are asked to help diagnose and manage. With its dramatic onset of dyspnea, hypoxia, hypotension, and disseminated intravascular coagulation in the first 48 h postpartum, it is difficult to distinguish from thromboembolism. It is an infrequent event, but accounts for up to 10 percent of maternal deaths. The mortality rate from amniotic fluid embolism can reach 80 percent. There are new diagnostic methods for this syndrome. You can withdraw blood from the pulmonary artery through a Swan-Ganz catheter. On a Papanicolaou smear of the buffy coat, you can find anucleate fetal squamous cells, lanugo hairs, fat, and mucin and confirm the diagnosis. Tumultuous labor, use of uterine stimulants, meconium staining, advanced maternal age, multiparity, and intruterine fetal death or tumor are all risk factors for the syndrome. The differential is expansive and includes air embolism, thromboembolism, myocardial infarction, peripartum cardiomyopathy, hemorrhagic or septic shock, anaphylaxis, and even severe pre-eclampsia can mimic amniotic fluid embolism. With current excellent obstetric care, Mendelson's syndrome from acute aspiration is no longer a common occurrence that can be confused with this syndrome.

Postpartum pleural effusions can be a common consult to the internal medicine service, but these may have nothing at all to do with dyspnea. Postpartum effusions are common; at least one-half to two-thirds of patients have pleural effusion 24 h postpartum. On lateral decubitus films they are small, <3 mm in half of the patients, but may be moderate and >3 mm in the other half. Most (75 percent) are bilateral and self-limited. They are caused by the increased blood volume, decreased colloid oncotic pressure, and the Valsalva maneuver used by the mother during labor. One should avoid performing invasive procedures to evaluate these effusions in this setting.

**EXERCISE IN PREGNANCY**

Dr. South-Paul and I have been particularly interested in evaluating exercise as a way to decrease the sensation of dyspnea during pregnancy. By comparing normal sedentary pregnant control subjects with pregnant women enrolled in a supervised moderate exercise program, we documented an increase in the recruited tidal volume in the exercised group. They also had increased oxygen consumption without a disproportionate increase in minute ventilation at equivalent work. The exercised subjects were able to increase tidal volume and oxygen consumption at both low and maximum work levels. It is also known that trained athletes who become pregnant will have severe detraining effects if they do not continue some type of exercise. I would now like Dr. South-Paul to elaborate on the beneficial influence exercise has on the dyspnea of pregnancy.

**Dr. South-Paul:** We have already mentioned that 70 percent of pregnant women exhibit some degree of breathlessness. With more and more women interested in maintaining physical activity during pregnancy, it is important for clinicians to recognize the physiologic adaptations that occur with exercise in the pregnant patient. The question arises as to the risks of maintaining activity vs the benefits accrued from such activity. Some studies have focused on the issue of exercise in pregnancy. One of the original studies, in the mid-1970s, evaluated a regular exercise program in a primiparous population. They found that women who train during pregnancy can exhibit 18 percent more physical work capacity than those not trained. Another study demonstrated certain characteristics of good aerobic fitness in an exercised pregnant group. They had lower resting and exercise heart rates, decreased body fat, and lower diastolic blood pressures. This has implications in terms of some complications of pregnancy such as preecclampsia. Possibly, in a trained population, we will be able to continue to show lower diastolic blood pressure and less risk of that particular condition. Figure 3, discussed earlier, shows the oxygen consumption required by different organs. If we can minimize body fat in women as they progress through gestation, we may be able to ensure distribution of oxygen to more vital organs. In a larger study, favorable outcomes were again realized in those groups who exercised regularly, especially those who maintained a relatively high level of exercise. Hospitalization was shorter. The incidence of cesarean section was lower, and fetal outcomes (Apgar scores and weight) were better in the exercising group. Is this beneficial effect seen in women who are not trained athletes, but only do certain recreational activities? It appears so. There is no increase in
neonatal morbidity or obstetric complications. The women's perception of well-being, whether during exercise or even when not exercising, is significantly improved by participation in a regular training program.

Are there any contraindications to physical activity during pregnancy? Several have been outlined by the American College of Obstetricians and Gynecologists and divided into relative and absolute contraindications. Relative refers to those which, under controlled conditions, allow a woman to maintain a moderate program of physical activity. Medical conditions such as hypertension, anemia or blood disorder, thyroid disease, diabetes, and cardiac arrhythmias fall into this category. Others are related to the pregnancy itself, such as a history of precipitous labor in a prior pregnancy, history of intrauterine growth retardation, bleeding during the present pregnancy, or a breech presentation in the last trimester. Other constitutional problems such as excessive obesity or anorexia or a history of an extremely sedentary lifestyle could all be considered relative contraindications for increasing activity level during pregnancy. Each patient must be evaluated on an individual basis when any of these relative factors is present alone or in combination with others.

The absolute contraindications to physical activity include: more than three spontaneous abortions or ruptured membranes; a history of premature labor, especially in the current pregnancy; diagnosed multiple gestation; and the presence of an incompetent cervix, even if the woman has a cerclage in place. The presence of bleeding or a diagnosed placenta previa that has not bled and diagnosed unmanaged cardiac disease greater than New York Heart Association class 2 also prohibit participation in exercise programs.

So what advice can we give the mother who asks, "I want to feel better. How can I do it?" Specific program guidelines can be recommended for her. The manner of exercise and the mode in which she performs physical activity should be outlined. The frequency should be at least three times per week. The duration should be no greater than 30 minutes. The intensity should be such that she can maintain a conversation while she is participating in her activity. If her breathlessness is such that she cannot comfortably say at least three words in a row, the woman is exercising to excess.

We must encourage the woman to use a warm-up and a cool-down period to minimize dizziness and syncope after any activity. The woman should also limit exercise in the heat, use low-intensity programs, especially the nonathlete, and avoid exercising to exhaustion or chronic fatigue. Once the woman has been delivered, she can resume her level of activity anywhere from 2 to 6 weeks postpartum. Breast feeding is not a contraindication to exercise. With these guidelines, a woman can participate in activity, whether organized in a supervised program or on her own, can participate on a regular basis, and can maintain almost all exercises that she enjoyed prior to conception. These women should benefit from an overall sense of well being and a decrease in their sensation of dyspnea.

Question, Dr. Thomas Dillard: What are the most common causes of respiratory failure during pregnancy?

Answer: Dr. Tenholder: Acute respiratory distress syndrome or respiratory failure postpartum can be caused by all of the diseases that we mentioned that mimic amniotic fluid embolism in this setting. The incidence of pulmonary edema after pregnancy is rare and its occurrence should be viewed as a serious development that mandates a search for all of these possibilities. We should also emphasize that patients with underlying pulmonary disease can often go unrecognized. We have recently consulted on a patient whose pregnancy was complicated by pulmonary alveolar proteinosis. Since most patients have not had a chest film during pregnancy, we should carefully review the medical history for a missed pulmonary disorder whenever an ARDS or interstitial pattern appears postpartum.

References
5. Greenberger PA. Pregnancy and asthma. Chest 1985; 87 (suppl):85-87
Weekend Refresher in Pulmonary Medicine

The University of Illinois at Chicago College of Medicine will present this course at the Eagle Ridge Inn and Resort, Galena, Illinois, October 5-7. For information, contact the Conference Registrar (312:995-5225).

Applied Ultrasound: Accent on Doppler

The Department of Radiology, University of Alabama, Birmingham School of Medicine will present this course at the Wyndowy Hotel Galleria Plaza, Birmingham, October 13-15. For information, contact Ms. Dawne Ryals, PO Box 1925, Roswell, Norcross, GA 30077-1925 (404:641-9773).