

# From dyspnoea to respiratory failure in pregnancy

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## Introduction

Non-pregnancy related infections (mainly deaths in HIV infected pregnant women complicated by tuberculosis and pneumonia) contributed 40.5% of maternal deaths in 2010-2012, most of which are highly preventable. Two thirds of the women with AIDS had respiratory complications including tuberculosis (26.9%), *Pneumocystis carinii* pneumonia (13.3%) and other non-specified pneumonia (26.7%). Early recognition and intervention of patients with these respiratory complications is important in the reduction of maternal mortality (Saving mothers report 2010-2012). Despite often ignored chronic respiratory symptomatology these women commonly present in an acute state with dyspnoea which is a subjective experience of breathing discomfort that is comprised of qualitatively distinct sensations that vary in intensity. The experience derives from interactions among multiple physiological, psychological, social, and environmental factors, and may induce secondary physiological and behavioural responses. Dyspnoea is a common symptom that afflicts millions of patients with pulmonary disease and it may also be the primary manifestation of myocardial ischaemia or dysfunction.

## Physiological changes in pregnancy

There are several important physiological principles that need to be known when evaluating the pregnant woman with dyspnoea:

**Respiratory changes** - the normal respiratory tract changes during pregnancy result in a compensated respiratory alkalosis, with a higher  $PO_2$  and a lower  $PCO_2$  than in the non-pregnant state. The lower  $PCO_2$  is thought to provide a diffusion gradient that may facilitate the foetus' ability to eliminate waste from aerobic metabolism.

**Elevation of the diaphragm** - Although the progressively enlarging uterus causes diaphragm position to rise up to ~

4 cm above its usual resting position, diaphragmatic excursion during respiration is not impaired since chest wall mobility increases and there is flaring of the ribs.<sup>1</sup>

**Decreased FRC and stable FEV1** - Functional residual capacity (FRC) decreases approximately 20 percent during the latter half of pregnancy, due to a decrease in both expiratory reserve volume (ERV) and residual volume (RV).<sup>2,3</sup> There are variable and generally minor changes in vital capacity (VC) and total lung capacity (TLC) and these changes are not likely to be clinically significant.

**Airway function** is preserved during pregnancy, as reflected by an unchanged forced expiratory volume in one second (FEV1) and an unchanged FEV1/FVC ratio. Minor changes, which are of little clinical importance, have been described in diffusing capacity for carbon monoxide (DLCO) which includes an increase during the first trimester followed by a decrease until 24 to 27 weeks of gestation.<sup>4</sup>

**Increased ventilation and respiratory drive** - There is up to 40% increase in tidal volume which yields up to 50% increase in resting minute ventilation at term. Respiratory rate remains essentially unchanged.<sup>5</sup>

**Increased levels of progesterone** during pregnancy are thought to be responsible for the rise in ventilation above that explained by the enhanced metabolic requirements. Progesterone is a known stimulant of respiration and respiratory drive, and its levels gradually rise from approximately 25 ng/mL at six weeks to 150 ng/mL at term.<sup>6</sup>

**Respiratory alkalosis and increased arterial  $O_2$  tension** - due to progesterone-induced increase in alveolar ventilation, arterial  $PCO_2$  falls to a plateau of 27 to 32 mmHg during pregnancy. This respiratory alkalosis is followed by compensatory renal excretion of bicarbonate, so that the resultant arterial pH is normal to slightly alkalotic (usually between 7.40 to 7.45).<sup>7</sup> The maternal

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arterial oxygen tension (PaO<sub>2</sub>) is generally increased because of hyperventilation, ranging from 106 to 108 mmHg in the first trimester to 101 to 104 mmHg in the third trimester.<sup>8,9</sup>

### Etiological factors

Possible causes of acute respiratory distress include:

- Asthma
- Pulmonary embolus
- Pulmonary oedema
- Pneumonia
- Heart failure
- Bronchitis
- Chest wall trauma
- Exercise
- Progesterone effect
- Anxiety
- Amniotic fluid embolus
- Restriction of chest cavity due to uterine growth
- Severe anaemia
- Viral illness

**Asthma** is a chronic, ongoing lung disease marked by acute flare-ups or attacks of difficulty with breathing. It is a common disease that can present at any age but most often occurs during childhood and can continue into adulthood. It is the most common chronic respiratory illness seen during pregnancy. Its course in pregnancy can be variable. Common causes of asthma exacerbation in pregnancy include noncompliance with medications, sinusitis and gastroesophageal reflux disease. More so, the effects of asthma itself on pregnancy ranges from intrauterine growth restriction to preterm labour with associated increase in perinatal morbidity and mortality. The management of asthma in pregnancy is basically unchanged from that of the non-pregnant individual. Inhaled beta agonists, inhaled steroids, systemic steroids (both oral and intravenous) and cromolyn sodium have all been used safely in pregnancy.<sup>10</sup>

**Pulmonary oedema** - Pregnant women are predisposed toward developing pulmonary edema because of the decrease in serum oncotic pressure that normally occurs in pregnancy. This decrease in serum oncotic pressure occurs because the 50% increase in blood volume that occurs in pregnancy is predominantly achieved through an increase in plasma free water. This has a dilutional effect on the blood and is manifested by the significant drop in serum albumin levels that is seen during gestation. The conditions that can precipitate pulmonary oedema in pregnant women include pyelonephritis, tocolytics, cardiac disease and preeclampsia. Although the pulmonary oedema seen in pregnancy can be very severe in its clinical and radiologic presentation, it is important to remember that it often responds dramatically to withdrawal of the underlying cause combined with use of diuretics.<sup>11</sup>

**Pulmonary embolism** - Venous thromboembolism is a leading cause for maternal mortality. Pregnancy is an important risk factor for thromboembolic disease in young

women. Unfortunately, its presentation is often subtle in pregnant women. This is likely because pregnant women are generally healthy with greater cardiopulmonary reserve than the general medical population, which tends to be older with comorbid medical conditions. As well, symptoms that suggest thromboembolism such as dyspnoea, palpitations and leg oedema are often part of normal pregnancy. A high degree of suspicion about pulmonary embolism is required in pregnancy. The diagnostic evaluation and investigation for pulmonary embolism in pregnancy are the same as it is for the non-pregnant individual. Use of appropriate diagnostics in a timely manner so that prompt anticoagulation therapy can be initiated is important. Chest x-rays, ventilation perfusion scans and pulmonary angiograms all involve radiation levels that are well below accepted upper limits for the gravid woman.<sup>12</sup>

**Amniotic fluid embolus** - A dreaded cause of shortness of breath that is unique to pregnancy is amniotic fluid embolism. It is rare and often fatal. It has been reported to occur at any time during the third trimester, but usually occurs at the time of delivery. It is characterized by a rapid and progressive respiratory failure that is associated with hemodynamic instability and the rapid onset of disseminated intravascular coagulation. There is reason to believe that at least a portion of the manifestation of this condition is related to some allergic or immunologic response to the fetal tissue present in the amniotic fluid. This is because some women who have had pulmonary artery catheters inserted during labour have been found to have fetal tissue in their circulation without any of the clinical manifestations of amniotic fluid embolism. It is unclear why some women present with such dramatic symptoms while others are asymptomatic. Treatment is supportive. Blood products and factor replacements should be given if the patient is bleeding. Mechanical ventilation and hemodynamic support with cardiac inotropes and vasopressors is often also necessary.

**Cardiac diseases** - heart disease can present for the first time during pregnancy as unexplained dyspnoea because of the increased cardiac work associated with pregnancy. Typically, this will occur at 24 to 28 weeks gestation when blood volume reaches its maximum. Classically mitral stenosis often presents for the first time in young, reproductive age women. Up to 25% of cases of mitral stenosis will present for the first time during pregnancy.

**Dyspnoea of pregnancy** is a specific entity that is quite common and characterized by an often dramatic increase in a patient's perceived shortness of breath during pregnancy. It usually begins sometime in the middle of gestation and can be very frightening for the patient. Investigations of these patients reveal a completely normal physical examination, oxygenation, chest x-ray, and pulmonary function testing. Once underlying pathology has been ruled out, reassurance is the mainstay of treatment. Surprisingly, dyspnoea of pregnancy may occur in women who have had other pregnancies not complicated by this clinical entity. The explanation for this

condition is not established, but is believed to be some central (i.e., CNS) misperception of the normal increase in minute ventilation that occurs in pregnancy

**Pneumonia** is a general term for a wide variety of conditions that cause an inflammation of the lungs. It is most often caused by a bacterial infection (bacterial pneumonia) or a viral infection (viral pneumonia). However, pneumonia can also be caused by a fungal infection, yeast infection, trauma, or from inflammation of the lungs due to exposure to toxic substances, such as poisonous gases. Community-acquired pneumonia is a relatively common cause of acute respiratory failure in pregnant patients.<sup>13-15</sup> In those affected by HIV, TB and PCP are the most common causes of respiratory deaths in South Africa (Saving mothers report 2010-2012). The reduction in cell-mediated immunity that is associated with pregnancy (especially during the third trimester) also places women at increased risk for severe pneumonia and disseminated disease from atypical pathogens, such as herpesvirus, varicella, and coccidioidomycosis.<sup>16,17</sup> The clinical features, diagnosis, and treatment of community-acquired pneumonia are the same for pregnant and nonpregnant patients.

Clinical features include fever, a cough productive of mucopurulent sputum, dyspnea, tachycardia, and tachypnea. Many patients also have hypoxemia, chills, rigors, pleuritic chest pain, gastrointestinal symptoms (nausea, vomiting, diarrhoea), and mental status changes. Auscultation may reveal evidence of consolidation. Gram stain of the sputum may reveal abundant polymorphonuclear leukocytes, while a culture of the sputum may grow the pathogen. The major blood test abnormality is leukocytosis with a leftward shift. Urinary antigen testing may also be a useful diagnostic tool for specific pathogens such as *Streptococcus pneumoniae* and *Legionella pneumophila* serotype-1. Diagnosis requires a chest radiograph that reveals focal or multifocal airspace disease, as well as supportive clinical and microbiological features. Antibiotic therapy is necessary to treat community-acquired pneumonia. It is generally initiated empirically and then tailored to the microbiological results. Adjunctive therapies may include supplemental oxygen and suctioning. Mechanical ventilation may be necessary.

**Aspiration pneumonitis** follows aspiration of gastric contents (pregnancy is associated with delayed gastric emptying and more relaxed oesophageal sphincters). This is more increased with patients who have decreased level of consciousness/comatose, anaesthesia (especially general anaesthesia), lying supine, severe vomiting, etc. In its severe form (Mendelson's syndrome) when highly acidic liquid is aspirated, patient will be tachypnoeic, tachycardic, cyanosed, hypoxic, hypercapnic, acidotic and hypotensive. Radiological changes are usually delayed and variable.

**Acute respiratory distress syndrome (ARDS)** - is defined as a syndrome of acute and persistent lung inflammation with increased vascular permeability. ARDS is

characterized by four features:

- Timing – Develops within one week of a known clinical insult or new or worsening respiratory symptoms.
- Radiographic appearance – Bilateral opacities not fully explained by effusions, lobar/lung collapse, or nodules.
- Pulmonary oedema – Respiratory failure not fully explained by cardiac failure or fluid overload; need objective assessment (eg, echocardiography) to exclude hydrostatic oedema if no risk factor is present.
- Compromised oxygenation – Mild: 200 mmHg  $< PaO_2/FiO_2 \leq 300$  mmHg with PEEP or CPAP  $\geq 5$  cmH<sub>2</sub>O; Moderate: 100 mmHg  $< PaO_2/FiO_2 \leq 300$  mmHg with PEEP  $\geq 5$  cmH<sub>2</sub>O; Severe:  $PaO_2/FiO_2 \leq 100$  mmHg with PEEP  $\geq 5$  cmH<sub>2</sub>O.

### Management of acute respiratory distress

**INITIAL MANAGEMENT** - is the same regardless of the cause of the acute respiratory failure (Inadequate gas exchange by the respiratory system accompanied by inadequate tissue oxygenation and hypercapnia). It is directed at the stabilization of the acute state of patient, therefore rapid systemic assessment (Circulation, airway and breathing) is mandatory with goal directed management.

Supplemental oxygen should be administered and preferred method of administering the oxygen depends upon the severity of the hypoxemia. For patients with mild hypoxemia, administration via nasal cannula may be sufficient. More severe hypoxemia generally requires administration via a facemask or a nonrebreather mask. Oxygenation should be monitored continuously by pulse oximetry. A reasonable goal is to maintain the oxyhemoglobin saturation  $\geq 95$  percent to optimize the fetal oxygen content. Adequate fetal oxygenation requires a maternal arterial oxygen tension ( $PaO_2$ )  $> 70$  mmHg, which corresponds to an oxyhemoglobin saturation of 95 percent.<sup>18</sup>

Mechanical ventilation may be required. The decision about whether to intubate a patient should be considered in the context of pregnancy. Indications of ventilation include any event that might be associated with failure to ventilate which is characterized by hypercapnia and hypoxaemia. These include a high block from neuraxial anaesthetic, local anesthetic/other drugs systemic toxicity, respiratory and neurologic emergencies and maternal cardiac arrest.

Intubation may be difficult during pregnancy and the peripartum period due to upper airway oedema and diminished airway calibre, especially late in pregnancy, therefore difficult airway management protocols should be followed.

An arterial blood analysis (ABG) and chest radiograph should be obtained after initial stabilization. The ABG identifies and quantitates the severity of any ventilatory abnormalities. It also guides ventilator adjustments in mechanically ventilated patients.

Goals for ventilation are different in pregnant patients. The target  $PaCO_2$  is 30 to 32 mmHg, since this is the normal level during pregnancy. Marked respiratory alkalosis should be avoided because it may decrease uterine blood flow. Maternal permissive hypercapnia may

also be deleterious to the fetus because of resultant fetal respiratory acidosis.<sup>19</sup>

The chest radiograph is used to narrow the differential diagnosis of the respiratory failure and, among intubated patients, to confirm that the endotracheal tube is in the correct position.

### EVALUATION OF PREGNANT WOMEN WITH DYSPNEA

When a pregnant woman complains of dyspnoea, distinguishing between underlying disease and progesterone-induced hyperventilation can be a difficult diagnostic problem.<sup>20-23</sup> There are relatively few pregnancy-specific causes of respiratory failure. These include amniotic fluid embolism and pulmonary oedema secondary to tocolytics, preeclampsia/eclampsia, or pregnancy-associated cardiomyopathy (peripartum cardiomyopathy).

#### History and physical examination

Is dyspnea of acute or gradual onset? - Physiologic dyspnea during pregnancy has a gradual onset. In contrast, pulmonary embolism is characterized by the sudden onset of dyspnea. Other common findings suggesting pulmonary embolism are tachypnea, pleuritic chest pain, and hemoptysis, which do not occur with normal dyspnea of pregnancy. Heart rate may increase above the elevated baseline rate of normal pregnancy.

Acute dyspnea from upper airway obstruction is a symptom of anaphylaxis, and is often accompanied by other symptoms of an acute IgE mediated hypersensitivity reaction (eg, flushing, itching, urticaria, angioedema, tachycardia, hypotension)

Is cough or wheezing present? - Physiological dyspnoea of pregnancy is not associated with cough or wheezing.

Acute cough is most commonly due to an acute respiratory tract infection. Other considerations include an acute exacerbation of underlying chronic pulmonary disease, pneumonia, and pulmonary embolism. Cough that has been present longer than three weeks is either sub-acute or chronic (TB should be excluded).

Cough and wheezing are also common symptoms of asthma and cardiac diseases with pulmonary venous hypertension. A history of asthma symptoms, especially antedating pregnancy, suggests asthma as the cause of dyspnoea during pregnancy; although occasional patients present with new onset asthma while pregnant. Evidence of airflow obstruction on pulmonary function testing supports the diagnosis of asthma.

Is the chest clear on auscultation? - The lungs should be clear in women with physiological dyspnoea. Crackles are indicative of abnormalities affecting the distal lung parenchyma, such as interstitial pulmonary oedema from left ventricular failure. Heart failure affecting right ventricular filling pressures is associated with peripheral oedema and prominent neck veins.

Pulmonary oedema represents a final common pathway of several complications of pregnancy and the peripartum period: this include severe preeclampsia/eclampsia, tocolytic-induced pulmonary

oedema, cardiac disease (which may be acquired or congenital, pre-existing, or related to pregnancy, and amniotic fluid embolism.

Focal crackles with or without consolidation may also be suggestive of pneumonia. Associated common signs and symptoms include cough, fever, pleuritic chest pain, dyspnoea, and sputum production

Are other symptoms present? - Physiologic dyspnoea is not accompanied by pain or other symptoms. Thoracic tumours and pulmonary emboli may present with dyspnoea and chest pain, haemoptysis, cough, or wheezing.<sup>24</sup> Dyspnoea accompanied by fever and cough suggests an infectious process (eg, bronchitis, pneumonia).

Is onset early in gestation or near term? - Physiologic dyspnoea typically begins in the first or second trimester. Women with peripartum cardiomyopathy commonly complain of dyspnoea, but onset is rarely before 36 weeks of gestation, and affected patients usually present during the first four to five months postpartum.

Other frequent symptoms include cough, orthopnoea, paroxysmal nocturnal dyspnoea, and haemoptysis. Nonspecific fatigue, chest discomfort, or abdominal pain may confuse the initial evaluation due to the occurrence of similar symptoms during normal pregnancy.

#### Laboratory and imaging tests

The history and physical examination lead to accurate diagnoses in most patients with dyspnoea.

*Chest radiography and pulmonary function testing* should be the first tests obtained in the majority of cases in which additional information is required.

The amount of radiation exposure to the foetus from a chest radiograph is extremely small and not known to have adverse foetal consequences.<sup>25</sup> Nevertheless, chest radiography during pregnancy should be done only when there is a good medical reason, and appropriate shielding of the mother's abdomen should be used. A chest radiograph should be obtained in patients with suspected pneumonia or lung lesions.

*Radionuclide lung scanning*, which requires use of technetium-labeled macro aggregates of albumin (for the perfusion scan) and inhaled xenon (for the ventilation scan), is also thought to pose little risk to the foetus, therefore should only be used when there is a serious consideration of pulmonary embolic disease.

*Brain natriuretic peptide (BNP) levels* may be useful in pregnant women with a suspected cardiac cause of dyspnoea. Levels are not affected by pregnancy, with typical values of <50 pg/mL and median levels of approximately 20 pg/mL throughout pregnancy.<sup>26</sup> Thus, BNP levels may be useful in the patient with suspected ventricular dysfunction as the cause of dyspnoea.

*Spirometry* is useful for diagnosis of asthma and can be performed safely during pregnancy.

#### Ongoing management

Once the patient has been stabilized and the cause of the acute respiratory failure has been determined, ongoing management includes supportive care and treatment of the aetiology.

Supportive care includes oxygenation and ventilation (i.e. supplemental oxygen or mechanical ventilation), sedation, pain control, hemodynamic support (i.e. vasopressors), monitoring, volume management (i.e. intravenous fluids or diuretics), nutritional support, stress ulcer prophylaxis, and venous thromboembolism prophylaxis.

Delivery of the fetus should be individualized and is guided by gestational age (prematurity is associated with high neonatal ICU admissions and chronic lung diseases), fetal status at admission (alive/dead and viability) and maternal diagnosis and prognosis

Delivery is often deferred until the patient is out of danger unless it impedes ventilation.

### Summary and Conclusion

Patients with acute respiratory failure generally present with respiratory distress.

When a pregnant woman complains of dyspnoea, the clinician should determine whether her symptoms are due to an underlying disease or physiologic progesterone-induced hyperventilation

Two-thirds of pregnant women experience a sensation of dyspnoea (often described as "air hunger"), which starts gradually during the first or second trimester.

Physiologic dyspnoea of pregnancy is of gradual onset; sudden onset or presence of cough, wheezing, crackles, chest pain, fever, or haemoptysis suggests a pathologic process that requires further evaluation.

Acute respiratory failure during pregnancy or the peripartum period may be due to a conventional respiratory insult or a pregnancy-specific disorder. The usual differential diagnosis includes pulmonary oedema, community-acquired pneumonia, aspiration pneumonitis, pulmonary embolism, asthma exacerbation, amniotic fluid embolism, and venous air embolism. Pulmonary oedema is most often due to the treatment of preterm labour, heart failure, severe preeclampsia or eclampsia

Initial management is the same regardless of the cause and the ongoing management includes supportive care and treatment of the aetiology.

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# Erratum

## Review O&G Forum 2014; 24:9-13: From dyspnoea to respiratory failure in pregnancy

1. Dr S. Cebakulu = Dr S.N. **Cebekhulu\***
2. Non-pregnancy related infections (mainly deaths in HIV infected pregnant women complicated by tuberculosis and pneumonia) contributed 40.5% of maternal deaths in **2008-2010\*** most of which are highly preventable
3. Early recognition and intervention of patients with these respiratory complications is important in the reduction of maternal mortality (Saving mothers report **2008-2010\***).
4. In those affected by HIV, TB and PCP are the most common causes of respiratory deaths in South Africa (Saving mothers report **2008-2010\***).

\*= Corrections