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ESSENTIALS Respiratory changes in pregnancy include an increase in tidal volume and minute ventilation, leading to a primary respiratory alkalosis. During a normal and uncomplicated pregnancy many women ex- perience the sensation of dyspnoea, hence it is important—but sometimes difficult—for the clinician to distinguish breathlessness resulting from normal physiological changes from that caused by underlying medical diseases. Chest conditions arising in pregnancy—these include (1) amniotic fluid embolism—unique to pregnancy; (2) venous air embolism—a rare condition that can occur in pregnancy; (3) venous and pul- monary thromboembolism—pregnancy is a risk factor; (4) pul- monary oedema—this can be caused by heart disease, as in the
nonpregnant state, but it can also be associated with pre-eclampsia or HELLP syndrome and be induced by tocolysis; (5) aspiration; (6) varicella pneumonia—a potentially devastating complication of primary varicella-zoster virus infection; (7) influenza—associated with increased maternal morbidity. Pregnancy in women with known chest disorders—(1) asthma— patients with a history of admission to an intensive care unit for asthma, prior mechanical ventilation, or frequent healthcare visits are at risk of developing severe or life-threatening asthma exacerba- tions during pregnancy. The treatment of chronic asthma and acute asthma exacerbations during pregnancy is largely the same as in the nonpregnant state; (2) pulmonary arterial hypertension—associated with high maternal mortality. Introduction Pregnancy is associated with increased minute ventilation and rela- tive hyperventilation, increase in thoracic diameter, pulmonary function changes, as well as cardiovascular and hormonal changes. Most women with mild pulmonary disease can have successful pregnancies, but some pre-existing chest diseases can put women at risk during pregnancy and parturition. During a normal and uncomplicated pregnancy many women experience the sensation of dyspnoea, hence it is important—but sometimes difficult—for the clinician to distinguish breathlessness resulting from normal physiological changes from that caused by underlying medical dis- eases. In this chapter, we review chest conditions unique to preg- nancy as well as management principles for important pre-existing chest diseases during pregnancy. Respiratory changes in pregnancy The respiratory system undergoes many changes during preg- nancy, in part as a result of elevated oestrogen and progesterone levels (Fig. 14.8.1).

Chest wall and diaphragm Changes to the chest wall and diaphragm occur early in pregnancy in response to changing hormone levels and before the uterus is large enough to exert any mechanical effects. The diaphragm rises up to 4 cm into the chest, and the ligaments of the ribs relax and cause the subcostal angle of the rib cage to increase, resulting in in- creased circumference of the chest. Ventilation and gas exchange Very early in pregnancy, changes in respiratory drive, ventilation, and gas exchange occur secondary to the stimulatory response from increased progesterone levels. The mechanism by which pro- gesterone affects respiratory drive and ventilation is explained by alterations in the sensitivity of chemoreceptors in the medulla to CO_2: even slight increases in arterial P_{CO_2} will cause an increase in tidal volume. Tidal volume increases up to 40% during preg- nancy, resulting in increased minute ventilation. The arterial P_{CO_2} falls from 40 mm Hg (5.3 kPa) in the nongravid patient to approxi- mately 30 mm Hg (4.0 kPa) in the pregnant patient, resulting in a primary respiratory alkalosis. In response, the kidney excretes bi- carbonate to normalize the pH, hence it is usual to see bicarbonate levels of approximately 20 mEq/litre during pregnancy. A second ef- fect of increased minute ventilation is a rise in alveolar and arterial oxygenation that produces arterial P_{O_2} levels ranging from 100 to 110 mm Hg (13.3–14.6 kPa). Lung volumes As tidal volume increases during pregnancy, the functional residual capacity decreases because the diaphragm rises up into the chest, 14.8 Chest diseases in pregnancy Meredith Pugh and Tina Hartert

Section 14 Medical disorders in pregnancy 2614 Fig. 14.8.1 Normal physiological changes of the upper airway, lung, and cardiovascular system during pregnancy that may result in dyspnoea or exacerbation of existing pulmonary disease. * (From Prowse CM, Gaensler EA (1965). Respiratory and acid-base changes during pregnancy. Anesthesiology, 26, 381–92. Top left and right insets duplicated, with permission.)

14.8 Chest diseases in pregnancy 2615 resulting in lower residual and expiratory reserve volumes. The forced expiratory volume in 1 s (FEV_1), FEV_1/forced vital capacity (FVC) ratio, and peak expiratory flow rates are unchanged during pregnancy. Hence a reduction in FEV_1 or FVC should prompt the clinician to seek out underlying pulmonary disease pathology to ex- plain the spirometric changes. Cardiovascular As outlined in Fig. 14.8.1, the cardiovascular changes that impact pulmonary physiology are observed as early as six to eight weeks' gestation. There is a 50% increase in blood volume to meet the in- creased metabolic demands of pregnancy and an increase in cardiac output by 1–2 litre/min. Cardiac output increases further during delivery and the post-partum period. Systemic and pulmonary vas- cular resistances are also reduced. Respiratory conditions in pregnancy Up to 70% of women experience the sensation of dyspnoea during a normal and uncomplicated pregnancy, hence it is important for the clinician to distinguish dyspnoea resulting from normal physio- logical changes of pregnancy from that caused by underlying med- ical diseases. The obstetric patient presenting with acute onset of shortness of breath requires a careful assessment to exclude life- threatening conditions detrimental to the mother or the fetus. Approach to dyspnoea in pregnancy Physiologic dyspnoea in pregnancy is common and due to hor- monal influences and physiologic changes described here. Physiologic dyspnoea has a gradual onset, often beginning in the first and second trimester, and lacks other associated symptoms or exam findings. Acute onset dyspnoea or the presence of wheezing, stridor, chest pain, fever, cough, and/or abnormal physical examin- ation (i.e. crackles on lung auscultation) suggest cardiopulmonary disease and further evaluation may be indicated. Pulmonary func- tion testing and chest radiography (with abdominal shielding) are low-risk and

may identify concomitant lung disease in the setting of these symptoms and signs. Specific disease considerations and diag- nostic testing are discussed in the remainder of this chapter. Pregnancy-associated rhinitis Upper airway mucosal oedema, hyperaemia, and hypersecretion occur during pregnancy and result in nasal congestion and/or naso- pharyngeal obstruction that may significantly impact a patient's ability to breathe comfortably. Treatment of this pregnancy-associated rhinitis includes raising the head of the bed 30–45 de- grees, saline nasal spray washings to help clear secretions; nasal ipratropium bromide and corticosteroids have not been shown to be clearly beneficial. Nasal sprays containing phenylephrine or oxymetazoline may also be used. Embolic disease Venous thromboembolism Venous thromboembolism complicates approximately 1–2 out of every 1000 pregnancies and is a leading cause of maternal mortality in the developed world. Diagnosing the condition during pregnancy may be challenging, since many of the symptoms in the nongravid patient can be a part of the normal physiologic changes observed in pregnancy (e.g. tachycardia, dyspnoea, lower extremity oedema). The diagnostic evaluation of venous thromboembolism in pregnant patients is similar to nonpregnant patients. D-dimer testing, widely used in low- and intermediate-risk nonpregnant patients, has limited utility for venous thromboembolism diagnosis during pregnancy. Compression ultrasonography is frequently used as the first test in pa- tients with leg symptoms and avoids ionizing radiation. If pulmonary embolism is suspected and venous ultrasound is negative, or no leg symptoms are present, then a chest X-ray followed by a ventilation- perfusion scan or computed tomography (CT) angiogram should be performed. CT angiography exposes the fetus to similar amounts of radiation as ventilation-perfusion scanning and offers the potential to make other diagnoses, but it delivers more ionizing radiation to maternal breast tissue. Low molecular weight heparin is the agent of choice for prophylaxis or treatment for venous thromboembolism in most cases. See Chapter 14.7 for further discussion. Venous air embolism Venous air embolism, with air travelling through the placental venous sinuses into the venous circulation and through the right ventricle leading to obstruction of the right ventricular outflow tract, has been reported during labour and delivery (most com- monly during caesarean delivery). Presenting symptoms and signs are dyspnoea, hypotension, tachycardia, tachypnoea, a character- istic 'millwheel murmur', or sudden cardiac arrest. Treatment is supportive. Amniotic fluid embolism Amniotic fluid embolism is a rare disorder unique to pregnancy. It is thought to be a severe systemic inflammatory response to fetal tissue components entering the maternal circulation, most com- monly during labour and delivery, or in the immediate post-partum period. The characteristic presentation is sudden onset of hypoxia, hypotension, and coagulopathy. The diagnosis of amniotic fluid em- bolism is a clinical one, and while detection of fetal elements may be found in maternal pulmonary artery aspirates, this finding is not exclusive to amniotic fluid embolism and there are no other proven laboratory markers specific to amniotic fluid embolism. Treatment is supportive, focusing on rapid cardiorespiratory stabilization and delivery of the fetus if necessary. Maternal mortality is very high (60–90%). Acute pulmonary oedema Acute pulmonary oedema during pregnancy is uncommon, with an incidence of 0.08% in a large retrospective study from a single med- ical centre. In this series, half of all patients developed pulmonary oedema as a result of either cardiac disease or tocolytic therapy; pre-eclampsia and iatrogenic volume overload were other common causes. Clinical signs and symptoms include dyspnoea, cough, orthopnoea, tachycardia, tachypnoea, crackles, and/or wheezing on lung auscultation, and hypoxia. Chest radiography findings are similar to pulmonary oedema in nonpregnant patients, including interstitial or alveolar pulmonary infiltrates and Kerley B lines. While we review here specific causes of acute pulmonary oedema related to pregnancy, other causes of pulmonary oedema not related to preg- nancy must be considered in the

appropriate context (i.e. acute re- spiratory distress syndrome, transfusion-related acute lung injury).

Section 14 Medical disorders in pregnancy 2616 Cardiogenic pulmonary oedema Cardiogenic pulmonary oedema can result from established or newly diagnosed cardiac disease. Valvular heart disease and other structural cardiac diseases are common precipitants of cardiogenic pulmonary oedema, but with advancing maternal age and presence of maternal comorbid conditions including diabetes mellitus, sys- tolic and diastolic heart disease contribute to some cases. Antenatal diagnosis, multidisciplinary management, and meticulous fluid management can reduce risks. Peripartum cardiomyopathy, defined as the development of heart failure towards the end of pregnancy or in the months following delivery in the absence of other causes, is another important cause of pulmonary oedema in pregnancy. See Chapter 14.6 for further discussion. Pre-eclampsia While uncommon (occurring in 3% of cases of pre-eclampsia), the development of pulmonary oedema is a leading cause of death in women with pre-eclampsia. The risk of developing pulmonary oe- dema is highest after delivery when plasma oncotic pressures drop to their lowest values and the distal air spaces of the lung fill with fluid. HELLP syndrome (haemolysis, elevated liver enzymes, and low platelets) can also result in pulmonary oedema. See Chapters 14.4 and 14.9 for further discussion. Tocolytic-induced pulmonary oedema Tocolytic-induced pulmonary oedema accounts for about 25% of all cases of pulmonary oedema during pregnancy. Most cases occur in the setting of systemic β2-agonist use for refractory preterm labour. The mechanism is unclear, but it is suspected that changes in haemo- dynamics as a secondary effect of β-receptor stimulation (i.e. tachy- cardia and increased stroke volume) accompanied by increased hydrostatic pressures and capillary leak lead to pulmonary oedema. Pulmonary oedema can be seen with the use of other tocolytics including magnesium sulfate and more recently used agents such as nifedipine and nicardipine, although believed to be less common. Treatment of tocolytic-induced pulmonary oedema consists of oxygen, diuretics, and discontinuing the offending drug. Aspiration Aspiration pneumonitis occurring during pregnancy, also termed Mendelson’s syndrome, is an important cause of acute respiratory distress syndrome and maternal morbidity. During normal preg- nancy, hormonal changes and enlargement of the uterus lead to de- creased oesophageal sphincter tone, delayed gastric emptying, and increased abdominal pressures. These physiological changes place pregnant patients at risk for aspiration, particularly during labour and delivery. Preventive measures to reduce the risk of aspiration include the use of regional anaesthesia, gastric acid suppression, and limiting oral intake around the time of labour and delivery. Asthma Asthma is a common chest disease in pregnancy, occurring in up to 8% of pregnant women in the United States of America. Poorly controlled or severe maternal asthma during pregnancy is asso- ciated with increased risk of pre-eclampsia, low birth weight in- fants, preterm delivery, and perinatal mortality. Patients with a history of admission to an intensive care unit for asthma, prior mechanical ventilation, and frequent healthcare visits are at risk of developing severe or life-threatening asthma exacerbations during pregnancy. Several studies have demonstrated that optimal management of asthma is associated with improved maternal and infant outcomes. The principles of managing asthma during pregnancy are not different from those in nongravid women, and management goals focus on avoidance of triggers, controlling symptoms, optimizing pulmonary function, and preventing and appropriately treating exacerbations. The treatment of chronic asthma and acute asthma exacerbations during pregnancy is largely the same as in the non- pregnant state. Short-acting β agonists (salbutamol) are used as initial therapy and for rapid-acting relief of bronchospasm. When asthma symptoms are persistent, use of a controller therapy,

usually an inhaled corticosteroid, is initiated. Therapy can be ‘stepped up’ to achieve adequate control with addition of long-acting β agonists (formoterol, salmeterol) when necessary. There is limited information regarding the safety and efficacy of asthma medications in pregnant women, primarily because preg- nant women are generally excluded from clinical studies, but ex- tensive clinical experience and registry studies inform clinicians regarding the safety of asthma therapy in pregnancy. β agonists are felt to be safe, with more evidence available for salbutamol than the newer longer acting β agonists. Inhaled corticosteroids have not been linked to adverse pregnancy outcomes, with most data avail- able for budesonide. In a large cohort, Schatz and colleagues did not identify a significant relationship between use of chronic asthma medications and perinatal adverse outcomes. Pulmonary arterial hypertension Pregnancy in patients with pulmonary arterial hypertension, a dis- ease of increased pulmonary vascular resistance and consequent right ventricular failure, is poorly tolerated due to the cardiopulmonary demands of pregnancy including increased cardiac output, stroke volume, and increased circulating blood volume. Deterioration is commonly related to right heart failure and can occur during early second and third trimester as well as during delivery and the post- partum period. Earlier series reported maternal mortality of 30–50% in pulmonary arterial hypertension patients during pregnancy, and while recent reports show improved mortality (as low as 12% in one series), pregnancy-related mortality remains unacceptably high even with best available treatments. Pregnancy is generally contra- indicated in pulmonary arterial hypertension because of the high maternal mortality, and pregnant patients with pulmonary arterial hypertension should be referred to a specialist centre. Pulmonary infections during pregnancy Despite new antibiotics and advances in respiratory support, pneu- monia during pregnancy is still a significant cause of maternal and fetal morbidity and mortality, even though the incidence is similar to the general population. Respiratory failure due to pneumonia is the leading cause of fatal nonobstetric infection and the third leading cause for intubation during pregnancy. Adverse fetal outcomes in- clude preterm labour, increased need for tocolytics, and lower birth weights. Here we highlight the common respiratory infections com- plicating pregnancy as well as some atypical pathogens for which pregnant patients are at higher risk.

14.8 Chest diseases in pregnancy 2617 Community-acquired pneumonia The causative pathogens, presentation, and management of community-acquired pneumonia during pregnancy are similar to that in the nonpregnant state. Evaluation of the pregnant patient with suspected community-acquired pneumonia should include chest X-ray (with abdominal shielding) and initial antimicrobial treatment should be directed at S. pneumonia, H. influenzae, and atypical pathogens. Vaccination with the 23-valent pneumococcal polysaccharide vaccine (PPSV23, pneumococcal vaccine) has been shown to be effective in decreasing the prevalence of pneumococcal pneumonia in patient populations considered at high risk for mor- tality from pneumonia: it is recommended for patients considered immunosuppressed (e.g. diabetes mellitus, asthma, chronic ob- structive pulmonary disease) and may be given during pregnancy. Recently the Centers for Disease Control and Prevention (CDC)’s Advisory Committee on Immunization Practices recommended ad- ministration of the pneumococcal conjugate vaccine (PCV13) for adults with certain immunocompromising conditions (asplenia, cochlear implant, cerebrospinal fluid leak, chronic kidney disease, HIV), but there is no consensus recommendation about timing of and safety of PCV13 immunization in pregnancy. Varicella pneumonia Varicella-zoster virus infection in pregnancy is serious for both mother and fetus and is associated with high mortality (see Chapter 14.15). Signs and symptoms of varicella-zoster virus in- clude vesicular rash, dyspnoea, cough, fever, malaise, and pleuritic chest pain. The risk of varicella pneumonia complicating primary varicella-zoster virus infection during

pregnancy occurs particu- larly in the second or third trimester. The diagnosis is usually made clinically; radiographic findings are nonspecific. Mechanical ven- tilation may be required in about 50% of pregnant patients with varicella pneumonia (25% mortality in this group). Early treatment with aciclovir 10 mg/kg every 8 h intravenously is recommended, and observational data suggest improved outcomes with aciclovir treatment. While rare, congenital varicella syndrome is a feared complication of maternal varicella-zoster virus infection, with the greatest risk when maternal disease occurs before 20 weeks' gestation. The best method for preventing maternal and fetal complica- tions of varicella-zoster virus infection is preconception coun- selling and documentation of a history of varicella or presence of serum varicella antibodies (IgG). If either of these conditions is not met, then varicella vaccination is recommended before preg- nancy, preferably one to three months before conception. Varicella vaccination is not recommended for use during pregnancy as it is a live-attenuated vaccine. Pregnant women without evidence of im- munity to varicella-zoster virus who have been exposed to varicella- zoster virus are eligible to receive varicella-zoster immune globulin as recommended by the United States Advisory Committee on Immunization Practices. Influenza Influenza A and B are common causes of respiratory illness, with influenza A being the most virulent strain in humans. During the influenza season pregnant women have over fivefold higher influenza-related morbidity compared to nongravid women, and also increased mortality during pandemic years. During the in- fluenza A H1N1 pandemic in 2009–2010, mortality among preg- nant women was high related to severe influenza pneumonia and acute respiratory distress syndrome, particularly during late preg- nancy (second and third trimester). While asthma and obesity were common comorbidities in pregnant patients with severe H1N1, nearly half of women with severe complications related to H1N1 had pregnancy as the only risk factor. Vaccination against influenza can reduce the risk of maternal and fetal influenza illness, and no adverse fetal outcomes have been identified in women who received the inactivated vaccine during pregnancy. The live-attenuated intranasal spray vaccine should not be given to pregnant women. Influenza vaccine is recommended for all women pregnant during the influenza season, regardless of trimester. Antiviral medications (amantadine, oseltamivir, zanamivir) are effective in prophylaxis for influenza in high-risk pregnant pa- tients and in treatment of influenza illness. While there are reports describing congenital malformations associated with amantadine and oseltamivir, the benefits of therapy may outweigh the risks depending on the clinical scenario. During the 2009 H1N1 epi- demics, use of antiviral therapy (oseltamivir, amantadine, or zanamivir, alone or in combination) resulted in fewer deaths. Fungal pneumonia Fungal pneumonia during pregnancy is rare, but in the setting of disseminated disease carries an increased risk of maternal mor- tality, preterm births, and perinatal mortality. Coccidioidomycosis is primarily found in semiarid areas in the western hemisphere, such as the south-western portion of the United States, central and nor- thern areas of Mexico, and endemic pockets in Central and South America. Coccidioidomycosis pneumonia tends to occur in the third trimester of pregnancy. Other causes of fungal pneumonia including Cryptococcus neoformans, Histoplasma capsulatum, and Blastomyces dermatitidis can similarly complicate pregnancy but are more uncommon. For patients with severe fungal pneumonia or disseminated disease, amphotericin B is recommended, followed by oral antifungals such as fluconazole after delivery. In pregnant women without pre-existing medical diseases, coccidioidomycosis pneumonia usually resolves on its own regardless of whether or not treatment is given. Acute respiratory failure during pregnancy Acute respiratory failure is a rare complication in pregnancy but needs to be promptly identified and treated to minimize maternal and fetal morbidity. Initial management is similar to management in the nonpregnant state, with support of oxygenation and ventila- tion, identification of the cause, and prompt initiation of directed therapy.

Many of the major causes of acute respiratory failure are discussed earlier in this chapter. The physiological changes of preg- nancy need to be considered when optimizing pulmonary mech- anics and gas exchange during mechanical ventilation. Pregnant women normally exhibit a respiratory alkalosis with a mean baseline arterial pH of 7.44 and an arterial CO_2 of approximately

Section 14 Medical disorders in pregnancy 2618 32 mm Hg (4.3 kPa), and this level of CO_2 should be considered the target during mechanical ventilation. Animal studies suggest that overventilation to an arterial partial pressure of CO_2 significantly below this level may compromise uterine blood flow and should be avoided. The strategy of low tidal volume ventilation with permis- sive hypercapnoea, proven to improve mortality in acute respira- tory distress syndrome, has not been rigorously studied in pregnant women but does not appear to have adverse effects on the fetus, at least to a CO_2 level of 60 mm Hg (8 kPa). To optimize fetal oxy- genation, maternal oxygenation goals may be higher than in non- gravid patients, as adequate fetal oxygenation requires an arterial oxygen tension of at least 70 mm Hg (9.3 kPa), corresponding to a maternal oxygen saturation of 95%. FURTHER READING Physiology of pregnancy Gilroy RJ, Mangura BT, Lavietes MH (1988). Rib cage and abdominal volume displacements during breathing in pregnancy. Am Rev Resp Dis, 137, 668–72. Lim VS, Katz AI, Lindheimer MD (1976). Acid-base regulation in pregnancy. Am J Physiol, 231, 1764–9. Pernoll ML, et al. (1975). Ventilation during rest and exercise in preg- nancy and postpartum. Resp Physiol, 25, 295–310. Prowse CM, Gaensler EA (1965). Respiratory and acid-base changes during pregnancy. Anesthesiology, 26, 381–92. Embolic disease Bates SM, et al. (2012). VTE, thrombophilia, antithrombotic therapy, and pregnancy: Antithrombotic Therapy and Prevention of Thrombosis, 9th ed: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines. Chest, 141 (Suppl), e691S–e736S. Bourjeily G, et al. (2010). Pulmonary embolism in pregnancy. Lancet, 375, 500–12. Clark SL (2014). Amniotic fluid embolism. Obstet Gynecol, 123, 337–48. Leung AN, et al. (2011). An Official American Thoracic Society/ Society of Thoracic Radiology Clinical Practice Guideline: evalu- ation of suspected pulmonary embolism in pregnancy. Am J Respir Crit Care Med, 184, 1200–8. Acute pulmonary oedema Dennis AT, Solnordal CB (2012). Acute pulmonary oedema in preg- nancy. Anaesthesia, 67, 646–59. Pisani RJ, Rosenow EC, III (1989). Pulmonary edema associated with tocolytic therapy. Ann Inter Med, 110, 714–18. Sciscione AC, et al. (2003). Acute pulmonary edema in pregnancy. Obstet Gynecol, 101, 511–15. Asthma Asthma in pregnancy in: 2016 BTS/SIGN Guideline for the manage ment of asthma. https://www.brit-thoracic.org.uk/document-library/ clinical-information/asthma/btssign-asthma-guideline-2016/ Bonham CA, Patterson KC, Strek ME (2018). Asthma outcomes and management during pregnancy. Chest, 153, 515–27. National Heart, Lung, and Blood Institute, National Asthma Education and Prevention Program Asthma and Pregnancy Working Group (2005). J Allergy Clin Immunol, 115, 34–46. Schatz M, et al. (2004). The relationship of asthma medication use to perinatal outcomes. J Allergy Clin Immunol, 113, 1040–5. Pulmonary arterial hypertension Banerjee D, Ventetuolo C (2017). Pulmonary hypertension in preg- nancy. Semin Respir Crit Care Med, 38, 148–59. Jais X, et al. (2012). Pregnancy outcomes in pulmonary arterial hyper- tension in the modern management era. Eur Respir J, 40, 881–5. Pulmonary infections during pregnancy Brito V, Niederman MS (2011). Pneumonia complicating pregnancy. Clin Chest Med, 32, 121–32. Martin SR, Foley MR (2006). Intensive care in obstetrics: an evidence- based review. Am J Obstet Gynecol, 195, 673–89. Pastuszak AL, et al. (1994). Outcome after maternal varicella infection in the first 20 weeks of pregnancy. N Engl J Med, 330, 901–5. Siston AM, et al. (2010). Pandemic 2009 influenza (H1N1) virus illness among pregnant women in the United States. JAMA, 303, 1517–25. Zaman K, et al. (2008). Effectiveness of maternal influenza immuniza- tion in mothers and infants. N Engl J Med, 359, 1555–64. Acute respiratory failure during pregnancy Ende H, Varelmann D (2016).

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