



Review Article

Dyspnea in pregnancy



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ABSTRACT

Dyspnea in pregnancy is common. It can result from adaption to body changes in pregnancy and also from complications therein. Understanding the mechanisms of change in the respiratory system during pregnancy helps with the differential diagnosis of dyspnea in normal pregnancy as opposed to pathological dyspnea.

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Introduction

Dyspnea in pregnancy is common [1–5]. Physiological changes during pregnancy influence maternal respiratory function and gas exchange, and may cause dyspnea in normal pregnancy. On the other hand, dyspnea can be caused by pregnancy complications, thus requiring optimal medical treatment. Making a correct assessment requires an understanding of the cardiopulmonary changes that occur during normal pregnancy, so as to recognize the syndrome of dyspnea. In this review, the aim was to outline the mechanisms of change within the respiratory system during pregnancy, and thus to enable a differential diagnosis of dyspnea in normal pregnancy as opposed to pathological dyspnea.

Material and methods

A focused literature search with consultation from a professional librarian was performed on data from January 1966 to December 2016. References of identified studies were also checked

for relevancy. Hand-searches of relevant journals were also performed.

Respiratory system changes in pregnancy

Pregnancy induces profound changes in the mother, resulting in significant alterations to the normal physiology. Anatomical and respiratory physiological changes affect the respiratory system during pregnancy [6–9].

Alterations to pulmonary physiology during pregnancy

Pulmonary function changes in pregnancy

The volume in the lung, as total lung capacity, can be divided into tidal volume, inspiratory reserve volume, expiratory reserve volume, and residual volume. The net result of anatomical changes to the thoracic cage during pregnancy is a more barrel-chested appearance. The enlarging uterus moves the resting position of the diaphragm cephalad, and decreases the expiratory reserve volume (an approximate 10–20% reduction), as well as functional residual capacity [8,10–13].

However, the enlarged uterus does not limit the movement of the thoracic cage, but results in a 4.0 cm maximal increase in the level of the diaphragm, together with a 2.1 cm maximal increase

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in the transverse diameter of the chest [4,13,14]. The subcostal angle increases progressively from an average of 68.5° in early pregnancy to 103.5° in late pregnancy, which remains the maximal inspiratory and expiratory pressure, and compensates for the reduction in abdominal replacement. Vital capacity (the volume combination with tidal volume, inspiratory reserve volume and expiratory reserve volume), the maximal amount of air that can be exhaled following maximal inspiration therefore remains constant or slightly increases during pregnancy [4,8,12,13,15].

Hormonal changes in pregnancy affect the upper respiratory tract and airway mucosa. The increase in estrogen during pregnancy causes airway mucosal hyperemia, edema and hypersecretion, and friability [16–18]; however, the patency of the airway and the ability of the gas exchange across the alveoli remain stable. The forced vital capacity, forced expiratory volume in 1 s, and the ratio of forced expiratory volume in 1 s to forced vital capacity tested by spirometer standing for the patency of the airway are similar in pregnancy and postpartum [5,8,19–21]. The differences of gas change of pulmonary respiration measured by the diffusing capacity of the lung for the monoxide are not significant between normal pregnant and non-pregnant women [22].

Taken together, although anatomical changes in pregnancy decrease the expiratory reserve volume and functional residual capacity, the vital capacity, airway patency, and gas exchange in healthy pregnant women are maintained (Table 1).

Ventilation changes in pregnancy

Minute ventilation and oxygen consumption increase over time in pregnancy [1,12,13,23,24]. The former increases significantly, beginning in the first trimester, and escalating to 20–40% above baseline at term [1,5,23,24]. Tidal volume increases by 30–35% and alveolar ventilation by 50–70%, with partial pressure of oxygen in arterial blood (PaO₂) ranging from 100 to 110 mmHg [12,24–26]. Oxygen consumption increases at the beginning of the first trimester, and by 20–33% per term, because of fetal demands and increased maternal metabolic processes [12]. In addition, physiological hyperventilation results in respiratory alkalosis, with the compensatory renal excretion of bicarbonate. Partial pressure of carbon dioxide in arterial blood (PaCO₂) reaches a plasma level of 28–32 mmHg, and bicarbonate decreases to

18–21 mmol/L. An arterial pH is maintained in the range of 7.40–7.47 [5,12,25–28].

Multiple factors control breath (Fig. 1). The respiratory loop consists of a forward part (pulmonary respiration to maintain appropriate PaO₂ and PaCO₂), and a feedback part (chemoreceptors initiate pulmonary ventilation as the response to the PaO₂, PCO₂ and the concentration of hydrogen ions in the blood) [29–32]. Hyperventilation in human pregnancy has been shown to result from an increase in wakefulness drive to breathe [33,34], and an increase in the response to the chemoreceptor stimuli, including the increases in central and peripheral chemoreceptor sensitivity [12,34–36] and a reduction in the ventilator recruitment threshold [29,37–39], together with an increase in metabolism (the elevation of CO₂ production) [24]. In addition, when chemoreceptors response to the stimuli, tidal volume increases before the change in respiratory frequency [32]. Hyperventilation in pregnancy usually shows a higher tidal volume rather than a tachypnea [8,13,14].

The progressive increase in progesterone and estrogen during pregnancy is one of the factors that account for an increase in physical demands and hyperventilation [6,13,40]. Progesterone acts as a trigger of the primary respiratory center by reducing the threshold and by increasing the sensitivity of the respiratory center to CO₂ [35,36,41,42], while estrogen increases the number and sensitivity of progesterone receptors within the hypothalamus and medulla (the central neuronal respiratory-related areas) [42,43]. In addition, progesterone and estrogen increase the sensitivity of the peripheral chemoreceptor to hypoxic conditions [36]. Taken together, the elevation in progesterone and estrogen cause an increase in the wakefulness drive and central and peripheral chemoreceptor sensitivity, and a reduction in the ventilator recruitment threshold, resulting in hyperventilation in pregnancy (Fig. 2).

Dyspnea in normal pregnancy

Dyspnea occurs in normal pregnancy [5]. Milne et al. studied the incidence of dyspnea in 62 normal pregnant women [2]. Nine were aware of dyspnea during the first trimester, 31 by week 19, and 46 by week 31. Since dyspnea commences in the first gestation trimester, it is likely that biochemical and mechanical changes co-contribute to dyspnea during pregnancy. Additionally, the increase in respiratory drive in pregnancy has led to an increase in tidal volume rather than the respiratory frequency [8,13,14,24], reflecting that the dyspnea is not associated with tachypnea.

Dyspnea is awareness of respiratory distress or breathlessness [44,45]. The perception of breathlessness correlates well with respiratory drive [23,46,47]. Thus, an increase in respiratory drive in pregnancy can contribute to the perception of dyspnea [36,44,48]. Garcia-Rio et al. showed that normal pregnant women with dyspnea had a higher respiratory drive when responding to serum CO₂ and hypoxia, even though they had similar oxygen consumption, lung volume distribution and respiratory muscle strength to those with normal breathing [23].

Dyspnea is believed to be caused by a discrepancy or mismatch between the feed-forward message to the ventilatory muscles and the feedback from receptors monitoring the response of the ventilatory pump [45,49]. When the discrepancy is high, the dyspnea occurs [45,49]. The unfamiliar low level of PaCO₂ resulting from hyperventilation may contribute to women who are prone to developing dyspnea in pregnancy [50]. However, the other scientists believe that the excessive ventilation response to PaO₂ or PaCO₂ is related to the physiologic dyspnea in pregnancy [23,34,36]. Nevertheless, not only the increased respiratory drive because of

Table 1
Modifications of pulmonary function in pregnancy.

	Changes
Spirometry	
FVC	↔
FEV1	↔
FEV1/FVC	↔
Lung volume	
TLC	↔
VC	↔ ↑
TV	↑
ERV	↓
FRC	↓
Lung diffusion test	
D _L CO	↔

D_LCO: diffusing capacity of the lung for carbon monoxide; ERV: expiratory reserve volume; FEV1: forced expiratory volume in 1 s; FEV1/FVC: ratio of forced expiratory volume in 1 s to forced vital capacity; FRC: functional residual capacity; FVC: forced vital capacity; VC: vital capacity; TLC: total lung capacity; TV: tidal volume.

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