

# Alveolar Oxygen And Carbon Dioxide Tensions During Pregnancy, Measured With A Novel Technique\

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## Citation

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## Abstract

Alveolar O<sub>2</sub> and CO<sub>2</sub> tensions (P<sub>A</sub>O<sub>2</sub> and P<sub>A</sub>CO<sub>2</sub>) and alveolar-to-arterial oxygen difference (P<sub>(A-a)</sub>O<sub>2</sub>) during pregnancy have been calculated with mathematical formulas that require arterial blood taking and are based on the assumption that alveolar and arterial CO<sub>2</sub> tensions are equal (P<sub>(A-a)</sub>CO<sub>2</sub>=0). The aim of this study was to investigate the changes of alveolar gas tensions during pregnancy, using a novel non-invasive technique. P<sub>A</sub>O<sub>2</sub>, P<sub>(A-a)</sub>O<sub>2</sub>, P<sub>A</sub>CO<sub>2</sub> and P<sub>(A-a)</sub>CO<sub>2</sub> were measured in 10 healthy volunteers during the 3rd trimester of pregnancy and when not pregnant. During pregnancy, the P<sub>A</sub>O<sub>2</sub>, P<sub>(A-a)</sub>O<sub>2</sub>, P<sub>A</sub>CO<sub>2</sub> and P<sub>(A-a)</sub>CO<sub>2</sub> values (in mmHg with SD) were: 101.67 (8.931), 0.78 (7.848), 26.05 (4.670), -4.86 (3.257). When not pregnant the respective values were: 95.75 (8.976), 3.56 (6.047), 35.28 (1.618), -2.63 (1.532). P<sub>(A-a)</sub>CO<sub>2</sub> was different to zero and its absolute value was increased during pregnancy (p=0.01). We detected a change in P<sub>(A-a)</sub>CO<sub>2</sub> during pregnancy suggesting an increase of the shunt, possibly caused by the elevation of the diaphragm and the collapse of alveoli at the lung bases. The changes in P<sub>A</sub>O<sub>2</sub> and P<sub>A</sub>CO<sub>2</sub> can be attributed to the hyperventilation of pregnancy and are comparable to the literature.

## INTRODUCTION

Even though respiratory function in pregnancy has been thoroughly studied, there are only few papers reporting on alveolar O<sub>2</sub> and CO<sub>2</sub> tensions (P<sub>A</sub>O<sub>2</sub> and P<sub>A</sub>CO<sub>2</sub>) and alveolar-to-arterial differences (P<sub>(A-a)</sub>O<sub>2</sub> and P<sub>(A-a)</sub>CO<sub>2</sub>). Mathematical calculations of P<sub>A</sub>O<sub>2</sub> had been based on the assumptions that alveolar PCO<sub>2</sub> equals end-tidal (P<sub>ET</sub>CO<sub>2</sub>) or arterial (P<sub>a</sub>CO<sub>2</sub>) CO<sub>2</sub> tension (1,2,3). A non invasive technique has recently been developed for measuring alveolar O<sub>2</sub> and CO<sub>2</sub> tensions (1,4,5,6). Using this, it was found that P<sub>A</sub>CO<sub>2</sub> is closely similar to P<sub>a</sub>CO<sub>2</sub> in normal subjects but significantly different in other cases, as in chronic obstructive pulmonary disease (COPD) (1). P<sub>A</sub>CO<sub>2</sub> is significantly higher than P<sub>ET</sub>CO<sub>2</sub> in all subjects, especially in COPD patients (1).

The aim of this study was to investigate the changes of the alveolar O<sub>2</sub> and CO<sub>2</sub> tensions and the alveolar-to-arterial differences during pregnancy, using this novel technique.

## METHODS

Alveolar and arterial O<sub>2</sub> and CO<sub>2</sub> tensions were measured during the third trimester of pregnancy and at least three months after delivery on ten healthy volunteers. The theoretical background of the method used in this paper has been presented in details elsewhere(1). The experimental set

up is also referred elsewhere(1,6). The subjects were studied while seated, breathing room air through the mouthpiece apparatus with a nose-clip on, at their own resting tidal volume and respiratory frequency. They were offered training and allowed time to relax and become accustomed to the apparatus and the procedure. After regular breathing had been achieved, a series of breaths over a period of 2 minutes were recorded. At the end of the recording time, an arterial blood sample was taken for gas analysis. An expert physician using 21G needle performed a quick and direct puncture of the brachial artery. The arterial gases were measured with a blood gas analyser (CIBA-CORNING; 288 Blood gas system, MA, USA). For the analysis paired t-test was used and p<0.05 was considered statistically significant. The study had the approval of the local ethics committee and all the women had given informed consent.

## RESULTS

Ten healthy pregnant volunteers entered the study. Mean age was 31 (range 24-37), mean height 163.8cm (159-170) and mean weight when not pregnant 67kg (56-85). All denied cigarette smoking or drug taking (other than iron and vitamin supplements). All pregnancies were singleton and uncomplicated.

P<sub>A</sub>CO<sub>2</sub> was significantly reduced in pregnancy [26.05mmHg

(SD 4.670) versus 35.28mmHg (1.618),  $p < 0.001$ ].  $P_A O_2$  showed some increase that did not reach statistical significance [101.67mmHg (8.931) versus 95.75mmHg (8.976),  $p = 0.14$ ]. Mean  $P_{(A-a)} CO_2$  in pregnancy was found to be different to zero ( $p < 0.001$ ) and its absolute value was increased compared to non pregnant status [-4.86mmHg (3.257) versus -2.63mmHg (1.532),  $p = 0.01$ ].  $P_{(A-a)} O_2$  did not change significantly [0.78mmHg (7.848) versus 3.56mmHg (6.047)].

### DISCUSSION

Alveolar  $CO_2$  tension was found significantly decreased during pregnancy, as expected due to the hyperventilation of pregnancy. For the same reason  $P_A O_2$  was increased, even though this difference did not reach statistical significance, probably due to the small size of the population studied. The same changes were noted for arterial tensions. Previous studies of  $P_A O_2$  in the third trimester of pregnancy, based on arterial blood taking, suggest values of 119.3mmHg<sup>(7)</sup>, 115.5mmHg<sup>(8)</sup>, 117.8mmHg<sup>(9)</sup>.

All the previous studies of alveolar gases in pregnancy were based on the assumption that alveolar  $CO_2$  pressure is equal to arterial. Even though  $P_a CO_2$  may be a good estimate of the  $P_A CO_2$  in normal subjects, this is not so in other cases<sup>(1)</sup>. Our study confirmed a statistically significant difference between the two during the third trimester of pregnancy. However, because the  $P_{(A-a)} CO_2$  difference is relatively small, its effect on the calculations in the previous studies is also small.

There was an increase of the absolute value of  $P_{(A-a)} CO_2$  during pregnancy. Because the diffusing capacity of  $CO_2$  is high, the tension of  $CO_2$  in the blood that perfuses ventilated alveoli becomes equal to that of the alveolar air. If all the ventilated alveoli were perfused, the alveolar and arterial  $CO_2$  tensions should be equal. The difference between the two ( $P_{(A-a)} CO_2$ ) represents the physiological shunt that is normally very small. The increased  $P_{(A-a)} CO_2$  in pregnancy suggests that the shunt in pregnancy is increased. It has been suggested that elevation of the diaphragm in pregnancy

causes ventilation/perfusion imbalance in the lower lung zones, and a resulting increased venous admixture (shunt)<sup>(10)</sup> but others<sup>(7)</sup> argued there was no evidence of this. The present study demonstrates a change of the alveolar to arterial difference in pregnancy and suggests that the shunt during pregnancy increases.

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