

NAME; YUSUF JEMIMAH SULE

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ASSIGNMENT

1. Elucidate the physiological adaptations of the female to pregnancy.

Maternal cardiovascular adaptations during pregnancy, such as cardiac output, blood volume, hormones, water and electrolyte balance, arterial blood pressure, vascular resistance, uterine blood flow amongst others ensure the well-being of the mother and of the fetus. Several of these changes are not yet well understood. This report reviews the factual changes in cardiovascular physiology during pregnancy. Methodological experimental problems are identified and their relationship to the interpretation of the data discussed. Open scientific questions are identified and the experimental designs necessary to answer them are discussed. The possible mechanisms which are responsible for these changes are addressed. Special attention is given to arterial blood pressure during pregnancy as the regulated variable. It is shown how cardiovascular parameters are involved in the regulation of blood pressure and the signals, which trigger these changes. The effects of the vasoactive agents, angiotensin II and catecholamines, on the circulation during pregnancy are reviewed. Alterations of baroreceptor function are discussed for this time period. Further investigations should assess venous function during pregnancy to elucidate mechanisms by which such maternal adaptations occur.

Pregnancy leads to diverse physiologic changes to accommodate the demands of the developing fetoplacental unit, which affect many major organ systems. Understanding these physiologic adaptations to pregnancy is important for all clinicians because they have important implications for the diagnosis and management of various disorders. This article provides a brief overview of the most notable of these adaptations, including cardiovascular, hematologic, respiratory, renal, immunologic, and gastrointestinal.

Pregnancy is a physiological phenomenon that imposes numerous changes on various organs and body systems of pregnant women, including their respiratory system, which naturally affect the health of both mother and fetus. In pregnant women, because of the increased anteroposterior and transverse diameter of the chest, displacement of the diaphragm, increased stress and need for oxygen, high prevalence of respiratory disorders is observed

This study examined the role of pregnancy-induced changes in wakefulness (or non-chemoreflex) and central chemoreflex drives to breathe, acid-base balance and female sex hormones in the hyperventilation of human pregnancy. Thirty-five healthy women were studied in the third trimester (TM<sub>3</sub>; 36.3 ± 1.0 weeks gestation; mean ± S.D.) and again 20.2 ± 7.8 weeks post-partum (PP). An iso-oxic hyperoxic rebreathing procedure was used to evaluate wakefulness and central chemoreflex drives to breathe. At rest, arterialized venous blood was obtained for the estimation of arterial PCO<sub>2</sub> (PaCO<sub>2</sub>) and [H<sup>+</sup>]. Blood for the determination of plasma strong ion difference ([SID]), albumin ([Alb]), as well as serum progesterone ([P<sub>4</sub>]) and 17β-estradiol ([E<sub>2</sub>]) concentrations was also obtained at rest. Wakefulness and central chemoreflex drives to breathe, [P<sub>4</sub>] and [E<sub>2</sub>], ventilation and V̇CO<sub>2</sub> increased, whereas PaCO<sub>2</sub> and the central chemoreflex ventilatory recruitment threshold for PCO<sub>2</sub> (VRTCO<sub>2</sub>)

decreased from PP to TM<sub>3</sub> (all  $p < 0.01$ ). The reductions in PaCO<sub>2</sub> were not related to the increases in [P<sub>4</sub>] and [E<sub>2</sub>]. The alkalinizing effects of reductions in PaCO<sub>2</sub> and [Alb] were partly offset by the acidifying effects of a reduced [SID], such that arterial [H<sup>+</sup>] was still reduced in TM<sub>3</sub> vs. PP (all  $p < 0.001$ ). A mathematical model of ventilatory control demonstrated that pregnancy-induced changes in wakefulness and central chemoreflex drives to breathe, acid–base balance, V̇CO<sub>2</sub> and cerebral blood flow account for the reductions in PaCO<sub>2</sub>, [H<sup>+</sup>] and V<sub>R</sub>TCO<sub>2</sub>. This is the first study to demonstrate that the hyperventilation and attendant hypocapnia/alkalosis of human pregnancy results from a complex interaction of pregnancy-induced changes in wakefulness and central chemoreflex drives to breathe, acid–base balance, metabolic rate and cerebral blood flow.