



Comparison Between Hypertonic Saline (3%) and Normal Saline (0.9%) as a Preload Before Spinal Anaesthesia in Caesarean Section

Thesis

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By

Mariam Gamal Motawashleh Salama

(M.B.B.CH. Ain Shams University)

Supervised by

Prof. Dr. Fahmy Saad Latif

*Professor of Anaesthesiology, Intensive Care & Pain Management
Faculty of Medicine – Ain Shams University*

Dr. Dalia Ahmed Ibraheem

*Lecturer of Anaesthesiology, Intensive Care & Pain Management
Faculty of Medicine – Ain Shams University*

Dr. Amin Mohammed Alansary

*Lecturer of Anaesthesiology, Intensive Care & Pain Management
Faculty of Medicine - Ain Shams University*

***Faculty of Medicine
Ain Shams University***

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بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

قَالَ

لَسْبَقَ أَنْتَ لَا أَعْلَمُ لَنَا
إِلَّا مَا عَلَّمْتَنَا إِنَّكَ أَنْتَ
الْعَلِيمُ الْعَظِيمُ

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List of Abbreviations

Abb.	Full term
BP	Blood pressure
CO	Cardiac output
DBP	Diastolic blood pressure
GA	Gestational age
Ht	Height
MBP	Mean blood pressure
NaCl	Sodium chloride
NO	Nitric oxide
ODS	Osmotic Demyelination Syndrome
PCWP	Pulmonary capillary wedge pressure
PVR	Pulmonary vascular resistance
RBC	Red blood cells
SBP	Systolic blood pressure
SVR	Systemic vascular resistance
WBC	White blood cells

WT WEIGHT

INTRODUCTION

Hypotension is the most common complication following spinal anaesthesia for cesarean delivery. In severe cases, it can have detrimental effects on both mother (unconsciousness and pulmonary aspiration) and neonate (hypoxia, acidosis, and neurological injury) (*Mitra et al., 2013*).

The prevention of hypotension appears more likely to decrease maternal symptoms than the treatment of established hypotension (*Iqbal et al., 2010*).

Initial fluid administration with isotonic fluids is often used for the prevention of hypotension. It is well tolerated by healthy young patients but not in patients with cardiovascular restrictions.

Because in patients with diminished cardiac reserve, If preloading is performed with a large volume of fluid, a substantial amount of excess free water will remain in the body after spinal anaesthesia, the excess free water may be harmful during postoperative recovery (*Jarvela et al., 2000*).

Colloid preload seems to be more effective than crystalloid in the prevention of spinal anesthesia induced hypotension but they are linked to a number of complications like anaphylactoid reactions and changes in coagulation function (*Ah-Young Oh et al., 2014*).

Hypertonic saline increases plasma osmolarity and causes fluid shift from the intracellular to the extracellular space. This improves the hemodynamic changes occurring with spinal anaesthesia.

There is no risk of allergic reactions like colloid infusion and is desired for cardiovascular restrictions because there is reduction of free water administration (*Michael, 2013*).

AIM OF THE STUDY

This study compares between hypertonic saline (3%) and normal saline (0.9%) in prevention of spinal induced hypotension in females undergoing elective caesarean section.

Chapter One

CARDIOVASCULAR CHANGES DURING PREGNANCY

Physiological changes occur in pregnancy to nurture the developing foetus and prepare the mother for labour and delivery. These changes begin after conception and affect every organ system in the body. They resolve after pregnancy with minimal residual effects in an uncomplicated pregnancy (*Priya et al., 2016*).

Cardiovascular changes are profound and occur early in pregnancy. They include including increases in cardiac output, arterial compliance, and extracellular fluid volume and decreases in blood pressure (BP) and total peripheral resistance (*Michael et al., 2011*).

Cardiac output gradually increases during the first 2 trimesters with the largest increase occurring by 16 weeks of gestation. The increase in cardiac output is well established by 5 weeks of gestation and increases to 50% above prepregnancy levels by 16 to 20 weeks of gestation. The rise in cardiac output typically plateaus after 20 weeks of gestation and remains elevated until term. The increases in cardiac output are associated with significant increases in stroke volume and heart rate (*Sanghavi and Rutherford, 2014*).

Uterine contractions lead to auto transfusion of 300–500 ml of blood back into the circulation and the sympathetic response to pain and anxiety further elevate the heart rate and blood pressure. Cardiac output is increased between contractions but more so during contractions. Following delivery there is an immediate rise in cardiac output due to relief of the inferior vena cava obstruction and contraction of the uterus. Cardiac output increases by 60–80%, followed by a rapid decline to pre-labour values within about one hour of delivery (*Priya et al., 2016*).

Increased cardiac output and slight decrease in BP during pregnancy is associated with a marked reduction in systemic vascular resistance. Total peripheral resistance decreases very early during pregnancy and continues to decrease throughout the second and third trimester (*Rebelo et al., 2015*).

A number of important factors are thought to contribute to physiological changes in the vascular system that occur during pregnancy. Substantial evidence indicates that nitric oxide (NO) production is elevated in normal pregnancy and that these increases appear to play an important role in the vasodilation of pregnancy resistance (*Michael et al., 2011*).

Hormonal factors such as estrogen and relaxin are thought to be important in stimulating the production of NO during pregnancy. Relaxin, which is primarily produced by the corpus luteum, has been shown to chronically reduce total

peripheral resistance and increase cardiac output and systemic arterial compliance (*Tkachenko et al., 2014*).

Neutralization of endogenous circulating relaxin by antibodies during early gestation markedly attenuate the changes in cardiac output, systemic vascular resistance, and arterial compliance during pregnancy (*Hall et al., 2011*).

Pulmonary vascular resistance (PVR), like systemic vascular resistance (SVR), decreases significantly in normal pregnancy. Although there is no increase in pulmonary capillary wedge pressure (PCWP), serum colloid osmotic pressure is reduced by 10–15%. The colloid osmotic pressure/pulmonary capillary wedge pressure gradient is reduced by about 30%, making pregnant women particularly susceptible to pulmonary oedema. Pulmonary oedema will be precipitated if there is either an increase in cardiac pre-load (such as infusion of fluids) or increased pulmonary capillary permeability (such as in pre-eclampsia) or both (*Priya et al., 2016*).

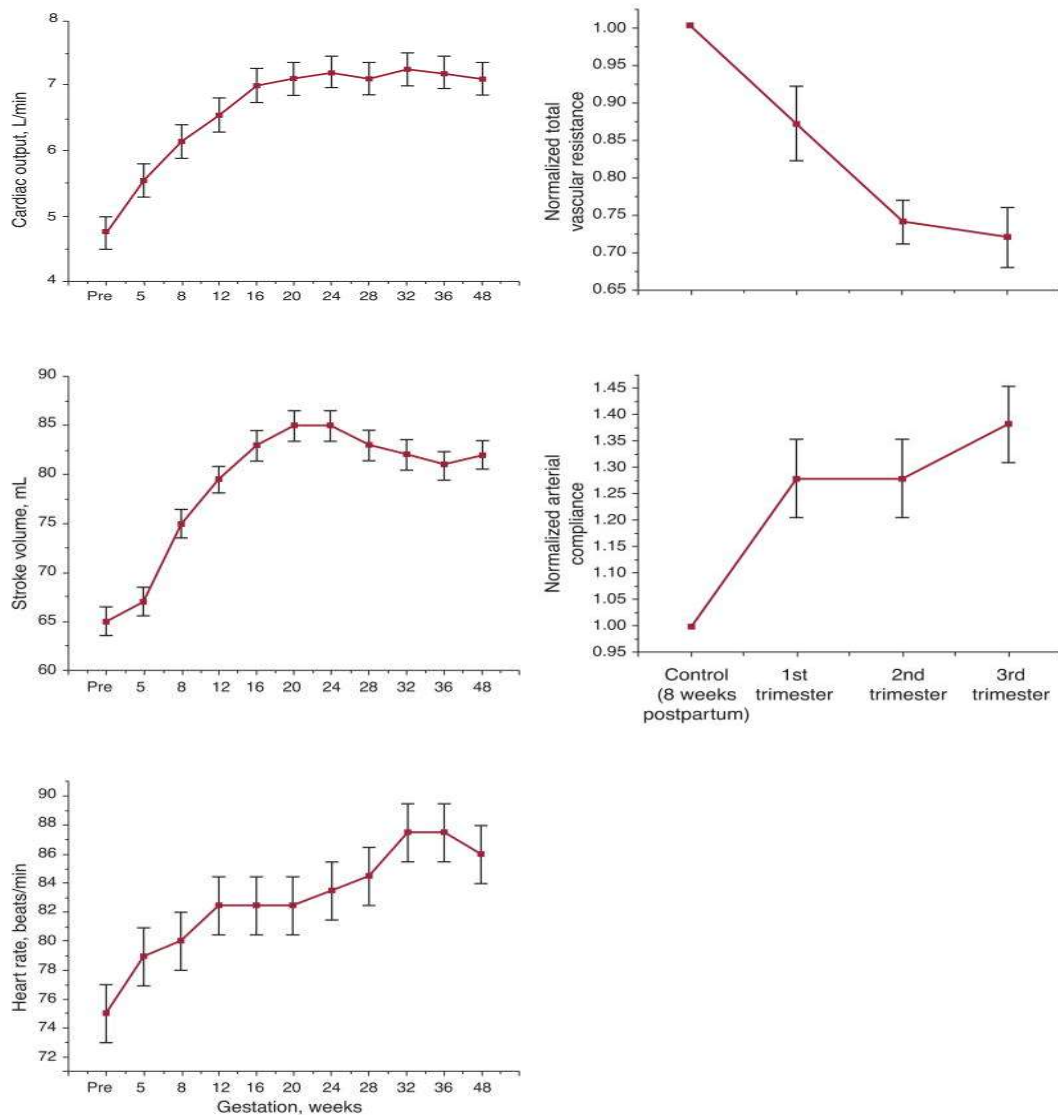


Figure (1): Changes in cardiac output, stroke volume, and heart rate during pregnancy (*Michael et al., 2011*).

Maternal blood volume increases by 40-50% at 6 to 8 weeks gestation. This, coupled with drop in serum albumin concentration, leads to decreased serum colloid osmotic pressure and hemodilutional anemia (*Costantine, 2014*).

While exact origin of the increased blood volume is not fully understood, the mechanism may be through nitric oxide mediated vasodilatation and increased arginine vasopressin production and mineralocorticoid activity, with water and sodium retention, leading to hypervolemia (*Tkachenko et al., 2014*).

The pregnancy induced hypervolemia is thought to provide survival advantage to the pregnant women, protecting her from hemodynamic instability with the blood loss at the time of delivery (*Pacheco et al., 2013*).

According to hematological changes; White (WBC) and red blood cells (RBC) counts increase during pregnancy. The first is thought to be secondary to bone marrow granulopoiesis; whereas the 30% increase in RBC mass (250–450 mL) is mainly driven by the increase in erythropoietin production (*Pacheco et al., 2013*).

Despite the increase in RBC mass, and as previously described, plasma volume increases significantly much higher (~45%), which leads to “physiologic anemia” of pregnancy. Anemia usually peaks early in the third trimester (30–32 weeks) and may become clinically significant in patients