

**Anaesthetic Considerations In Post Transplant  
Women Undergoing Elective  
Cesarean Section**

*Essay*

Submitted for partial Fulfillment of Master Degree  
in Anaesthesia

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**2018**



سورة المؤمنون - آية ٢٩



## Acknowledgement

First of all, I would like to express my deep gratitude to **ALLAH** for His care and generosity throughout my life.

I would like to express my sincere appreciation to **Prof. Dr. Waleed Abdelmageed Altaher**, Professor of Anesthesiology, Intensive Care and Pain Management, Faculty of Medicine – Ain Shams University, for his keen supervision and guidance and his overwhelming support that has been of great help throughout this work. I really have the honor to complete this work under his supervision.

I am very thankful to **Assist. Prof. Dr. Noha Sayed Hussein**, Assistant Professor of Anesthesiology, Intensive Care and Pain Management, Faculty of Medicine – Ain Shams University, for her great support and encouragement, and for the efforts and time she has devoted to accomplish this work.

 Mohamed Abdelsamad Abdelmohdy

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## List of Abbreviations

<i>Abbr.</i>	<i>Full-term</i>
<b>ALP</b>	: Alkaline phosphatase
<b>AP</b>	: Arterial pressure
<b>CNIs</b>	: Calcineurin inhibitors
<b>CO</b>	: Cardiac output
<b>CRF</b>	: Chronic renal failure
<b>ECG</b>	: Electrocardiogram
<b>EDV</b>	: End diastolic volume
<b>ERA-EDTA</b>	: European Dialysis and Transplant Association
<b>ESLD</b>	: End stage liver disease
<b>FDA</b>	: Food and Drug Agency
<b>FEV1</b>	: Forced expiratory volume in one second
<b>FRC</b>	: Functional residual capacity
<b>GRF</b>	: Glomerular filtration rate
<b>H+LTx</b>	: Heart and lung transplantation
<b>Hcg</b>	: Human chorionic gonadotropin
<b>HELLP S</b>	: Hemolytic anemia, elevated liver enzymes, low platelets syndrome

<b>HR</b>	: Heart rate
<b>HTx</b>	: Heart transplantation
<b>LTx</b>	: Lung transplantation
<b>MA</b>	: Maximum amplitude
<b>MAC</b>	: Minimal alveolar concentration
<b>NTPR</b>	: National Transplantation Pregnancy Registry
<b>OLT</b>	: Orthotopic liver transplantation
<b>PaCO<sub>2</sub></b>	: Pressure carbon dioxide
<b>SOT</b>	: Single organ transplantation
<b>SV</b>	: Stroke volume
<b>SVR</b>	: Systemic vascular resistance
<b>SVR</b>	: Systemic vascular resistance
<b>TEG</b>	: Thromboelastography
<b>TSH</b>	: Thyroid stimulating hormone
<b>UKTPR</b>	: United Kingdom (UK) Transplant Pregnancy Registry

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## **Introduction**

Successful pregnancy outcomes are possible among all solid organ transplant recipients. Patients should be counseled regarding the potential adverse fetal outcomes, including prematurity and low birth weight. Transplant recipients are at increased risk of both maternal and neonatal complications (*Deshpande et al., 2013*).

Renal transplantation restores fertility and successful pregnancies have been reported in renal transplant women. In women with normal graft function pregnancy usually has no adverse effect on graft function and survival (*Atallah et al., 2015*).

Women constitute <30% of patients undergoing liver transplantation and about 8% are of reproductive age (*Ramirez and Doria, 2014*).

Although pregnancy in orthotopic liver transplantation is associated with an increased incidence of hypertension, preeclampsia, anemia, preterm deliveries and cesarean section, acute rejection and liver allograft loss doesn't appear to be increased and pregnancy related maternal death is uncommon (*Hammoud et al., 2013*).

Since 1988, approximately 12,900 heart transplants have been performed in women with a 5 year graft and patient

survival of approximately 67.4% and 69%, respectively (*Taylor et al., 2007*).

Fertility and pregnancy in this patient raise complex issues although successful outcomes have been reported these pregnancies should be considered high risk for potential maternal and fetal complications (*Gundlapalli et al., 2015*).

Pregnancy affects drug absorption, distribution, and elimination. For instance, gut motility is slowed in pregnancy, but drug transfer through the gastrointestinal membranes is enhanced as a result of increased blood flow. Nausea and hyperemesis gravidarum may affect immunosuppressant intake.

Adequate counseling about return of fertility, contraception, and possibility of pregnancy is imperative for any woman of child bearing age undergoing evaluation for transplantation.

In deed following transplantation the disruption of hypothalamic gonadal axis associated with end organ disease is often corrected within 2- 6 months.

In general female recipients should be advised to wait 1-2 years after the transplant to become pregnant.

Vaginal delivery is recommended as the best for the patient, graft and the new born because of limited blood loss and lower risk of infections.

Cesarean section should be restricted for obstetrical indications or severe deterioration of maternal condition such as preeclampsia.

Cesarean section is performed in approximately 30% of heart transplant patient, 38% of lung transplant, which is comparable to liver recipients 35% but far less than renal recipients 50% (*Qi et al., 2016*).

## **Aim of the Work**

**T**he aim of the study is to review and focus on giving power and strength to transplanted patients undergoing elective cesarean section better than past times.

## Chapter (1)

# Physiology of pregnancy: Clinical anaesthetic implications

Pregnancy causes anatomical and physiological changes that have implications for the anaesthetist not only for intrapartum management but also when surgery is required incidentally to pregnancy. These adaptations primarily occur, so that the metabolic demands of the growing fetus may be met (*Bajwa et al., 2013*).

### Cardiovascular

Physiological changes occur very early in pregnancy, leading to an overall hyperdynamic circulation. These early hormonal effects lead to the primary event of peripheral vasodilatation which causes a decrease in the systemic vascular resistance (SVR). This occurs as early as 8 weeks of gestation (*Chang, 2004*).

If arterial pressure (AP) is to be maintained which is essential for an effective uteroplacental functioning unit, then the cardiac output (CO) has to be increased (*Bedson and Riccoboni, 2014*).

$$AP = CO (HR \times SV) \times SVR$$

This is initially achieved by increasing stroke volume (SV), but as pregnancy progresses, the increase in SV plateaus and there is an increase in heart rate (*Carpenter et al., 2015*).

The increase in SV occurs secondary to an increase in both end-diastolic volume (EDV) and ventricular muscle wall mass. Increased pre-load and EDV is a result of the increased blood volume which is progressive from 6 to 8 weeks gestation to a maximum volume at 32 weeks. There is an overall increase of up to 2000 ml in blood volume compared with the nonpregnant individual. As a result of this, the pregnant patient compensates well for blood loss (Table 1). By the time the classical symptoms and signs of hypovolaemia such as tachycardia, hypotension, and oliguria are evident, more than 1500 ml may have already been lost (*Wong, 2004*).

**Table (1):** The effects of blood loss in pregnancy

<b>% blood loss</b>	<b>Actual blood loss (ml)</b>	<b>Abnormal clinical findings</b>
15-20	1200-1500	None
20-25	1500-2000	Respiratory rate 14-20 Heart rate f 100-120 Systolic AP slight 4
>25%	>2000	Respiratory rate 20-30 Heart rate ff >120 AP 4 Restless Oliguria

Despite an increased CO, there is an early transient decrease in AP, resulting in a widened pulse pressure and a reduced mean arterial pressure. This activates the renin-angiotensin system leading to retention of water and sodium and ultimately an increase in plasma volume.

While the increase in plasma volume is in the region of 40-50%, the increase in red blood cell mass is only 20% resulting in the dilutional physiological anaemia of pregnancy. Central venous pressure and pulmonary capillary wedge pressure are unchanged (*Bedson and Riccoboni, 2014*).

The heart is physiologically dilated and displaced in both cephalad and lateral directions. A normal pregnancy ECG may have 15-20° left axis deviation and T waves may be inverted in lateral leads and lead III mimicking left ventricular hypertrophy and other structural disease (*Sunitha and Chandrasekharappa, 2014*).

It must be remembered however that incipient cardiac disease is an important cause of maternal death in the UK. Many of the initial presenting symptoms and signs may be ‘soft’ and wrongly attributed to the physiological changes of pregnancy (*Mocumbi et al., 2016*).

A high index of suspicion should be maintained and if there is any doubt, further investigations and cardiology referral should be considered (*CMACE, 2011*).

Anatomically, the iliac veins join to form the inferior vena cava at a level corresponding to the L4/5 interspace. Once the uterus is at this level, inferior vena cava compression may occur. By the time enlarging uterus approaches the level of the umbilicus, corresponding to 20 weeks in a singleton pregnancy, the mechanical effects of the enlarging uterus can cause compression of both the inferior vena cava and the descending aorta in the supine position. The combination of these leads to a reduced venous return and decreased CO (*Bedson and Riccoboni, 2014*).

By 38-40 weeks gestational age, there is a 25-30% decrease in CO when turning from the lateral to the supine position. As the uteroplacental circulation possesses no autoregulation properties, this causes a decreased uterine blood flow and reduced placental perfusion (*Griffiths and Campbell, 2014*).

Aortocaval compression can therefore lead to maternal hypotension and a subsequent fetal acidaemia. The maternal compensatory mechanisms for aortocaval compression comprise an increase in sympathetic tone, causing vasoconstriction and tachycardia and diversion of blood flow