

Cardiac Surgery and Percutaneous Intervention in Pregnant Ladies with Heart Diseases

An Essay

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Presented by

Mohamed Abdel Naby Alrahawy
M.B.B.Ch.

Under Supervision of

Prof. Dr. Azza Youssef Ibrahim

Professor of Anaesthesiology and Intensive Care
Faculty of Medicine- Ain Shams University

Dr. Waael Ahmed Abd Alaal

Lecturer of Anaesthesiology and Intensive Care
Faculty of Medicine- Ain Shams University

Dr. Rafik Emad Latif

Lecturer of Anaesthesiology and Intensive Care
Faculty of Medicine- Ain Shams University

**Faculty of Medicine
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بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

قالوا

سبحانك لا علم لنا
إلا ما علمتنا إنك أنت
العليم الحكيم

صدق الله العظيم

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List of Abbreviations

Abbreviation	Meaning
ACS	Acute coronary syndrome
AF	Atrial fibrillation
AMA	American heart association
ASD	Atrial septal defect
BV	Blood volume
CABG	Coronary artery bypasses grafting
CBV	Circulating blood volume
CHD	Congenital heart disease
CK	Creatine kinase
COP	Cardiac out put
CPB	Cardio pulmonary bypass
DES	Drug eluting stent
DVT	Deep venous thrombosis
ECG	Electro cardio graph
FDPS	Fragmented degradation products
FHR	Fetal heart rate
FRC	Function residual capacity
GFR	Glomerular filtration rate
HR	Heart rate
IUGR	Intera uterine growth retardation
IVC	Inferior vena cava
LMWH	Low molecular weight heparin
LV	Left ventricle

Abbreviation	Meaning
MI	Myocardial infarction
OPCABG	Off pump CABG
PCI	Percutaneous coronary intervention
PDA	Patent ductus arteriosus
PTMC	Percutaneous transluminal mitral commissurotomy
PV	Plasma volume
PVR	Peripheral vascular resistance
RV	Right ventricle
SV	Stroke volume
UBF	Uterine blood flow
VC	Vital capacity
VR	Venous return
VSD	Ventricular septal defect

Introduction

In pregnant women with heart disease, complications can arise due to the haemodynamic burden of pregnancy and to hypercoagulation. Most problems can be managed medically, but sometimes cardiac surgery or percutaneous intervention is unavoidable. Cardiac surgery has similar maternal mortality to that outside pregnancy, but foetal mortality and morbidity are considerable. When gestational age is > 28 weeks, pre-surgery delivery of the foetus should be considered. Percutaneous intervention exposes the foetus to radiation. The radiation dose for common cardiac procedures, however, does not result in detectable harmful foetal effects (*Smith et al., 2012*).

Heart disease is an increasingly important cause of maternal morbidity and mortality during pregnancy. Most cardiac problems that arise in pregnant women with heart disease can be managed without interventional procedures. However, the haemodynamic changes of pregnancy may sometimes lead to deterioration in previously stable women. Indications for intervention may arise when cardiac conditions worsen during pregnancy when the severity of the disease was undiagnosed or underestimated before pregnancy or when new complications or diagnoses arise for example prosthetic valve thrombosis (*Langesæter et al., 2011*).

Pregnancy in women with mechanical valve prostheses has a high maternal complication rate including valve thrombosis and death. Coumarin derivatives are relatively safe for the mother with a lower incidence of valve thrombosis than un-fractionated and low-molecular-weight heparin, but carry the risk of embryopathy, which is probably dose-dependent. When valve thrombosis occurs during pregnancy, thrombolysis is the preferable therapeutic option. Bioprostheses have a more favourable pregnancy outcome than mechanical prostheses but due to the high re-operation rate in young women they do not constitute the ideal alternative (*Mebazaa et al., 2011*).

Worldwide many prosthetic valves are yearly implanted in girls and young women with rheumatic or congenital heart disease. Sooner or later many of them wish to become pregnant. Mechanical prostheses, bioprostheses and native valve disease each carry specific risks during pregnancy. These may affect the timing and type of surgical therapy. Pre-pregnancy counselling as well as adequate monitoring and treatment when pregnancy is achieved are challenging tasks for cardiologists who care for these young women (*Hoeper et al., 2011*).

Aim of the Work

The aim of this work is to understand the severity of cardiac problems and their complications in pregnant females and when should the appropriate time for surgical intervention to solve these problems and prevent complications.

Chapter (1):

Physiology of Pregnancy

In many societies' medical or legal definitions, human pregnancy is divided into three trimester periods, as a means to simplify reference to the different stages of prenatal development (*Vissenberg et al., 2012*).

The first trimester carries the highest risk of miscarriage (natural death of embryo or fetus). During the second trimester, the development of the fetus can be more easily monitored and diagnosed. The beginning of the third trimester often approximates the point of viability, or the ability of the fetus to survive, with or without medical help, outside of the uterus (*Vissenberg et al., 2012*).

Physiological changes during pregnancy.

Cardiovascular changes

Cardiac output (COP) increases by up to 50% by the third trimester. Stroke volume increases by 35% predominately because of the increased blood volume. The increase in circulating estrogen and progesterone causes vasodilatation and a fall in peripheral vascular resistance (PVR). HR increases by 15 – 25%. LV hypertrophy and dilatation facilitate this change in COP but contractility remains unchanged. Together with the

upward displacement of the diaphragm, the apex is moved anterior and to the left. These changes may result in ECG findings of left axis deviation, depressed ST segments and inversion or flattening of the T-wave in lead III (*Yazbeck & Sullivan et al., 2012*).

Aortocaval compression syndrome

From mid-pregnancy the enlarging uterus compresses both the inferior vena cava IVC and the lower aorta when the patient lies supine. Compression of the IVC reduces venous return (VR) to the heart leading to a fall in pre-load and COP. The fall in blood pressure may be severe enough for the mother to lose consciousness. Compression of the aorta may lead to a reduction in uteroplacental and renal blood flow. During the last trimester, maternal kidney function is markedly lower in the supine than in the lateral position. Furthermore, fetal transplacental gas exchange may be compromised. For these reasons no lady should lie supine in late pregnancy (*Yazbeck & Sullivan et al., 2012*).

Most un anaesthetized ladies are capable of compensating for the resultant decrease in stroke volume (SV) by increasing systemic vascular resistance (SVR) and HR. Blood from the lower limbs may return through the paravertebral and azygos-systems. General anaesthesia,

subarachnoid and epidural blocks abolishes the sympathetic response and increase the risk of supine hypotension (*Yazbeck & Sullivan, 2012*).

Hematological changes

Blood Volume increases progressively from 6-8 weeks of gestation and reaches a maximum at approximately 32-34 weeks. The plasma volume increases by 45% mediated by progesterone and estrogen acting on the kidneys initiating renin-angiotensin and aldosterone pathways. Total body water increases secondary to renal sodium retention. Most of the added volume is accounted for by an increased capacity of the uterine, breast, renal, striated muscle and cutaneous vascular systems, with no evidence of circulatory overload in the healthy lady (*Dittrich et al., 2011*).

Renal erythropoietin increases red cell mass by 20-30% which is a smaller rise than the plasma volume, resulting in hemodilution and a decrease in hemoglobin concentration from 15 g/dl to 12 g/dl. This is termed the physiological anemia of pregnancy. Supplemental intake of iron and folic acid help to restore hemoglobin levels (*Dittrich et al., 2011*).

The blood volume returns to normal 10-14 days post partum. The increased blood volume reduces the impact of maternal blood loss at delivery. An "autotransfusion" of blood

from the contracting uterus compensates for the typical losses of 300-500 ml for vaginal delivery and 750-1000 ml for a section. This can delay the onset of the classical signs and symptoms of hypovolemia (*Dittrich et al., 2011*).

Changes in Coagulation

Pregnancy affects the normal balance between intravascular coagulation and fibrinolysis, inducing a hypercoagulable state. With the exception of FXI and FXIII, plasma concentrations of all clotting factors increase. Increased levels of antithrombin III and (FDP) reflect enhanced fibrinolysis. Platelet production is increased but the platelet count falls because of dilution and consumption. Platelet function remains normal.

Respiratory changes

Changes in the respiratory system are of great significance to the anaesthetist and may be categorised as anatomical and physiological changes.

1: Anatomical changes

Hormonal changes to the mucosal vasculature of the respiratory tract lead to capillary engorgement and oedema of the upper airway down to the pharynx, false cords, glottis and arytenoids (*Tonguc et al., 2011*).

2: Physiological changes

Increased progesterone levels mediate many of the physiological changes in the respiratory system. Airway resistance is reduced due to the progesterone-mediated bronchial and tracheal smooth muscle relaxation (*Tonguc et al., 2011*).

Gastro intestinal Changes

Aspiration of gastric contents is an important cause of maternal morbidity and mortality in association with general anaesthesia. Heartburn can affect up to 80% of ladies at term and the supine position may exacerbate the reflux (*Arikan et al., 2011*).

Parturients should be considered to have a "full stomach" with increased risk of aspiration during most of gestation. There is common practice to premedicate females at risk of undergoing cs with an H₂ receptor block such as ranitidine and a prokinetic agent such as metoclopramide. For patients requiring a general anaesthetic, sodium citrate is given to neutralize gastric acid prior to a rapid sequence induction (*Arikan et al., 2011*).

Hepatic Changes

Changes in liver function such as high normal or elevated levels of GGT, ALT, AST and LDH are clinically insignificant.

Clinical signs of liver disease such as spider naevi and palmar erythema may occur during normal pregnancy, making the diagnosis of liver disease more difficult.

The pregnant patient is more inclined to develop gallstones as hormonal changes suppress the release of cholecystokinin and reduce the contractile response of the gallbladder (*Hudić et al., 2011*). Renal Physiological Changes.

The increased blood volume and COP cause the renal blood flow and glomerular filtration rate (GFR) to increase progressively during pregnancy and both are 50-60% higher at term. The increased clearance of urea, creatinine, urate and excretion of bicarbonate results in lower plasma levels than in the non-pregnant population. Mild glycosuria and/or proteinuria can occur in normal pregnancy because the increase in GFR may overwhelm the renal tubules ability to reabsorb glucose and protein (*Wahabi et al., 2011*).

Endocrinal Changes

In spite of increased insulin production, pregnancy is associated with insulin resistance caused predominantly by