Anesthetic Management of Pregnant Cardiac Patients Undergoing Cardiac Surgical Correction

An Essay

Submitted for Partial Fulfillment of Master Degree in Anesthesia

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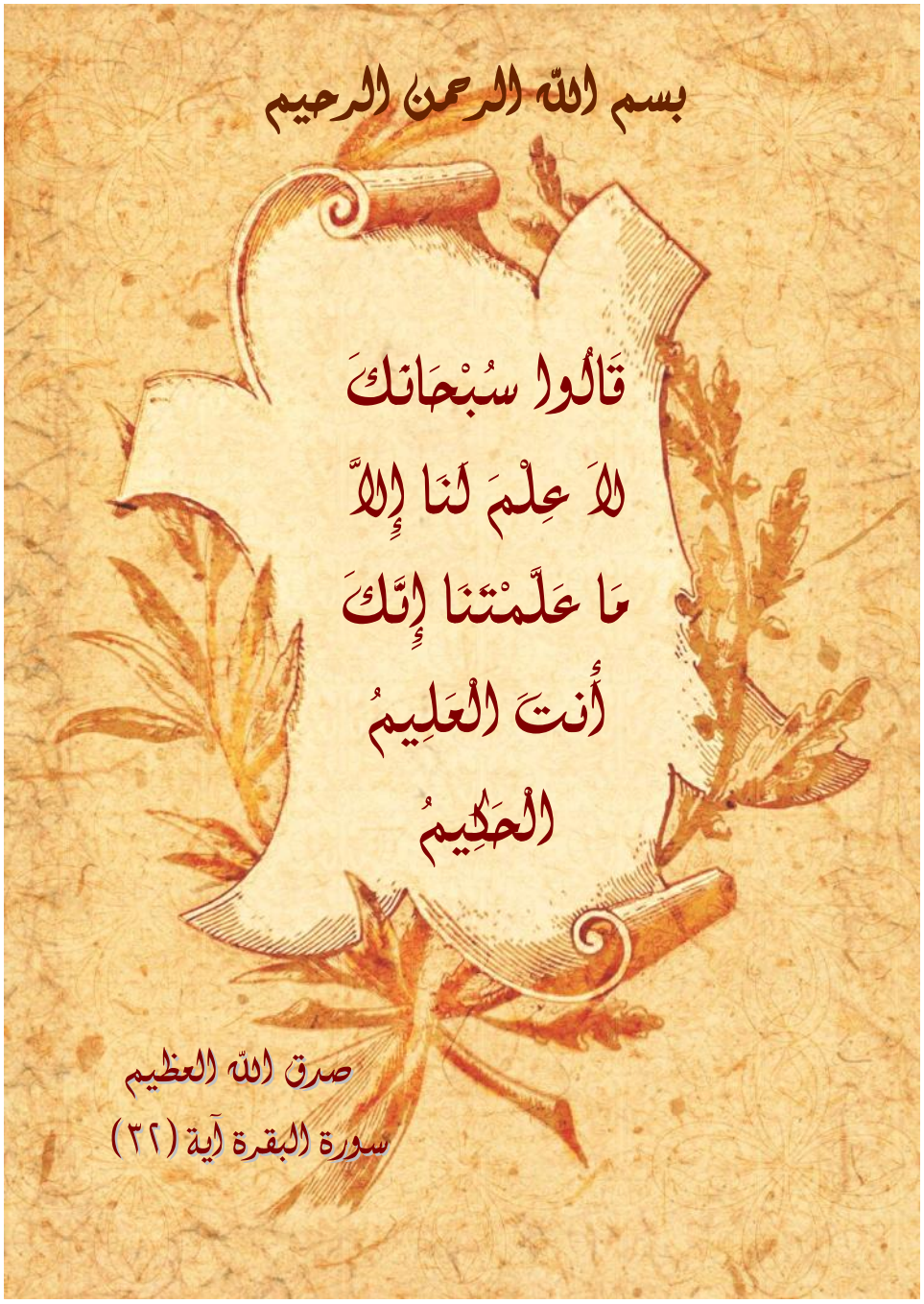
بسم الله الرحمن الرحيم
قَالُوا سُبْحَانَكَ
لاَ عِلْمَ لَنَا إِلاَّ
مَا عَلَّمْتَنَا إِنَّكَ
أَنتَ الْعَلِيمُ
الْحَكِيمُ
صدق الله العظيم
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List of Abbreviations

ACE	Angiotensin converting enzyme
ACT	Activated clotting time
ADP	Adenosine diphosphate
AF	Atrial fibrillation
AKI	Acute kidney injury
ALS	Advanced life support
AMI	Acute myocardial infarction
ANP	Atrial natriuretic peptide
ARDS	Acute respiratory distress syndrome
ASD	Atrial septal defect
ATLS	Advanced trauma life support
AV	Atrio-ventricular
BNP	Brain natriuretic peptide
bpm	Beat per min
CABG	Coronary artery bypass grafting
CK-MB	Creatine kinase MB isoenzyme
CNS	Central nervous system
CO	Cardiac output
CO_2	Carbon dioxide
COPD	Chronic obstructive pulmonary disease
CPB	Cardiopulmonary bypass
CRP	C-reactive protein
CVP	Central venous pressure
DDAVP	1-deamino-8-D-arginine vasopressin
DVT	Deep venous thrombosis
EACA	Epsilon-aminocaproic acid
ECG	Electrocardiogram

ECG	Electrocardiographic
FEV_1	Forced expiratory volume in 1 second
FHR	Fetal heart rate
FiO_2	Fraction of inspired oxygen
GFR	Glomerular filtration rate
GI	Gastrointestinal
GP IIb/IIIa	Glycoprotein IIb/IIIa
HF	Heart failure
ICU	Intensive care unit
IM	Intramuscular
IV	Intravenous
LA	Left atrial dimension
LCOS	Low cardiac output syndrome
LV	Left ventricle
LVd	Left ventricular diastolic dimension
LVEF	Left ventricular
MAC	Minimum alveolar concentration
MAP	Mean arterial pressure
N_2O	Nitrous oxide
NIBP	Non-invasive blood pressure
NO	Nitric oxide
NSC	No significant change
NYHA	New York Heart Association
OPCAB	Off-pump coronary artery bypass
PA	pulmonary artery
$PaCO_2$	Arterial carbon dioxide tension
PaO_2	Arterial oxygen tension
PCI	Percutaneous coronary intervention
PCO_2	Carbon dioxide partial pressure

PCWP	Pulmonary capillary wedge pressure
PDA	Persistent ductus arteriosus
$PETCO_2$	End-tidal CO_2 partial pressure
PFO	Patent foramen ovale
PGI_2	Prostacyclin
PO_2	Oxygen partial pressure
PVCs	Premature ventricular complexes
PVR	Pulmonary vascular resistance
RA	right atrial dimension
RBCs	Red blood cells
RV	Right ventricle
RVd	Right ventricular diastolic dimension
rVIIa	Recombinant factor VII
SD	Standard deviation
STS/SCA	The Society of Thoracic Surgeons and The Society of Cardiovascular Anesthesiologists
SV	Stroke volume
SVR	Systemic vascular resistance
TA	Tranexamic acid
TCD	Transcranial Doppler
TEE	Transesophageal echocardiography
TRALI	Transfusion-related acute lung injury
UBF	Uterine blood flow
VADs	Ventricular assist devices
VC	Vital capacity
VSD	Ventricular septal defect
VT	Ventricular tachycardia

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Introduction

Pregnancy produces numerous physiological adaptative changes on the female body. All of these physiological changes create a greater demand on the cardiovascular system during pregnancy, compared to the nonpregnant state. These changes are necessary to allow the maternal body to cope with the new and increased metabolic demands represented by the fetoplacental unit ***(Souza & Elias, 2001).***

Cardiovascular disease is an important non-obstetric cause of maternal and fetal morbidity and mortality during pregnancy. For a pregnant woman with cardiac disease, the potential inability of the maternal cardiovascular system to contend with normal pregnancy-induced physiologic changes may produce deleterious effects on both mother and fetus ***(Mahoori et al., 2007).***

The pregnant woman with cardiac disease even if well compensated, can be affected by heart failure in face of the increased cardiorespiratory requirements during pregnancy. Medical therapy is not always sufficient to drive a heart with a reduced functional reserve or acute complications, in a pregnant woman. Generally speaking, if the clinical conditions of a pregnant woman with cardiac disease deteriorates despite maximal medical therapy, surgical correction is the only alternative to restore cardiovascular function, and create conditions for the pregnancy to evolve normally ***(Souza & Elias, 2001).***

However, the pregnant state is not optimal for cardiac surgery as the principal interest of the mother and the fetus is different. Cardiac surgery should be reserved only for saving the patient's life when medical therapy proves insufficient or when conservative management leads to acute heart failure ***(Aranyosi et al., 2008).***

Pregnant patients who require cardiac surgery present conflicting issues for perioperative management, and creative compromise is required to meet the best interests of mother and fetus ***(Mahoori et al., 2007).***

Cardiac surgery with extracorporeal support in a pregnant patient constitutes a more complex endeavor, because it represents the sum of anesthetic, surgical and cardiopulmonary bypass (CPB) effects on two individuals under biologically distinct situations, the maternal and the fetal beings. It is not unusual for a cardiac team to be required to manage opposite or conflicting interests between both of those organisms ***(Souza & Elias, 2001).***

Heart surgery with CPB involves certain pathophysiologic effects, such as hypothermia, hemodilution, inhibition of coagulation, hemolysis, complement activation, and non-pulsatile flow, as well as acid-base changes that affect the uteroplacental circulation and fetus ***(Sullivan, 1995)***. The multidisciplinary approach, correct risk assessment, diagnosis, operative indication, timing along with appropriate anesthesia, extracorporeal circulation and alert monitoring of the uterine activity and fetal heart rate

patterns make the intervention technically safe ***(Aranyosi et al., 2008).***

Modern neonatal therapy can optimally care for a premature newborn. This favors a cesarean section prior to the start of CPB when gestational age is superior to 28 weeks in order to eliminate any deleterious effect of CPB on the fetus and thus contribute to reduce fetal morbidity and mortality ***(Souza & Elias, 2001).***

Aim of the Essay

The aim of this essay is to review the principal aspects of anesthetic management of pregnant cardiac patients undergoing cardiac surgery that can be associated with better results regarding maternal and fetal morbidity and mortality.

Physiological Changes during Pregnancy

Pregnancy is a complex physiological condition that involves the integration of a variety of regulatory and organ systems ***(Granger, 2002)***.

Cardiovascular system:

Normal pregnancy is associated with marked hemodynamic alterations within the maternal circulation, including increases in cardiac output and plasma volume and reductions in vascular resistance and arterial pressure. Associated with these changes are marked alterations in the activity of various neurohumoral systems and in vascular and endothelial function ***(Granger, 2002)***.

Blood volume:

Blood volume begins to increase in week 6 of gestation and by the end of pregnancy it will have reached approximately 50% more than in the prepregnant state ***(Silversides & Colman, 2007)***.

Most of the added volume of blood 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 pregnant woman ***(Ciliberto & Marx, 1998)***.

A number of mechanisms are postulated for the hypervolemia of pregnancy. Estrogen increases renin levels and causes sodium retention and an increase in total body water. Other hormones, such as prolactin, placental lactogen, prostaglandins and growth hormone,

are increased during pregnancy and may contribute to fluid retention ***(Silversides & Colman, 2007)***.

Blood constituents:

Red cell mass increases as much as 40% above pre-pregnancy levels. However, the plasma volume increase is proportionally greater than the increase in red blood cell mass, and the resulting hemodilution explains the so-called 'physiological anemia of pregnancy' ***(Silversides & Colman, 2007)***.

Leukocyte counts are variable during gestation, but usually remain within the upper limits of normal. Marked elevations, however, develop during and after parturition. Fibrinogen, as well as total body and plasma levels of factors VII, X and XII increases markedly. The number of platelets also rises, yet not above the upper limits of normal. Combined with a decrease in fibrinolytic activity, these changes tend to prevent excessive bleeding at delivery. Thus, pregnancy is a relatively hypercoagulable state, but during pregnancy neither clotting nor bleeding times are abnormal ***(Ciliberto & Marx, 1998)***.

Cardiac output:

Cardiac output increases by about 30–50%, with the first increase noted as early as week 5 of gestation and reaching a peak at approximately the end of the second trimester or later in the third trimester ***(Silversides & Colman, 2007)***.

The increase in CO is due to increased stroke volume (SV) – up to 30% above baseline – in the first half of pregnancy. This is in contrast to the latter half of pregnancy when CO is maintained by an increase in heart rate – up to 15% above baseline – in addition to the increased SV ***(Carvalho & Jackson, 2008)***.

Heart rate and rhythm:

Heart rate usually increases by 10–20 beats over the course of pregnancy, peaking in the late second trimester or early third trimester. Most women remain in sinus rhythm during pregnancy; however, premature atrial and ventricular complexes may become more frequent. The frequency of new-onset supraventricular arrhythmias and even ventricular tachycardia has been shown to increase during pregnancy. Furthermore, pregnancy may increase the frequency of supraventricular and ventricular arrhythmias in women with a prior history of such arrhythmias ***(Silversides & Colman, 2007)***.

Systemic vascular resistance (SVR):

During pregnancy, there is a fall in systemic (peripheral) vascular resistance beginning in week 5 of gestation with a nadir between weeks 20 and 32. After week 32 of gestation, the SVR slowly increases until term. There is a corresponding initial decrease in the systemic arterial pressure, which begins in the first trimester and reaches its nadir at mid-pregnancy ***(Duvekot et al., 1993)***. Thereafter, systemic pressure begins to increase again and ultimately