New Trends in Anesthetic Management of Cardiac Pregnant Patient Undergoing Elective Caesarean Section

An Essay
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Mostafa Mohamed Awad Noor

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List of Abbreviations

AAGBI	:	Association of Anaesthetists of Great Britain and Ireland
ACC\AHA	:	American College of Cardiology/American Heart Association guidelines
ACCP	:	American College of Chest Physicians
ACEI	:	Angiotensin converting enzyme inhibitor
ACOG	:	American collage of Obestetricians and Gynecologists
AF	:	Atrial fibrillation
AMI	:	Acute myocardial infarction
AR	:	Aortic Regurgitation
ARBS	:	Angiotensin II Recptor antagonist
AS	:	Aortic Stenosis
ASA	:	American Society of Anesthesiologist
ASD	:	Atrial septal defect
BIPAP	:	Bilevel positive airway pressure
BNP	:	Brain natriuretic peptide
BUN	:	Blood urea nitrogen
C\S	:	Ceaserean section
CC	:	Closing Capacity
CEI	:	Continuous epidural Infusion
CK-MB	:	Creatinine Kinase MB fraction
CO_2	:	Carbon dioxide
COP	:	Cardiac output
CPAP	:	Continues Positive Airway Pressure
CRP	:	C reactive protein
CSE	:	Combined Spinal Epidural
CSF	:	Cerebro-Spinal Fluid
CVP	:	Central Venous Pressure
CXR	:	Chest x-ray
Da	:	Dalton
ECG	:	Electro-Cardio-Gram
EF	:	Ejection Fraction

List of Abbreviations (Cont.)

ER	: Emergancy Room
ESC	: European Society of Cardiology
FDA	: Food and Drug Administration
FHR	: Fetal Heart Rate
FRC	: Functional Residual Capacity
GA	: General Anesthesia
GTN	: Glyceryltrinitrate
HDU	: High dependency unit
HR	: Heart Rate
ICP	: Intra Cranial Pressure
ICU	: Intensive Care Unit
IHD	: Ischemic Heart Disease
IHOC	: Idiopathic hypertrophic obstructive cardiomyopathy
IM	: Intra asccular
IV	: Intra Venous
LA	: Left atrium
LDH	: Lactate Dehydrogenase
LES	: Lower esophageal sphincter
LV	: Left Ventricle
LVEF	: Left Ventricle Ejection fraction
LVH	: Left Ventricle hypertophy
LVOT	: Left Ventricle outflow tract
MAC	: Minimal Alveolar Concentration
MAP	: Mean Arterial Pressure
METs	: Metabolic Equvilant Testes
MI	: Myocardial Infarction
MR	: Mitral Regurgitation
MS	: Mitral Stenosis
NIPPV	: Noninvasive positive-pressure ventilation
NSAIDs	: Non Steriodal Anti inflammatory Drugs
NYHA	: New-York Heart Association
PAI	: Plasminogen Activator Inhibitor

List of Abbreviations (Cont.)

PAPP-A	: Pregnancy-associated plasma protein-A
PCA	: Patient Controlled Analgesia
PCEA	: Patient Controlled Epidural Analgesia
PCI	: Percutaneous coronary intervention
PCO_2	: Partial CO_2 tension
PCWP	: Pulmonary capillary wedge pressure
PEEP	: Positive End Expiratory Pressure
PND	: Paroxysmal Nocturnal Dyspnea
PO_2	: Partial O_2 tension
PPCM	: Peripartum Cardiomyopathy
PS	: Pulmonary Stenosis
RA	: Regional Anesthesia
RCRI	: Revised Cardiac Risk Index
RSI	: Rapid Sequence Induction
RV	: Right ventricle
SCLMWH	: Subcutaneous Low Molecular Weight Heparin
SVR	: Systemic vascular resistance
TAP	: Transversus abdominus plain
TLC	: Total Lung Capacity
TPA	: Tissue Plasminogen Activator
TTE	: Transthoracic Echocardiography
UES	: Upper Esophageal Sphincter
UFH	: Un-fractionated Heparin
UV\MV	: Umbilical Vein to maternal vein concentration ratio
VD\VT	: Dead space to tidal volume ratio
VHD	: Valvular Heart Disease

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Introduction

Pregnancy makes a significant demand on the cardiovascular system. Therefore, it follows that women with cardiovascular compromise due to cardiac disease need special input and careful management peripartum. Cardiac disease was the most common cause of indirect maternal deaths and the most common cause of death overall. In particular, during ٢٠٠٣-٥, there was an increase in deaths due to myocardial infarction, thoracic aortic dissection, and rheumatic mitral stenosis (***Lewis,*** ***٢٠٠٧***).

Ischemic heart disease is also seen more commonly today due to both the increasing number of women of advanced maternal age who are electing to undergo pregnancy and childbirth as well as advances in medical therapy for ischemic heart disease, allowing women with this condition to carry a pregnancy to term ***(Goldszmidt et al.,*** ***٢٠١٠***).

In general regurgitant valvular lesions are well tolerated during pregnancy, where stenotic lesions have a greater potential for decompensation. Pregnant patients with valvular heart disease can expect to have worsening of their New York Heart Association (NYHA) functional class, some may develop congestive heart failure while others may have adverse foetal outcome i.e. preterm birth or still birth (***Hameed et al.,*** ***٢٠٠١***).

While the incidence of cardiac disease in pregnant patients has remained relatively unchanged, the maternal mortality from cardiac disease has decreased from ٦٪ in the ١٩٣٠ to ٠,٥-٢,٧٪ today. The last decade has shown a decline in maternal mortality from congenital heart disease, and now acquired heart disease has risen to be the leading cardiac cause of maternal death, with myocardial infarction, and cardiomyopathy as the main processes (***Curry et al.,*** ***٢٠٠٩).***

Aim of The Work

The aim of this review is to discuss the current anesthetic management of pregnant patient with common cardiac conditions, presenting for elective caesarean section.

Anatomical, Physiological and Pharmacological Considerations During Pregnancy

The adequate management of pregnant lady with cardiac disease requires the clinician to consider and understand the unique changes in anatomy and physiology that take place during pregnancy. The pathophysiology of cardiac disease in pregnancy may significantly differ from those that commonly occur in the non-pregnant state ***(Rudra et al., ٢٠٠٧)***.

I. Changes in the Cardiovascular System

An increase in cardiac output is one of the most important changes of pregnancy. Cardiac output increases by ٣٠–٤٠٪ during pregnancy, and the maximum increase is attained around ٢٤ weeks' gestation ***(Mashini et al., ١٩٨٧).***

The increase in heart rate occurs first by the end of the first month of pregnancy. Stroke volume increases by midfirst trimester and progressively increases through the second trimester. Echocardiography demonstrates increases in end diastolic chamber size and total left ventricular wall thickness but no change in end-systolic volume, so ejection fraction is increased (***Rudra et al., ٢٠٠٧***).

Cardiac output can vary depending on the uterine size and maternal position at the time of measurement. The enlarged gravid uterus can cause aortocaval compression and reduced cardiac filling while the pregnant woman is in the supine position, leading to an underestimation of cardiac function. Normal pregnant women exhibit a marked increase in femoral venous and inferior vena caval pressures. Collateral vessels maintain atrial filling but lead to engorgement of veins, including the epidural venous (Batson's) plexus. Filling pressures (CVP, pulmonary capillary wedge pressure, LV end-

diastolic pressure) do not change despite the increased cardiac dimensions, due to myocardial remodeling during gestation (***Rudra et al.,*** ٢٠٠٧).

Systemic vascular resistance is decreased approximately ٢٠٪. Blood pressure never increases in normal pregnancy, and systolic and diastolic blood pressures decrease by approximately ٨ and ٢٠٪, respectively, on average**.**Pregnancy hormones (estrogen and progesterone), prostacyclin, and nitric oxide all may play a role in the reduction in blood pressure observed despite an increase in cardiac output. As a result of the fall of systolic and diastolic blood pressures there is a reflex increase in heart rate by ٢٥٪ ***(Rudra et al.,*** ٢٠٠٧).

Cardiac output increases further during labor, up to ٥٠٪ higher than pre-labor values, although effective analgesia can attenuate some of this increase. In the immediate postpartum period, cardiac output increases maximally and can rise ٨٠٪ above pre-labor values and approximately ١٥٠٪ above non pregnant measurements. The heart is displaced to the left and upward during pregnancy because of the progressive elevation of the diaphragm by the gravid uterus ***(Tihtonen et al.,*** ٢٠٠٦***)***.

The electrocardiogram of normal parturients may include sinus tachycardia or benign dysrhythmias, depressed ST segments and flattened T waves, left axis deviation, and left ventricular hypertrophy. Auscultation frequently reveals a systolic murmur of aortic regurgitation, and a third or fourth heart sound. Cardiac output, heart rate, and stroke volume decrease to pre-labor values ٢٤–٧٢ h postpartum and return to nonpregnant levels within ٦–٨ weeks after delivery **(*Tihtonen et al.,*** ٢٠٠٦***).***

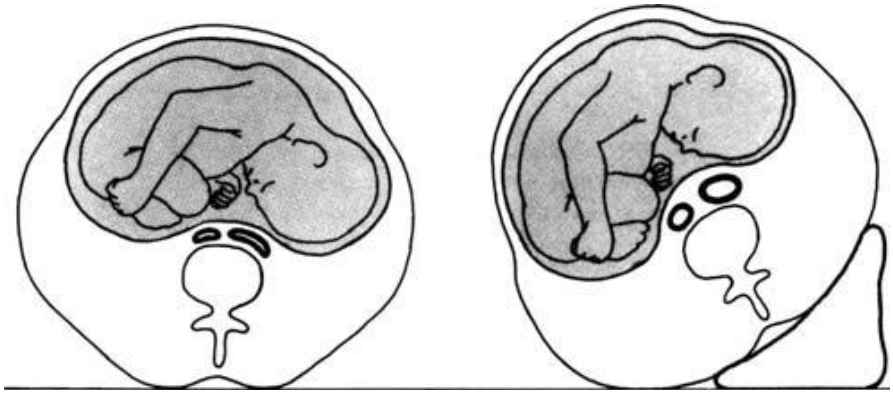

Fig.(١): Aortocaval compression. **(Chang, ٢٠٠٤).**

Clinical Implications

An increased cardiac output might not be well tolerated by pregnant women with valvular heart disease (e.g., aortic or mitral stenosis) or coronary arterial disease. Decompensation in myocardial function can develop at ٢٤ weeks' gestation, during labor, and especially immediately after delivery. Engorgement of the epidural venous plexus increases the risk of intravascular catheter placement in pregnant women; direct connection of the azygos system to the heart as well as brain also increases the risks of local anesthetic cardiovascular and central nervous system toxicity ***(Datta et al., ٢٠١٠).***

II. Changes in the Hematological System

Maternal blood volume increases during pregnancy, and this involves an increase in plasma volume as well as in red cell and white cell volumes. The plasma volume increases by ٤٠–٥٠٪, whereas the red cell volume increases by only ١٥–٢٠٪, which causes a "physiological anemia of pregnancy" (normal hemoglobin ١٢ g/dL; hematocrit ٣٥) ***(Tsuei, ٢٠٠٦).***

Because of this hemodilution, blood viscosity decreases by approximately ٢٠٪ ***(Rudra et al., ٢٠٠٧).***

Blood volume increases further during labor, as uterine contractions squeeze blood out of the intervillious space and into the central circulation. After delivery, involution of the uterus and termination of placental circulation causes an autotransfusion of approximately ٥٠٠ mL of blood. Levels of clotting factor I, VII, VIII, IX, X, and XII and fibrinogen are elevated during pregnancy as well. Platelet production is increased, thrombopoietin levels are increased and platelet aggregation measured in vitro is likewise increased however indices of platelet destruction are also increased ***(Frolich et al., ١٩٩٨).***

Endogenous anticoagulants, such as protein S, are decreased in normal pregnancy and there is acquired resistance to activated protein C during pregnancy, adding to the prothrombotic state. Fibrinolysis is impaired in normal pregnancy due to placentally derived plasminogen activator inhibitor (PAI), but returns to normal following delivery of the placenta. Overall indices of coagulation indicate that normal pregnancy is a hypercoagulable state ***(Sharma et al., ١٩٩٧).***

Clinical Implications

Increased blood volume and enhanced coagulation serve several important functions which are:

١- The increased circulatory needs of the enlarging uterus and growing fetus and placenta are met

٢- The parturient is protected from bleeding at the time of delivery. Anesthesiologists should consider the enlarged blood volume when making decisions on fluid and blood replacement in the peripartum period. Parturients become hypercoagulable as gestation progresses and are at increased risk of thromboembolism. After a rapid mobilization and diuresis of some fluid in the first few postpartum days, blood volume slowly returns to normal over ٨ weeks ***(Datta et al., ٢٠١٠).***

III.Changes in the Respiratory System

Changes in respiratory parameters start as early as the fourth week of gestation. Minute ventilation is increased at term by about ٥٠٪ above nonpregnant values. The increase in minute ventilation is mainly due to an increase in tidal volume (٤٠٪) and, to a lesser extent, an increase in the respiratory rate (١٥٪). Alveolar ventilation is greatly increased as the tidal volume increases without any change in the ratio of dead space to tidal volume (VD/VT) ***(Wise and Polito, ٢٠٠٠)***.

At term PCO_2 is decreased to ٣٢–٣٥ mmHg, although renal excretion of bicarbonate keeps arterial pH normal. Increased progesterone concentrations during pregnancy likely stimulate increased respiration, even before an increase in metabolic rate. Oxygen consumption and carbon dioxide production increase by approximately ٦٠٪ over prepregnant values. PaO_2 is increased in early pregnancy due to a decrease in PCO_2. Functional residual capacity, expiratory reserve volume, and residual volume are decreased at term. These changes are related to the cephalad displacement of the diaphragm by the large gravid uterus ***(Wise and Polito, ٢٠٠٠)***.

Closing capacity (CC) does not change, but the reduction in FRC contributes to a tendency toward earlier desaturation, as lung volume more easily falls below CC ***(Wise and Polito, ٢٠٠٠).***

Anatomic changes also accompany pregnancy. The respiratory mucous membranes become vascular, edematous, and friable. The voice may deepen and there is a progressive increase in the Mallampati score during gestation and labor**.**In labor, minute volume further increases in the absence of pain relief, and PCO^2 may decrease to ١٧ mmHg. Opioids some what attenuate this change, but epidural analgesia does so more completely. In the second stage, maternal expulsive efforts increase ventilation, even in the presence of effective