Anaesthetic Management of Patients with Low Ventricular Ejection Fraction

Essay

Submitted for Partial Fulfillment of Master Degree
in Anaesthesiology

Presented by

Mai Mohamed Othman

M.B,B.Ch
Faculty of Medicine
Ain Shams University

Under Supervision of

Prof. Dr. Gehan Fouad Kamel

Professor of Anaesthesia and Intensive Care
Faculty of Medicine, Ain Shams University

Dr. Yasser Abd El-Rahman Salem

Lecturer of Anaesthesia and Intensive Care
Faculty of Medicine, Ain Shams University

Dr. Amr Ahmad Kassem

Lecturer of Anaesthesia and Intensive Care
Faculty of Medicine, Ain Shams University

Faculty of Medicine
Ain Shams University
2015

Acknowledgement

First of all, my great thanks for **ALLAH**, *the Most Merciful, the Most Gracious, for giving me courage, health and patience to undertake and accomplish this essay and for all his blesses on me in my life.*

I would like to express my deepest thanks, gratitude and respect to my great **Prof. Dr. Gehan Fouad Kamel,** *Professor of Anaesthesia and Intensive Care, Faculty of Medicine, Ain Shams University, for her advices, creative ideas, her constant supervision and support throughout the performance of this work. I had the honor to complete this work under her supervision.*

Words fail to express my profound thanks and sincere gratitude to **Dr. Yasser Abd El-Rahman Salem,** *Lecturer of Anaesthesia and Intensive Care, Faculty of Medicine, Ain Shams University, for his generous supervision, continuous encouragement, unlimited help and continuous guidance throughout this work.*

I can't forget to thank with all appreciation **Dr. Amr Ahmad Kassem,** *Lecturer of Anaesthesia and Intensive Care, Faculty of Medicine, Ain Shams University, for his great efforts and time he has devoted for this work.*

Finally, I will never forget the sincere encouragement and great help of my ***FAMILY*** *throughout my life journey.*

Mai Mohamed Othman

Contents

List of Abbreviations

Abbrev.	Full term

ACC : American College of Cardiology
ACE : Angiotensin converting enzyme
ADP : Adenosine diphosphate
AHA : American Heart Association
ARBs : Angiotensin receptor blockers
ARVC : Arrhythmogenic right ventricular cardiomyopathy
BNP : Brain-type natriuretic peptide
CAD : Coronary artery disease
CCBs : Calcium channel blockers
CO : Cardiac Output
CPET : Cardiopulmonary exercise test
CVP : Central venous pressure
ECG : Electrocardiogram
EDV : End Diastolic Volume
EF : Ejection fraction
ESC : European Society of Cardiology
HF : Heart failure
HYD : Hydralazine
IABP : Intra-aortic balloon pump
ICDs : Implantable cardioverter–defibrillators
ICM : Ischemic cardiomyopathy
IHD : Ischaemic heart disease
ISDN : Isosorbide dinitrate
LV : Left ventricle

LVEF : Left ventricular ejection fraction%

MELAS : Mitochondrial encephalopathy, lactic acidosis and stroke like syndrome

MET : Metabolic equivalent task

NT-proBNP : N-terminal pro-B-type natriuretic peptide

NYHA : New York Heart Association

PAC : Pulmonary artery catheter

PAP : Pulmonary artery pressure

PD : Phosphodiesterase

POISE : Perioperative ischemic evaluation

RCRI : Revised cardiac risk index

RCRI : Revised Goldman cardiac risk index

REMATCH : Randomized Evaluation of Mechanical Assistance for the Treatment of Congestive Heart Failure

RV : Right ventricular

SVR : Systemic vascular resistance

TEE : Transesophageal echocardiography

VHD : Valvular heart disease

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Introduction

The number of patients with heart failure presenting for surgery continues to rise, and anesthesiologists are increasingly being called upon to provide quality, safe care in the operating room for patients with low ejection fraction (EF). Perioperative goals in the management of these patients include maintaining forward flow, promoting inotropy without inducing or exacerbating ischemia, and returning patients to their preoperative level of function after surgery ***(Chua and Nguyen, 2013).***

Oftentimes, these goals can be met with pharmacologic support, including the use of calcium channel blockers, phosphodiesterase inhibitors, and novel agents, such as nesiritide and levosimendan. Many patients with diminished EF have implantable cardioverter-defibrillators (ICDs) in place. Another helpful device is the intra-aortic balloon pump. These devices can serve a critical role in managing patients who have inadequate responses to pharmacologic therapy because of concerns for increasing myocardial work ***(Chua and Nguyen, 2013).***

The LVEF, which is the ratio of stroke volume to LV end-diastolic volume, is the standard clinical measurement of

systolic function. A normal EF indicates that the stroke volume is appropriate for the end-diastolic volume and vice versa. Normally, it is 55-70%, an ejection fraction below 35% the patient may be at risk for life threatening arrhythmias or sudden cardiac death. Low ejection fraction may be caused by heart valve disease, coronary heart disease or cardiomyopathy ***(Iwano and Little, 2013).***

Although aortic stenosis is a common condition associated with major morbidity, mortality, and health economic costs, there are currently no medical interventions capable of delaying or halting its progression. Aortic stenosis is characterized by progressive aortic valve narrowing and secondary left ventricular hypertrophy. Both processes drive the development of symptoms and adverse events that characterize the latter stages of the disease. Inflammation, fibrosis, and calcification lead to progressive valve narrowing and development of left ventricular hypertrophy, subsequent decompensation, and the transition to heart failure ***(Dweck et al, 2012).***

Coronary artery disease (CAD) is an increasingly important medical and public health problem, and is the leading cause of mortality. The underlying pathophysiology is poorly understood. Genetic predisposition, high prevalence

of metabolic syndrome and conventional risk factors play important role. Lifestyle related factors, including poor dietary habits, excess saturated and trans fat, high salt intake, and low-level physical activity may be important as well. Some novel risk factors, including hypovitaminosis D, arsenic contamination in water and food-stuff, particulate matter air pollution may play unique role ***(Islam and Majumder, 2013).***

Cardiomyopathies are defined as cardiac diseases of the myocardium with associated cardiac dysfunction. They are cardiac diseases in which heart muscle disease and/or measurable deterioration of cardiac muscle function occurs due to various causes, such as genetic and sporadic mutations of muscle proteins, as well as external factors such as hypertension, ischemia, and inflammation ***(Sanbi, 2013).***

Stress due to surgery leads to an increase in cardiac output which can be achieved by normal patients but which results in substantial morbidity and mortality in those with cardiac disease. These present difficult anesthetic challenges and place these patients at a high risk of perioperative morbidity and mortality ***(Sanders et al, 2009).***

The anesthesiologist should be knowledgeable about the pathophysiology underlying low ejection fraction and the appropriate perioperative management. Whether presenting for cardiac or general surgery, and to modify the care plan accordingly. With a directed preoperative assessment that focuses on certain aspects of the cardiovascular system, and the assistance of powerful investigating tools such as tissue Doppler, this can be achieved ***(Sanders et al, 2009).***

Aim of the Work

This study aims to describe the optimal way for anesthetic management of patients with low ventricular ejection fraction.

Chapter (1):
Anatomical and Physiological Background of Low Ejection Fraction

The heart provides the driving force for delivering blood throughout the cardiovascular system to supply nutrients and remove metabolic waste. Because of the complexity of RV anatomy, the traditional description of systolic function is usually limited to the LV. Systolic performance of the heart is dependent on loading conditions and contractility. Preload and afterload are two interdependent factors extrinsic to the heart that govern cardiac performance ***(Sun and Schwarzenberger, 2012).***

Preload and Afterload

Preload is defined as the ventricular load at the end of diastole, before contraction has started. First described by Starling, a linear relationship exists between sarcomere length and myocardial force ***(Berne and Levy, 2001).***

In clinical practice, surrogate representatives of LV volume such as pulmonary wedge pressure or central venous pressure are used to estimate preload. With the development of transesophageal echocardiography, a more direct measure of ventricular volume is available ***(Katz, 2001).***

Afterload is defined as systolic load on the LV after contraction has begun. Aortic compliance is an additional determinant of afterload. Aortic compliance is the ability of the aorta to give way to systolic forces from the ventricle. Changes in the aortic wall (dilation or stiffness) can alter aortic compliance and thus afterload ***(Berne and Levy, 2001).***

Examples of pathologic conditions that alter afterload are aortic stenosis and chronic hypertension. Both impede ventricular ejection, thereby increasing afterload. Aortic impedance, or aortic pressure divided by aortic flow at that instant, is an accurate means of gauging afterload. However, clinical measurement of aortic impedance is invasive ***(Katz, 2001).***

Echocardiography can estimate aortic impedance noninvasively by determining aortic blood flow at the time of its maximal increase. In more general clinical practice, measurement of systolic blood pressure is adequate to approximate afterload, provided that aortic stenosis is not present ***(Takayama et al, 2002).***

Preload and afterload can be thought of as the wall stress that is present at the end of diastole and during LV ejection, respectively. Wall stress is a useful concept because it includes preload, afterload, and the energy required to generate contraction. Wall stress and heart rate are probably

the two most relevant indices that account for changes in myocardial oxygen demand. The law of Laplace states that wall stress (σ) is the product of pressure (P) and radius (R) divided by wall thickness (h)[3]:

$$\sigma = P \times R/2h$$

The ellipsoid shape of the LV allows the least amount of wall stress such that as the ventricle changes its shape from ellipsoid to spherical, wall stress is increased. By using the ratio of the long axis to the short axis as a measure of the ellipsoid shape, shown in figure (1), a decrease in this ratio would signify a transition from ellipsoid to spherical ***(Frank, 1895).***

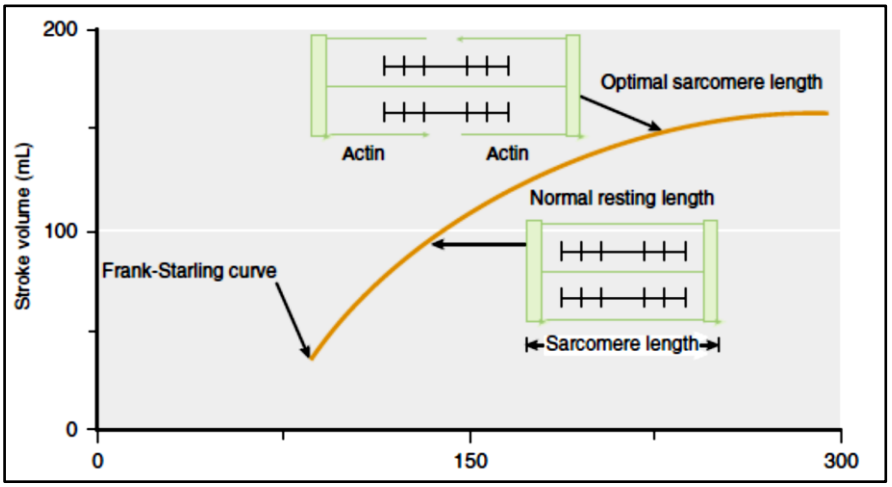

Figure (1): Frank-Starling relationship. The relationship between sarcomere length and tension developed in cardiac muscles is shown. In the heart, an increase in end-diastolic volume is the equivalent of an increase in myocardial stretch; therefore, according to Starling's law, increased stroke volume is generated ***(Frank, 1895).***