# UPDATES IN PERIOPERATIVE MANAGEMENT OF SECONDARY PULMONARY HYPERTENSION DURING MITRAL VALVE SURGERIES

An essay submitted for fulfillment

Of master degree MSc in ANESTHESIOLOGY

By

# RAMI MAGDI RAMZI

**MBBCH** 

Under supervision of

## Dr. MAHER FAWZY MAHMOUD

Professor of anesthesia

Faculty of medicine Cairo University

# Dr. DINA SOLIMAN MOHAMED IDRIS

Assist.Professor of anesthesia

Faculty of medicine Cairo University

# Dr. MOHAMED ALI BAKRY

Lecturer of anesthesia

Faculty of medicine Cairo University

#### **Abstract**

Patients with long standing mitral valve disease are at risk of developing pulmonary hypertension, which may present a formidable challenge for the cardiac anesthetist (perioperatively) during cardiac surgery. Pulmonary hypertension is an important risk factor for the development of acute right sided heart failure during cardiac surgery. Even with early and adequate therapy, right ventricular (RV) failure is associated with increased morbidity and mortality. Adequate treatment of RV failure consists of different strategies. The main goal is to decrease RV afterload by using vasodilating agents

Key Word

Rv

Mitral Valve Surgeries

Secondary Pulmonary Hypertension

Anesthesiology

## **AKNOWLEDGEMENT**

I would like to thank my supervisors:

Professor Dr Maher Fawzy Mahmoud
Assistant Professor Dr Dina Soliman
Lecturer Dr Mohammed Bakry

For their great care and support during our work for fulfillment of my Master degree .

# Contents

List of abbreviations	i
List of figures	·iv
List of tables	V
Introduction	1
Anatomy and physiology	2
Pathophysiology and management of	17
pulmonary hypertension	
Echocardiography in the operating room	38
Anesthetic management of pulmonary hypertension	49
Summary	73
References	- 75
Arabic summary	Í

#### List of abbreviations

**ASE** American Society Of Echocardiography

**cAMP** Cyclic Adenine Mono Phosphate

**cGMP** Cyclic Guanine Mono Phosphate

CI Cardiac Index

**CO** Cardiac Output

**COPD** Chronic Obstructive Pulmonary disease

**CPB** Cardio Pulmonary Bypass

**CVP** Central Venous Pressure

**EF** Ejection Fraction

**EtA** Endothelin-1 receptor type A

**EtB** Endothelin-1 receptor type B

**GTN** Glyceryl Tri-Nitrate

**HPV** Hypoxic Pulmonary Vasoconstriction Reflex

**HR** Heart Rate

IOTEE Intra Operative Trans Esophageal Echo

**Kg** Kilogram

**L** Liter

**LV** Left Ventricle

**LA** Left Atrium

**LVOT** Left Ventricle Outflow Tract

MAP Mean Arterial Pressure

ml Milliliter

mmHg Millimeter Mercury

MPAP Mean Pulmonary Artery Pressure

MR Mitral Regurge

MS Mitral Stenosis

MS/MR Double Mitral

NO Nitric Oxide

**NYHA** New York Heart Association

PA Pulmonary Artery

**PAH** Pulmonary Artery Hypertension

PaO2 Arterial O2 tension

PAP Pulmonary Artery Pressure

PASP Pulmonary Artery Systolic Pressure

**PCWP** Pulmonary Capillary Wedge Pressure

PDEI III Phospho Di-esterase Enzyme Inhibitor III

**PDEI V** Phospho Di-esterase Enzyme Inhibitor V

PH Pulmonary Hypertension

**PPH** Primary Pulmonary Hypertension

**PVOD** Pulmonary Veno-Occlusive Disease

PVR Pulmonary Vascular resistance

RAP Right Atrial Pressure

**RV** Right Ventricle

SaO2 Arterial Oxygen Saturation

**SPAH** Secondary Pulmonary Hypertension

**SV** Stroke Volume

**SVR** Systemic Vascular Resistance

**TEE** Trans Esophageal Echocardiography

TTE Trans Thoracic Echocardiography

WHO World Health Organization

# **List Of Figures**

	Page
Figure 1- The Pulmonary Circulation	4
Figure 2- Responses of the right and left ventricles to experimental	10
increase in afterload	
Figure 3- The effect of cardiac output on MPAP	14
Figure 4- Disease progression in pulmonary arterial hypertension	20
Figure 5- Chemical structure of sodium nitroprusside	23
Figure 6- Chemical structure of sildenafil citrate	32
Figure 7- Mechanism of action of sildenafil	33
Figure 8- Conventions of image display followed in TEE guidelines	41
Figure 9- 20 cross- sectional views composing the recommended	44
comprehensive TEE examination	
Figure 10- Short axis drawing of the mitral valve illustrating how	45
it is transected by mid esophageal views	
Figure 11- Flail posterior mitral valve leaflet (PMVL) as mechanism	47
Of mitral regurgitation	
Figure 12- Large paravalvular leak detected by color Doppler	48
echocardiography in a bioprothetic mitral valve	
Figure 13- TTE, Top: enlarged right side chambers	56
Bottom:severe PH	



# **List Of Tables**

	Page
Table 1- Revised clinical classification of pulmonary hypertension	18
Table 2- Recommended trans-esophageal echocardiography	42
cross sections	
Table 3- ASA physical status	50
Table 4- functional assessment of patient with	51
pulmonary hypertension	
Table 5- Clinical characteristics associated with increased	52
morbidity and mortality in PH patients	

### Introduction

Patients with long standing mitral valve disease are at risk of developing pulmonary hypertension, which may present a formidable challenge for the cardiac anesthetist (perioperatively) during cardiac surgery<sup>(1)</sup>. Pulmonary hypertension is an important risk factor for the development of acute right sided heart failure during cardiac surgery<sup>(2)</sup>.

Even with early and adequate therapy, right ventricular (RV) failure is associated with increased morbidity and mortality<sup>(3)</sup>. Adequate treatment of RV failure consists of different strategies. The main goal is to decrease RV afterload by using vasodilating agents <sup>(4)</sup>.

Strategies to reduce pulmonary vascular tone aim to enrich vascular smooth muscle cyclic adenosine monophosphate (cAMP) levels through beta agonists (e.g. isoproterenol) or with phosphodiesterase type III inhibitors (e.g. milrinone). Oral Sildenafil is a selective vasodilator that prolongs the action of cyclic guanosine monophosphate (cGMP) by selective inhibition of phosphodiesterase (PDE) type 5, of which the lung has a rich supply. It has been shown to be very effective in lowering pulmonary artery pressure Furthermore, its effects have been noted to last for at least three hours without affecting systemic arterial pressure (5,6).

Alternatively, increasing cyclic guanine monophosphate (cGMP) with nitroso vasodilators (sodium nitroprusside, nitroglycerin, inhaled nitric oxide [NO]) also reduces pulmonary vascular tone <sup>(7)</sup>.

## **Functional Anatomy**

## The Right ventricle:

"Two souls with but a single thought, two hearts that beat as one" (8), Although the right and left ventricles develop from the same primitive heart tube during morphogenesis, they evolve into two relatively independent structures with so many different characteristics that with some justification they may be regarded as two different organs. Despite that, they are closely linked physically, mechanically, and electrically and appear to "beat as one" (9).

The shape of each ventricle is genetically determined to suit its exact function. Thus, the left ventricle is "flask" shaped with the inlet and outlet sharing one orifice. This enables the ventricle to deliver a bolus of blood against high resistance <sup>(10)</sup>.

In contrast the right ventricle consists of a flattened tube wrapped around the left ventricle with separate inlet and outlet orificies and a presumed contraction pattern simulating peristalsis. Such an arrangement is suited for pumping blood against low resistance (11).

The right ventricle occupies a large triangular part of the anterior surface of the heart and extends from the right atrium almost to the apex. Superiorly the part of the right ventricle called the conus arteriosus or infundibulum joins the pulmonary trunk. Inferiorly its wall forms the acute margin of the heart and extends for some distance around the diaphragmatic surface <sup>(12)</sup>.

The right ventricular chamber can be divided to a main chamber (the inflow tract) and the infundibular portion (the outflow tract).

The main chamber has a triangular shape on the longitudinal axis and a crescentic shape in the horizontal axis. This peculiar shape results in a large surface area relative to the intercavitary volume, rendering the right ventricle well suited to eject a large volume of blood with little fiber shortening, Pumping action of the right ventricle has been compared to that of a bellows working in series with a low pressure circuit (13).

The main chamber is limited by two walls: a lateral, thin, concave, free ventricular wall and a medial, thick, convex, interventricular septal wall.

The right ventricular wall is about one third the thickness of that forming the left ventricle, but their capacities are the same, about 85ml.

The tricuspid valve encircles the right atrioventricular orifice and is notched into 3 triangular cusps named anterior, posterior and septal. It measures about 4 cm in diameter and is large enough to admit the tips of four fingers. The free border of each cusp presents a ragged edge for the attachment of chordae tendineae. In addition to their attachment to the cusps the chordae are secured to the papillary muscles in the ventricle<sup>(12)</sup>.

The right ventricular outflow tract includes the infundibulum, the pulmonary valve, and the main pulmonary artery.

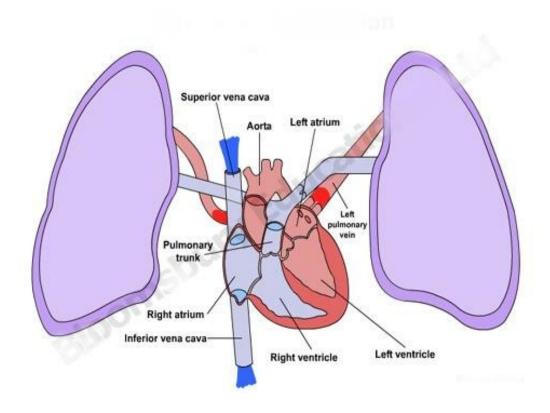


Figure 1: The Pulmonary circulation (12).

## The pulmonary artery

Arises from the infundibulum of the right ventricle at the pulmonary orifice. Its 5 cm in length and 3 cm wide in diameter, at about the level of fibro-cartliage between fifth and sixth thoracic vertebrae, the pulmonary trunk divides into right and left pulmonary arteries which are of nearly equal size<sup>(12)</sup>.

The right pulmonary artery passes under the arch of the aorta more or less horizontally and before entering the hilum divides into a superior division (which supplies the upper lobe) and the continuation of the main trunk. The superior division which is quite prominent and is some times referred to as the truncus anterior, lies in front of the right upper lobe bronchi and its branches follow those of the airways. The lower division proceeds downwards, lying in front of the intermediate and lower lobe bronchi passing outside the middle lobe bronchus <sup>(14)</sup>.

The left pulmonary artery takes a backward and upward course. It lies about 1cm higher than the right pulmonary artery. The remains of the ductus arteriosus of the neonate connect the left pulmonary artery to the arch of the aorta above. The artery divides into a short superior division, which promptly divides into branches supplying the upper lobe. The inferior division hooks backwards over the top of the upper lobe bronchus and continues downwards and backwards lateral to and a little behind the lower lobe bronchus. In doing so it forms a vascular arch, which is seen on a lateral radiograph as a smaller curved shadow lying below that of the aorta (14).

The distribution of the pulmonary arteries within the lobes of the lungs, although broadly following the branching pattern of the bronchi, shows considerable variation.

The pulmonary arterial vessels convey deoxygenated blood from the right ventricle of the heart to the lungs, consistent with the relatively thin wall of the right ventricle in comparison to that of the left ventricle; the pulmonary arterial vessels also have walls only one-third the thickness of vessels of comparable size in the systemic arterial circulation (12).