Assessment of Right Ventricular Functions during One Lung Ventilation for Lobectomy guided by Transesophageal Echocardiography

Thesis submitted for partial fulfillment of M.D.
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2005

Abstract

Background: Pulmonary resection and one-lung ventilation have a variable effect on pulmonary hemodynamics and right ventricular functions. Such intraoperative hemodynamics is classically monitored using invasive techniques. The use of TEE in hemodynamic monitoring was recently introduced as a category II indication according to ASA/SCA guidelines.

Objectives: We applied intraoperative TEE to study changes in pulmonary hemodynamics and right ventricular functions during one-lung ventilation and lobectomy.

Methods: Thirty patients with mild to moderate impairment of pulmonary functions, no echocardiographic signs of pulmonary hypertension, with normal cardiac functions and undergoing lobectomy were studied. TEE was used to assess systolic pulmonary artery pressure (SPAP) by measuring peak flow of tricuspid regurge, in those we could not obtain a reliable tricuspid regurge wave(12 patients)we calculated the mean pulmonary artery pressure (MPAP) from time intervals in the systolic pulmonary artery flow. Two recently introduced methods were used to measure the pulmonary vascular resistance (PVR) and right ventricular functions (Tei index) using Doppler-derived time intervals, we also measured right ventricular fractional area change(FAC) and cardiac output (CO) using pulmonary artery velocity time integral (PAVTI) and pulmonary artery diameter.

Results: The results show a mild but statistically significant increase in SPAP in 12 patients (from 23.o4±2.9 to 27.64±4.47mmHg) and MPAP in the rest of patients(from 18.33±2.8 to 22.2±3.1mmHg)with thoracotomy and one-lung ventilation, CO also showed a statistically significant increase(from 4.1±1.03 to 5.11±0.92mmHg)during the same period. A further mild increase in SPAP(from 27.64±4.47 to 28.79±4.36mmHg)and MPAP(from 22.2±3.1 to 23.57±2.5mmHg)with the end of resection, this was associated with statistically significant increase in PVR at the same stage(from148.6±37.5to179.7±47.3dyne.sec.cm-5).RVFAC and Tei index showed no change at any time. All hemodynamic parameters returned to near normal values at the end of the operation.

Conclusion: Mild changes in pulmonary hemodynamics occur with lobectomy in patients with mild to moderate impairment in pulmonary functions, these changes return to normal at the end of surgery, with no effect on right ventricular functions. TEE can be highly informative in monitoring pulmonary hemodynamics and right ventricle. Tei index is a simple, reproducible method to assess right ventricular functions. Using systolic time intervals of pulmonary flow to assess PVR is a new, obtainable method.

Key words:

One-lung ventilation, Lobectomy, Right ventricular functions, Pulmonary pressure, Pulmonary vascular resistance, Transesophageal echocardiography, Pulmonary flow time intervals,Tei index.

Acknowledgment

First of all thanks to Allah, without his help this work would have never existed.

I would like to express my sincere thanks and deep gratitude to Professor Dr. Fawzia Abo El Fotouh, Professor of anesthesiology, Faculty of medicine, Cairo University, for her kind guidance and generous help not only during this work but all through my career, she has been a real inspiration to everyone worked with her, I thank her for being an idol for all of us.

I wish also to express my extreme appreciation and respect to Dr. Hisham Khedr, Assistant Professor of Anesthesiology, Faculty of medicine, Cairo University, for his faithful supervision, constructive guidance and real interest in the progress of this work.

Many sincere thanks are expressed to Dr. Ahmad El Agaty, Assistant Professor of Anesthesiology, Faculty of medicine, Cairo University for his kind advice, great help and unlimited encouragement.

Special thanks for Dr. Amal Khalifa, Echocardiography unit, El Manial university Hospital, Cairo University, for her valuable help, and for all members of cardiothoracic anesthesia unit for their support.

My greatest respect and thanks to all my family members for their support, love and encouragement.

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RV:	Right ventricle
LV:	Left ventricle
HPV:	Hypoxic pulmonary vasoconstriction
PH:	Pulmonary hypertension
SPAP:	Systolic pulmonary artery pressure
MPAP:	Mean pulmonary artery pressure
PVR:	Pulmonary vascular resistance
IOE:	Intraoperative echocardiography
TEE:	Transesophageal echocardiography
EE:	Epicardial echocardiography
OLV:	One-lung ventilation
CO:	Cardiac output
SV:	Stroke volume
RVESA:	Right ventricular end-systolic area
RVEDA:	Right ventricular end-diastolic area
RVFAC:	Right ventricular fractional area of change
PA_{VTI}:	Pulmonary artery velocity time integral
PaO_2:	Arterial oxygen tension
PvO_2:	Venous oxygen tension
TR:	Tricuspid regurge
CWD:	Continuous wave Doppler
PWD:	Pulsed wave Doppler
CFD:	Color flow Doppler
2D:	Two dimensional

$ETCO_2$:	End-tidal carbon dioxide
COPD:	Chronic obstructive pulmonary disease
V_{TTF}:	Velocity of trans-tricuspid flow
PA_{CSA}:	Pulmonary artery cross-sectional area
CPB:	Cardiopulmonary bypass
SD:	Standard deviation

Functional Anatomy

The right ventricle:

"Two souls with but a single thought, two hearts that beat as one"[(1)], 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"[(2)].

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 [(3)].

In contrast, the right ventricle consists of a flattened tube wrapped around the left ventricle with separate inlet and outlet orifices and a presumed contraction pattern simulating peristalsis. Such an arrangement is suited for pumping blood against low resistance [(4)].

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 [(5)].

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 [(6)].

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 [(5)].

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

The pulmonary artery

Arises from the infundibulum of the right ventricle at the pulmonary orifice. It is 5 cm in length and 3 cm wide in diameter, at about the level of fibrocartliage between fifth and sixth thoracic vertebrae, the pulmonary trunk divides into right and left pulmonary arteries which are of nearly equal size[(5)].

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 [(7)].

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 [(7)].

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[(5)].

The large pulmonary arteries have prominent concentric elastic laminae in their walls, although the walls are strikingly thinner than systemic arteries of similar diameter. Elastic arteries are found down to vessels of 1mm diameter, after which the elastic laminae become limited to internal and external laminae and between these circular muscles is found. These small arteries are referred to as muscular arteries although the amount of muscle is slight compared with analogous systemic vessels, Vessels smaller than 100 um lose their muscular layer and have only a single elastic lamina [(7)].

The Right Ventricle as a Pump

Mechanical demands on the RV under normal conditions are minimal compared to demands on the left ventricle (LV) because the work required to pump blood through the low resistance, low impedance lung circulation is a fraction of that required to perfuse the high pressure systemic circulation. The structure of the RV compared to the left reflects the difference in their physiologic requirements [(8)].

In physiologic conditions, systolic right ventricular functions is somewhat secondary, because simple negative pleural pressure produced by breathing promotes blood flow through the pulmonary circulation and ensure sufficient pulmonary venous return, this is possible because the pulmonary circulation offers very little resistance to flow. Conversely, in pathologic conditions, in which there is some increase in pulmonary vascular resistance, right ventricular systolic function becomes essential to promote pulmonary blood flow[**(8)**].

During systole, the right ventricle ejects a large volume of blood through the pulmonary vascular bed, which is a low resistance circuit, thus the right ventricle doses not have to generate a high intracavitary pressure. When the right ventricle is afterloaded, it must adapt its shape to a configuration grossly mimicking that of the left ventricle [(6)].

The normal right Ventricular contraction is complex, including: 1) Shortening along its long axis by action of the spiral muscles, which moves the tricuspid annulus towards the apex, 2)An inward motion of the

free right ventricular wall toward the interventricular septum, 3)Systolic septal thickening, which further reduces the volume of the cavity, 4)Contraction of the left ventricle which tracts the free right ventricular wall and contributes to right ventricular systole, unlike the left ventricle, right ventricle systolic shortening is limited along its short axis and marked along its long axis[9].

The pattern of right ventricular ejection also differs from that of left ventricle, as right ventricle ejection persists after the main chamber end-systole, probably because infundibular end-systole is delayed [10].

Control of cardiac output

Cardiac output (CO) is the volume of blood pumped to the body tissues per minute. It is equal to the product of heart rate (HR) and stroke volume (SV). Normal values for CO are 5 to 6 L/minute in a 70-kg man, with HR of 60-80 beats/minute and an SV of 60 to 90 mL/ beat. CO varies in proportion to the work demand and oxygen needs of the body[11].

Stroke volume is the difference between end-diastolic volume and end-systolic volume. It is augmented by increased in end-diastolic volume (Starling's law) and in myocardial contractility; it is decreased by increases in afterload, The length-tension relation provides a basis for Starling's law of the heart, which states that the strength of contraction of the intact heart is proportional to the initial length of the cardiac muscle fibers - that is, the end-diastolic volume *(preload)*. In vivo, the cardiac muscle fibers are stretched by venous inflow. Normally, the volume in the ventricle before contraction (the preload) sets the sarcomere to a suboptimal length; the active tension that can be developed from that length is only about 20% of maximum. Increases in end-diastolic volume