A DISSERTATION ON

"MRI PHASE CONTRAST FLOW VELOCITY IMAGING OF MAIN PULMONARY ARTERY"

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BONAFIDE CERTIFICATE

Certified that this dissertation is the bonafide work of **Dr.LATHAP.K** on **"MRI PHASE CONTRAST FLOW VELOCITY IMAGING OF MAIN PULMONARY ARTERY"** during her **M.D.RADIODIAGNOSIS** course from May 2012 to April 2015 at the Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai – 600003.

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DECLARATION

I **Dr.LATHA P.K,** solemnly declare that this dissertation entitled**"MRI PHASE CONTRAST FLOW VELOCITY IMAGING OF MAIN PULMONARY ARTERY"** is a bonafide work done by me at the Barnard Institute of Radiology, Madras Medical College and Rajiv Gandhi Government General Hospital, under the under guidance of **Prof. S.BABU PETER.M.D**., **D.N.B**., and under the supervision of **Prof. K.VANITHA, M.D, D.M.R.D, D.R.M,** Director, Barnard Institute of Radiology. This dissertation is submitted to The Tamil Nadu Dr. M.G.R Medical University, towards partial fulfillment of requirement for the award of M.D. Degree Radiodiagnosis.

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Dr.LATHA .P.K

TITLE: MRI PHASE CONTRAST FLOW VELOCITY IMAGING OF MAIN PULMONARY ARTERY

OBJECTIVE :

To assess the pulmonary arterial flow parameters using phase contrast magnetic resonance imaging. To correlate the size of the main pulmonary artery as measured using spiral computerised tomography with the flow parameters as measured using phase contrast MRI and also with ECHO parameters.

MATERIALS & METHODS:

Studies were performed with a 1.5-T clinical magnet Magnetom symphony; Siemens Medical Solutions. Prospective study , study size - 50 subjects. After informed written consent, all subjects who had pulmonary artery size more than 2.9cm underwent phase contrast flow velocity imaging which includes in plane and through plane imaging. Mean VENC value for main pulmonary artery is kept between 75-100 cm/sec. Patients ECHO findings reviewed. Normal subjects with pulmonary artery size less 2.9cm also included in the study.

RESULTS:

37 subjects who had pulmonary artery (PA) size more than 2.9cm had pulmonary artery average velocity less than 11.7cm/sec and minimum

pulmonary artery area more than 10.5 $cm^{2,}$ measured using phase contrast flow velocity imaging.

Degree of correlation (86.5%) is higher between pulmonary artery(PA) size & Pulmonary artery average velocity and also between PA minimum area & PA average velocity (89.5%).

CONCLUSION:

The pulmonary artery size criteria for pulmonary hypertension is well correlated with PA average velocity and PA average area .

MRI Phase contrast imaging is a novel non invasive imaging modality that can be used in the diagnosis of Pulmonary arterial hypertension by means of measuring flow parameters.

Key words: Pulmonary arterial hypertension, MRI,CT,ECHO,pulmonary artery average velocity,pulmonary artery size.

INTRODUCTION

Pulmonary hypertension is a disease which is characterized by increased pulmonary arterial pressure and pathologic changes in precapillary pulmonary arteries. It is a progressive disease. Pulmonary arterial hypertension is defined as an elevation in mean pulmonary arterial pressure above 30 mmHg during exercise and 25 mmHg at rest. Arterial pressure may be considered as a function of blood flow and vascular resistance. Vascular resistance depends upon the cross-sectional area of the vascular bed. The pulmonary vessels are more compliant than their systemic counterparts owing largely to their thin walls and also their larger diameter. Furthermore, the pulmonary bed can also respond to increasing flow by opening up additional vascular channels.

Numerous classification schemes have been developed to categorize the causes of pulmonary hypertension. One method has been to examine the disease from a physiologic perspective, using the relations among pressure, pulmonary vascular resistance, and pulmonary flow. In this type of classification, diseases that cause increased resistance, increased flow, or increased pulmonary vascular pressure are grouped separately. The World Health Organization has classified pulmonary hypertension into pulmonary arterial hypertension, pulmonary venous

hypertension, pulmonary hypertension secondary to hypoxemia/ respiratory disease, pulmonary hypertension secondary to thromboembolic disease, and pulmonary hypertension secondary to processes affecting the pulmonary vasculature directly. Other investigators have classified pulmonary arterial hypertension into pre-and postcapillary etiologies.“Presenting symptoms included dyspnea (60%), fatigue (19%), and syncope (or near syncope) (13%)” (**Stuart rich et al[1])**

Generally it is grouped in to five categories

1. Pulmonary arterial hypertension (PAH)
2. Pulmonary hypertension due to left heart disease
3. Pulmonary hypertension due to lung diseases
4. Pulmonary hypertension due thrombus/embolism
5. Pulmonary hypertension with miscellaneous cause

Echocardiography is the intial mode of investigation of choice. PH due to left heart disease (group 2) is effectively investigated with echocardiography and accounts for the majority of pulmonary hypertensive patients. If a non-cardiac cause is suspected, a series of

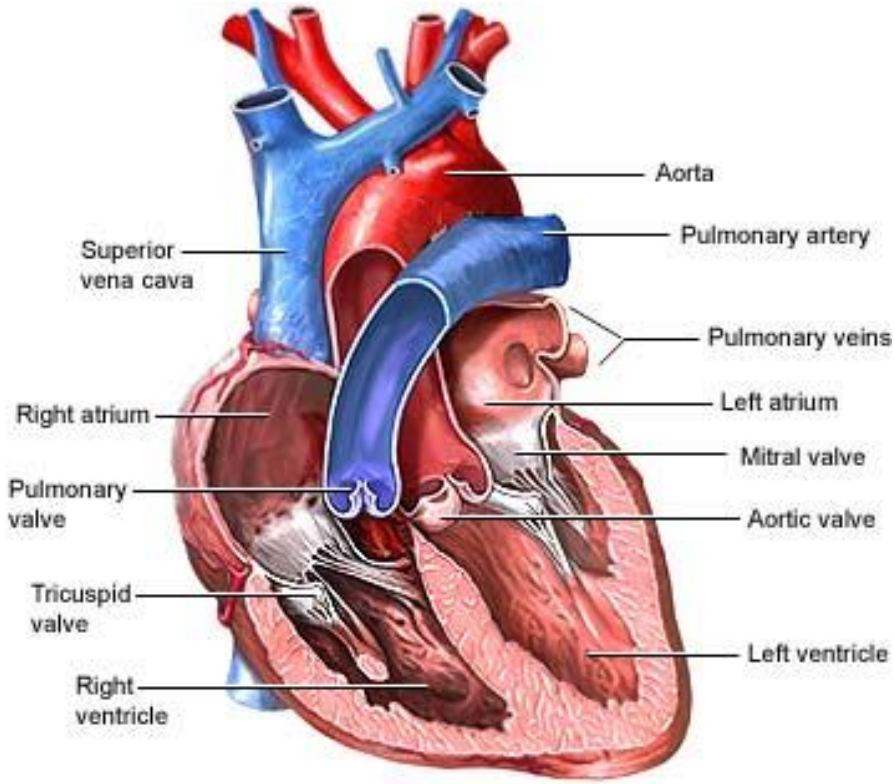
systemic investigations is performed including pulmonary function tests, sleep studies, ventilation perfusion scanning and serological markers.

Computerised tomography (CT) diagnoses pulmonary hypertension depending on the size criteria of the main pulmonary artery. Size of main pulmonary artery > 29mm is sensitive , size of main pulmonary artery > 35mm is specific. CT also useful in detecting lung parenchymal due to pulmonary hypertension and primary lung disease if any that would have caused pulmonary hypertension.

Magnetic Resonance Imaging (MRI) also plays an important role in the diagnosing and monitoring the patients with pulmonary hypertension. Phase contrast MRI is a novel non invasive modality of choice which measures the number of flow parameters like peak and average velocity , average and minimum area of the main pulmonary artery which helps in the diagnosis of pulmonary hypertension.

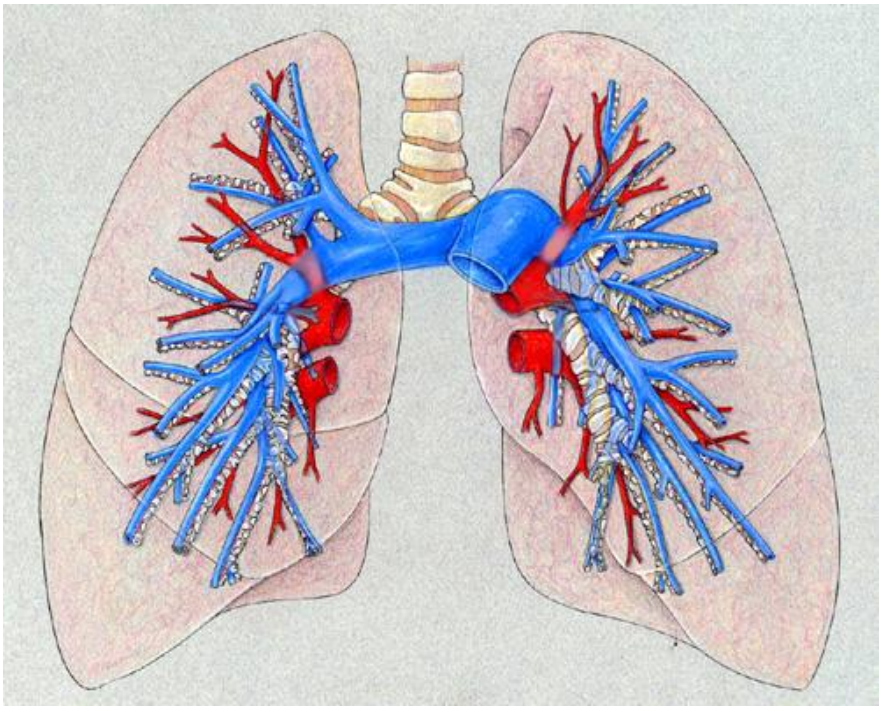
REVIEW OF LITERATURE

ANATOMY AND PHYSIOLOGY OF THE PULMONARY CIRCULATION



The main pulmonary artery origins from the right ventricle at the level of the pulmonary valve and runs approximately 5 cm before bifurcating into the left and right branches.Pulmonary artery carries deoxygenated blood from the right ventricle to the lungs for oxygenation. The right pulmonary artery passes anterior to the right main bronchus and divides into the truncus anterior, which extends into the right upper lobe of the lung, and the interlobar branch, which divides into segmental

arteries extending into the right middle and right lower lobes. The left pulmonary artery is shorter vessel and passes cephalad to the left main bronchus; it continues as an interlobar artery and extends into the left upper and lower lobes of the lung through segmental branches.



Pulmonary circulation : Blue –Pulmonary artery ; Red –Bronchial artery

The pulmonary circulation consists of two parallel networks: the pulmonary arterial circulation and the bronchial arterial circulation. Pulmonary arteries course along the lobar, segmental, and subsegmental airways to the level of the terminal bronchioles. Small pulmonary arteries from the subsegmental level to the terminal bronchioles possess a thick muscular media, and range from 50 to 1000 μm in size. These small

pulmonary arteries progressively lose much of their muscle within the arteriolar media as well as their external elastic membrane.

By the level of the respiratory bronchioles and alveolar ducts they are termed pulmonary arterioles, and range in size from 10 to 150 μm. These vessels ramify further within the alveolar walls to form a rich capillary network. Capillary blood collects in venules, which coalesce progressively to form veins, which course within the interlobular septa, eventually to empty into the left atrium.

Bronchial arteries, averaging two per lung, course within the pulmonary hila along the mainstem bronchi to the level of the terminal bronchiole, and form a plexus that extends from the adventitia through to the submucosa of the associated airway. Bronchial arteries freely form anastomoses with pulmonary arteries, primarily at the capillary and postcapillary levels.

Unlike the tracheobronchial system, in which the major component to air-flow resistance is located within the large airways, the major site of resistance to pulmonary arterial blood flow is located at the small muscular pulmonary arterial and arteriolar level.

Caliber changes in the vessels at this level regulate pulmonary arterial pressure and are critical for optimizing ventilation and perfusion matching.

The pulmonary circulation is a low pressure system—the mean arterial pressure is approximately one sixth that of the systemic circulation. This low pressure is maintained at a relatively consistent level even with large increases in pulmonary blood flow such as may occur with exercise. This is possible because when the body is at rest, numerous pulmonary capillaries normally are not perfused; these capillaries are "recruited" when increased pulmonary blood flow must be accommodated.

Normal pressure values in pulmonary circulation

Central venous pressure		3–8 mmHg
Right ventricular pressure	systolic	15–30 mmHg
	diastolic	3–8 mmHg
Pulmonary artery pressure	systolic	15–30 mmHg
	diastolic	4–12 mmHg
Pulmonary vein/ Pulmonary capillary wedge pressure		2–15 mmHg
Left ventricular pressure	systolic	100–140 mmHg
	diastolic	3-12 mmHg

PATHOGENESIS

Pulmonary hypertension is defined as a pulmonary systolic arterial pressure equal to or exceeding 25 mmHg at rest or 30 mmHg with exercise, or a mean pulmonary arterial pressure equal to or exceeding 18 mmHg (National Institutes of Health (NIH) registry of patients with primary pulmonary hypertension) (**Yoshiharu Ohno et al**[2]). Pulmonary venous hypertension is present when pulmonary venous pressure, usually approximated by measurement of the pulmonary capillary wedge pressure, is equal to or exceeds 18 mmHg.

Table 1 Hemodynamic definitions of PH

Definition	Characteristics	Clinical group(s)*
PH	MPAP ≥ 25 mm Hg	All
Precapillary PH	MPAP ≥25 mm Hg PCWP ≤15 mm Hg CO normal or reduced†	1. PAH 3. PH due to lung disease 4. Chronic thromboembolic PH 5. PH with unclear and/or multifactorial mechanisms
Postcapillary PH	MPAP ≥ 25 mm Hg PCWP > 15 mm Hg CO normal or reduced†	2. PH due to left-heart disease
Passive	TPG ≤ 12 mm Hg	
Reactive (out of proportion)	TPG > 12 mm Hg	

TPG, Transpulmonary pressure gradient (MPAP _ mean PCWP).

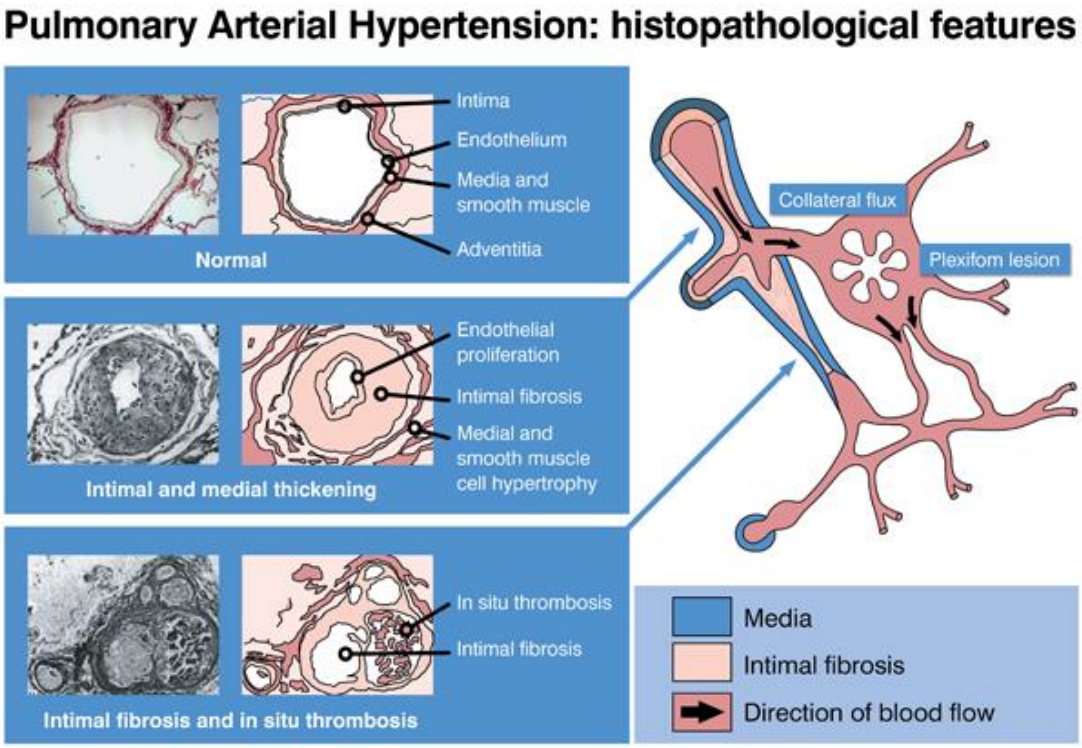
Values are measured at rest (**Galie et al**[3])

Several mechanisms may produce a decrease in the total number of small pulmonary arteries, thereby increasing pulmonary vascular resistance and producing elevated pulmonary arterial pressure. These mechanisms include intraluminal arterial occlusion, muscular contraction of small pulmonary arteries, vascular remodeling with wall thickening, or conditions that produce pulmonary venous hypertension.

Several of these mechanisms may be operative simultaneously in a patient with pulmonary hypertension.

The pulmonary vascular endothelium responds to changes in oxygen tension, transmural pressure, and pulmonary blood flow, and participates actively in the regulation of pulmonary arterial pressure through the elaboration of various vasoactive substances, such as prostacyclin, nitrous oxide, and endothelin. The agents have a direct effect on pulmonary vascular smooth muscle tone (promoting relaxation and vasodilation), and also may directly affect platelet function.

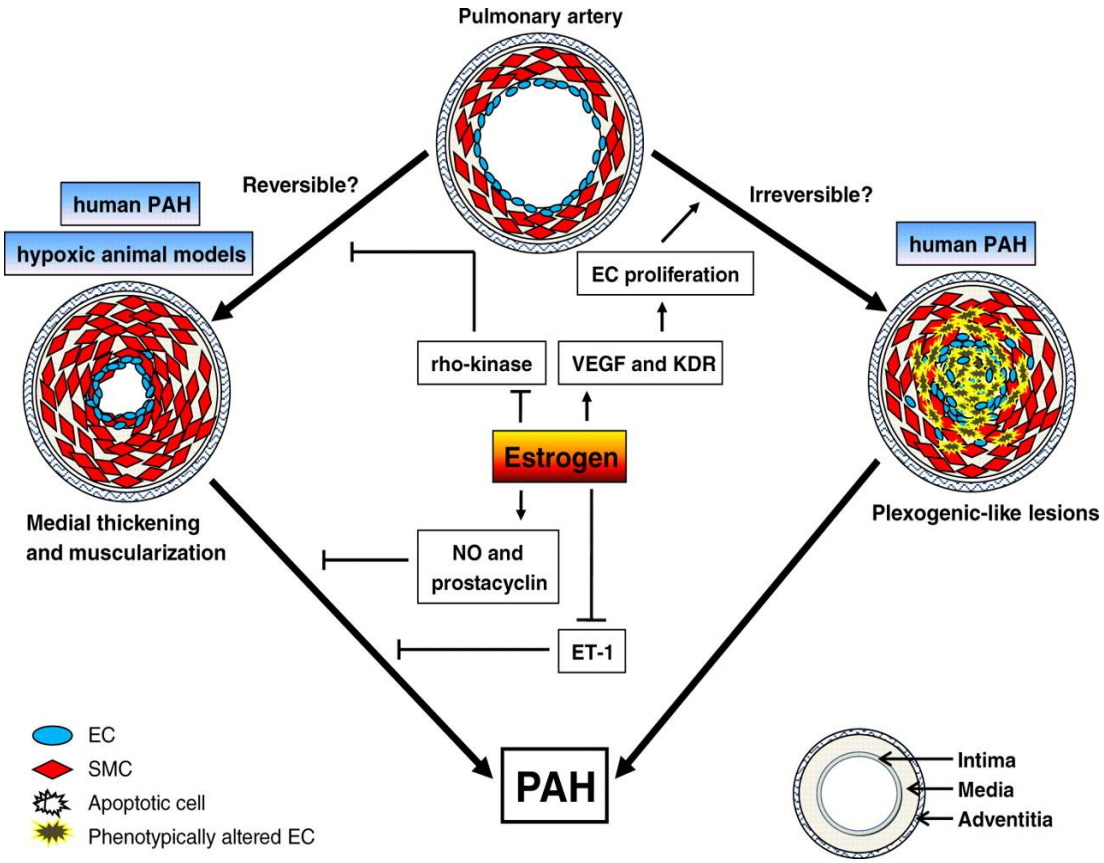
Abnormalities in endothelial cell function or injuries to these cells may be the fundamental derangement that ultimately produces the structural vascular changes observed in patients with pulmonary hypertension.



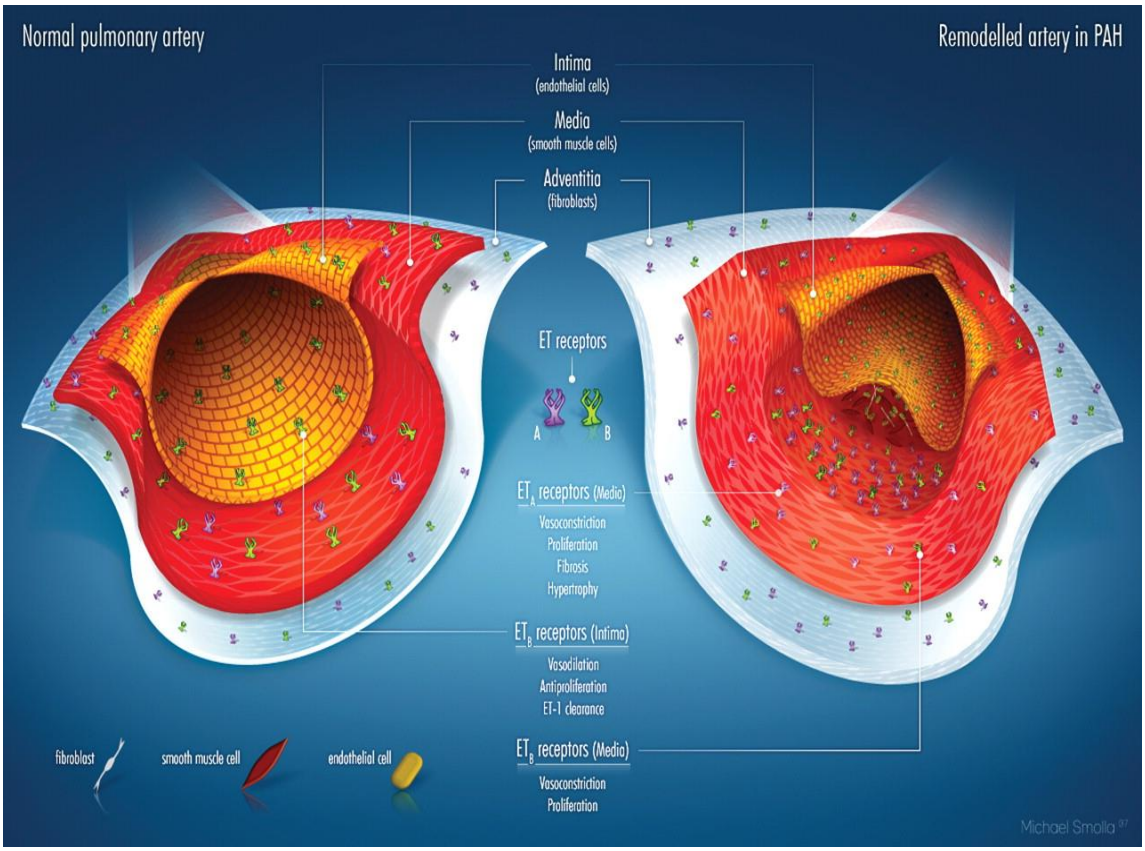
Various histopathological abnormalities may be observed in patients with pulmonary hypertension, varying somewhat depending on the cause of hypertension. In general, regardless of the specific cause of the pulmonary hypertension, the pulmonary arteries become dilated, occasionally to the point of being considered aneurysmal. Pulmonary arterial atherosclerosis, although occasionally present to a mild degree in the larger pulmonary arteries of normal adults, often is extensive in patients with pulmonary hypertension and commonly involves small arteries.

Pulmonary hypertension-related pulmonary arterial atherosclerosis pathologically appears similar to atherosclerosis in systemic arteries,

although complicating features, such as necrosis, ulceration, and calcification, are relatively uncommon. Thickening of the muscular media of small pulmonary arteries is a common feature in many causes of pulmonary hypertension, and usually results from a combination of muscular hyperplasia and hypertrophy. Often, extension of muscular tissue into arterioles that normally contain no muscle, or "arterialization," may be observed in patients with pulmonary arterial hypertension.



The term pulmonary plexogenic arteriopathy refers to a constellation of histopathological vascular changes that often is encountered in patients with primary pulmonary hypertension, but it also may be seen in patients with pulmonary hypertension of other etiologies, such as hepatic disease, connective tissue disorders, congenital cardiovascular disease, and some medication prescribed for weight loss.



PULMONARY PLEXOGENICARTERIOPATHY

Histopathological features present in pulmonary plexogenic arteriopathy include a combination of fibrinoid necrosis, dilation lesions, plexiform lesions, intimal fibrosis, and vasculitis. Plexiform lesions affect small muscular arteries ranging in size from 100 to 200µm, usually near vascular branch points, and consist of a focally dilated muscular vessel with a disrupted internal elastic membrane that contains very narrow vascular channels interspersed with fibroblasts and connective tissue. Plexiform lesions are characteristic of prolonged severe pulmonary hypertension.

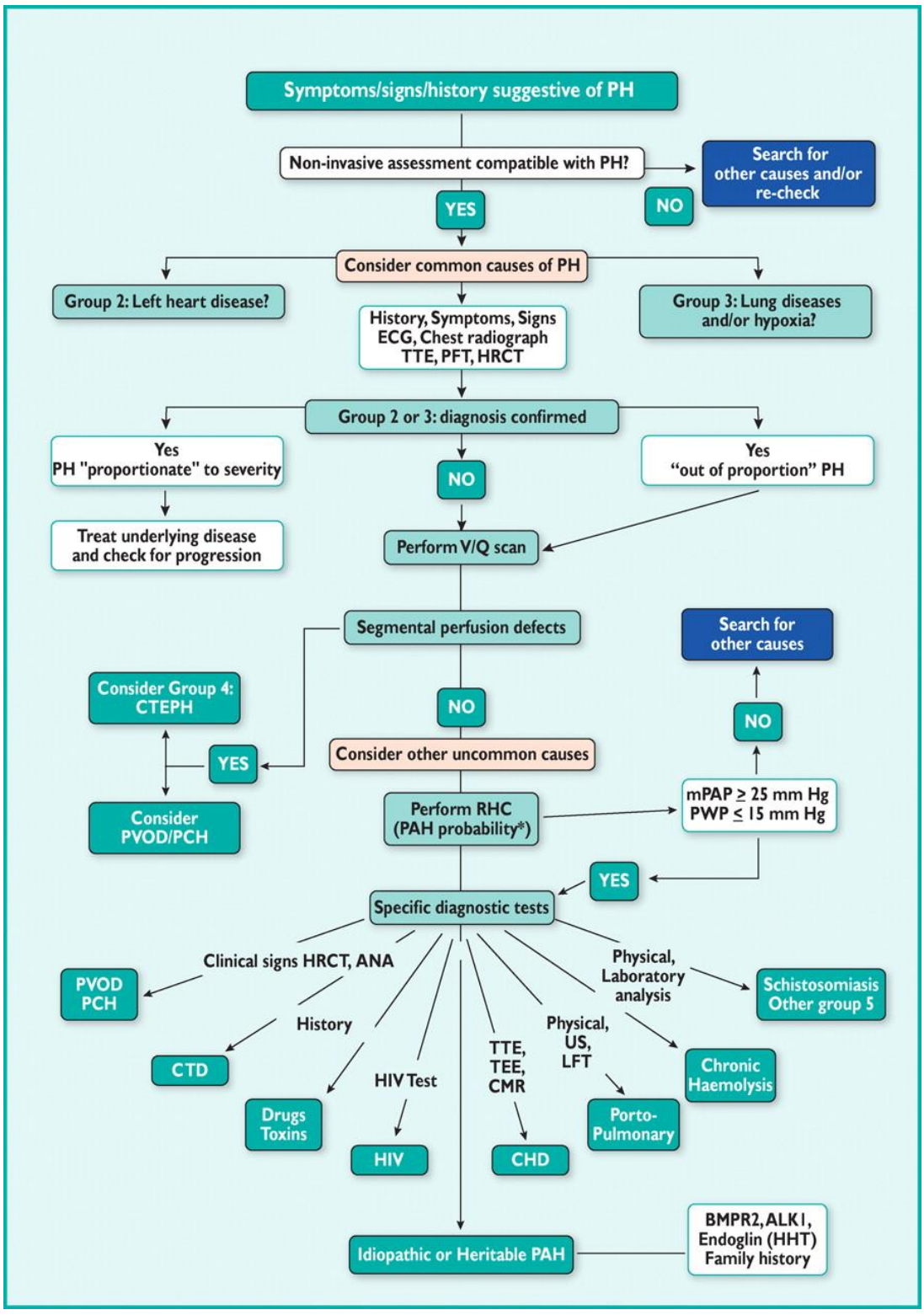
GRADING OF PULMONARY HYPERTENSION

	Grading of pulmonary arterial hypertension*		
	Systolic	Diastolic	Mean
Grade 1 (Mild)	30-50	20-25	>30
Grade 2 (Moderate)	50-70	26-35	>40
Grade 3 (severe)	70-110	36-45	>50
Grade 4 (Systemic or supra systemic)	>110	46-55	>60

*Data from 100 patients of PAH and rheumatic heart disease. Quintile 1 & 2 (Grade 1) qunitile 3 & 4 (Grade 2) quintile 5 (Grade 3) top 3%(Grade 4)

APPROACH TO PULMONARY HYPERTENSION

(Nazzareno Galie et al[4])



ROLE OF ECHOCARDIOGRAPHY

Echocardiography is a vital screening test in symptomatic patients at risk for Pulmonary Hypertension. It measures the pressure in the pulmonary vessels and also used to exclude cardiac cause for pulmonary hypertension. As an investigation modality, it has the advantage of being widely available, non invasive technique and cost effective.

Echo parameters include **qualitative assessment of right ventricle,** which is assessed by parasternal long-axis, short-axis and the apical four-chamber views. Dilated right atrium , right ventricle frequently seen. D- shaped interventricular septum is pathognomic. Right ventricular hypertrophy is seen but dilatation is more common. Right ventricular hypertrophy is defined by a wall thickness of more than 0.7cm on the apical four-chamber view. Hypertrophy of moderator band will also be seen. Dilatation of tricuspid annulus and dilated main pulmonary artery is noted classically.

Regional wall motion abnormalities, contractility assessment of right ventricle can be done in patients with pulmonary hypertension. **(Luke S. Howard et al[5])**

TRPG , tricuspid regurgitant pressure gradient measurement plays important role in the diagnosis and grading the degree of pulmonary hypertension. And depending on the values it is divided in to mild , moderate and severe. Value <25mmHg is normal, > 25mmHg is suspicious for pulmonary hypertension, 30-40 mild , 40-60 moderate, >60 severe.

Pulmonary valve motion abnormalities can be assessed in the echocardiography. In M mode echocardiography following features are seen in a case of pulmonary hypertension presence or absence and the amplitude of the "a" wave, magnitude of the e-f slope, fluttering of the posterior pulmonic leaflet presence of mid-systolic closure or notching,

"TAPSE, Tricuspid annular plane systolic excursion is the reflection of the movement of base to apex shortening of the RV in systole and can be derived from the four-chamber view .When measuring TAPSE, it is important to ensure that the entire RV is included in the view, in particular that there is no dropout in the endocardial outline along the IVS and RV free wall. Maximal TAPSE is defined by the total excursion of the tricuspid annulus from its highest position after atrial ascent to the peak descent during ventricular systole **(Kaul S et al[6])** ."

"A TAPSE of <15 mm is associated with a significantly higher risk of mortality compared with a TAPSE of > 15 mm **(Ghio S et al[7])** and TAPSE is recommended by treatment guidelines as a prognostic indicator in PAH for the assessment of disease severity and response to therapy." And Value of > 20mm is normal.

Many other parameters like right atrial volume index, inferior vena cava diameter etc ., can be measured.

And also to look for secondary cause of pulmonary hypertension like left ventricular dysfunction, left heart valve disease, congenital heart disease echocardiography plays important roll.

Transoesophageal echocardiography can be done.

ROLE OF RADIOGRAPH AND CT

The pulmonary arterial hypertension is characterised by dilation of the pulmonary arteries with abrupt tapering of the peripheral pulmonary vessels. This is the universal features in all etiology of the pulmonary hypertension.

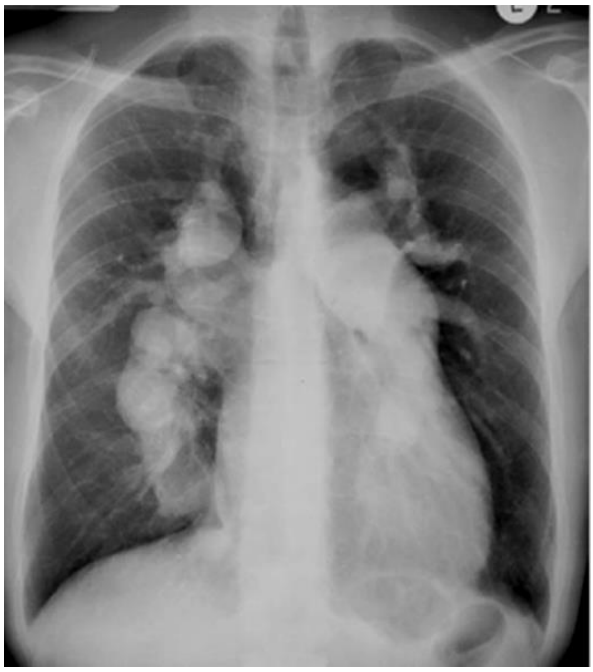
Chest radiography shows enlargement of the main pulmonary artery segment and dilation of the right and left interlobar pulmonary arteries. Diameter of interlobar pulmonary artery at the level bronchus intermedius, exceeds 15 mm in women and 16 mm in men is characteristic for pulmonary hypertension . The left pulmonary artery is best measured from the orifice of the left upper lobe bronchus to the posterior aspect of the vessel if it exceeds 18 mm, pulmonary hypertension probably is present.

The upper limit for a normal main pulmonary artery on axial CT or MR images is 29 mm. When the main pulmonary artery segment exceeds 29mm, pulmonary hypertension is usually suspected. However , pulmonary hypertension may be present in patients with a normal sized main pulmonary arterial segment. If the main pulmonary artery is visibly larger than the aorta, elevated pulmonary pressures usually are present.

Radiographically, right atrial and ventricular enlargement, enlargement of the main pulmonary arteries and tapering of peripheral arterial branches termed ‘peripheral pruning’—are seen. In chronic cases the main pulmonary arteries may develop calcification due to atheroma. Central arterial enlargement may mimic enlarged hilar lymph nodes. It is

differentiated by the feature that lymphadenopathy characteristically has a lobulated border whereas arterial enlargement has a smoother outline.

A recognized method of assessing pulmonary arterial size is by measuring the size of the right descending pulmonary artery. Enlargement may be diagnosed if the transverse diameter of the artery at its midpoint is greater than 17 mm. Overall, although relatively specific, the sensitivity of chest radiography for the diagnosis of PAH is low



Chest radiograph demonstrates gross dilatation of the main, left and right pulmonary arteries in a patient with Eisenmenger atrial septal defect.

When pulmonary hypertension is prolonged and severe, calcification of the pulmonary arteries, usually affecting the main, right, or left pulmonary arteries, and, less commonly, the lobar pulmonary arteries, may be present. This finding usually, but not invariably, is associated with irreversible vascular disease.

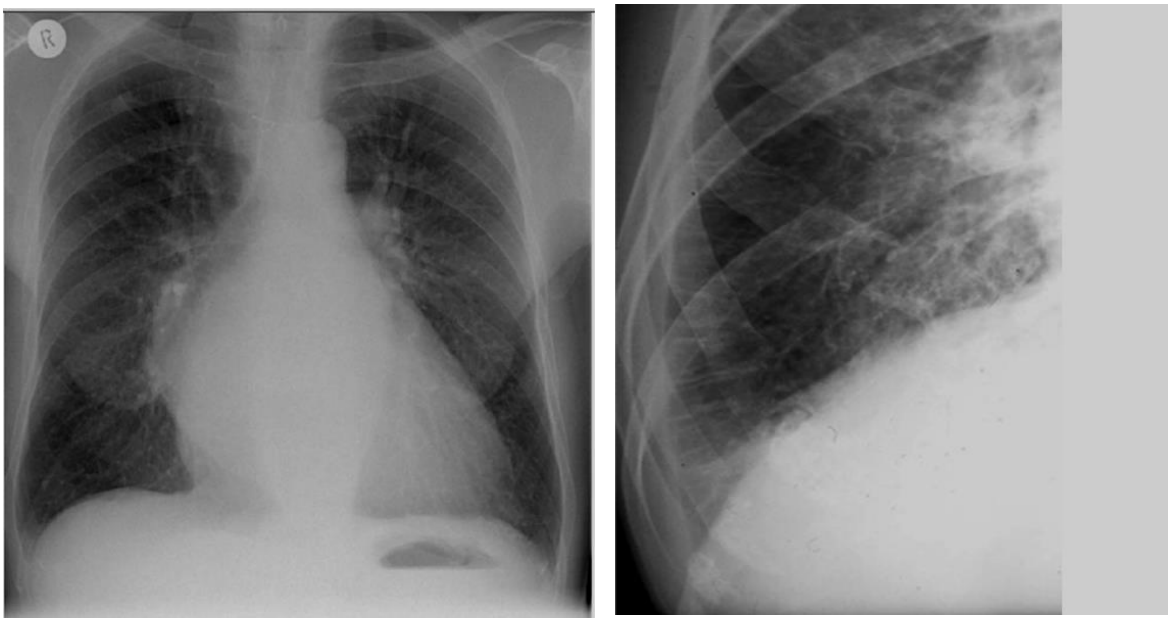
Although chest radiography and CT scanning often may suggest the presence of pulmonary hypertension, echocardiography is the examination most commonly used for noninvasive assessment of possible pulmonary hypertension. Echocardiography, using continuous wave or pulsed Doppler, provides noninvasive measurement of pulmonary arterial pressures and also allows detailed morphologic evaluation of the right ventricle.

As pulmonary venous pressure rises, the upper lobe veins distend. They initially reach the size of, and eventually become larger than, the lower lobe vessels (thus reversing the normal 'gravity-dependent' pattern). This is described as 'upper lobe venous diversion' and is often the first recognized radiological sign of pulmonary venous hypertension . If the pulmonary venous pressure continues to rise and exceeds the plasma oncotic pressure, fluid will begin to accumulate in the lung interstitium. This is known as interstitial pulmonary oedema. Radiologically this is associated with the appearance of interstitial (Kerley B) lines. These lines were first described in 1933 **(kerley .p[8])** and represent thickening of interlobular septa within the lung. They were originally classified into three groups:

1. Kerley A lines are 4 cm in length seen in the upper and mid portions of the lung. These deep septal line radiate from the hilum to the central part of the lungs but do not reach the periphery / pleura. These lines suggest more acute or severe degree of edema.

2. Kerley B lines are short measuring 1 cm or less interlobular septal lines, seen in the periphery of lower zones, parallel to each other and at 90 degree to the pleural surface.

3. Kerley C lines are now seen to overlap Kerley B lines. It is no longer used now (**Heitzman ER**[9])



A-Upper lobe venous congestion B-Thickened interlobular septae; kerley b

Certain patterns of opacification may suggest particular diagnoses. The often cited ‘perihilar bat's wing’ pattern of airspace consolidation is seen most commonly in left ventricular and renal failure, whereas alveolar oedema localized to the right upper zone is suggestive of severe mitral regurgitation. The latter is thought to be a result of predominant regurgitant blood flow in the right upper lobe pulmonary vein, from the superiorly and posteriorly positioned mitral valve.

Computed tomography (CT) findings in pulmonary oedema include thickening of septal and bronchovascular structures. In addition, perihilar ground-glass opacity may be found in cases of mild parenchymal oedema. Alveolar oedema may initially be recognized as peribronchovascular airspace nodules progressing to dense airspace consolidation.

In chronic pulmonary venous hypertension signs of pulmonary arterial hypertension may also develop. In addition, a fine nodular pattern may appear throughout both lungs. These nodules represent haemosiderin deposition. This pattern was previously most commonly seen in patients with long-standing severe mitral stenosis. In very severe chronic PVH pulmonary ossicles (up to 1 cm in size) may develop

Chest radiography in patients with Primary Pulmonary Hypertension shows enlargement of the main, right, and left pulmonary arteries, often with enlargement of the right ventricle and right atrium. High-resolution CT (HRCT) may show that the peripheral pulmonary arteries are substantially larger than usual.

Attempts have been made to correlate the stage of radiographic pulmonary venous hypertension with pulmonary venous pressure (as measured by the pulmonary capillary wedge pressure [PCWP]) (**10,11,12**). When the PCWP is normal (8–12 mmHg) the chest radiograph is not expected to demonstrate any specific abnormality related to pulmonary venous pressure. Mild PCWP elevation (12–18 mmHg) is associated with upper lobe venous distension. Further PCWP increase (19–25 mmHg) leads to interstitial oedema (peribronchial cuffing, Kerley lines). Above this value (25 mmHg) airspace opacities are seen. Although not accurate, these correlates serve to give an approximation of intravascular pressure.

Although most cases of PVH are associated with valvular and/or myocardial dysfunction leading to cardiomegaly, this is not always the case. An important example of this is in the early post-myocardial infarction phase. Here, up to 50% of patients have been shown to exhibit

radiographic signs of PVH in the first 24–48h post infarction (**Higgins CB et al**[13,14]). This is due to an acute decrease in myocardial compliance which essentially resolves in the first week post infarction. The other situation where signs of pulmonary oedema may be seen with a normal heart size is non cardiogenic pulmonary oedema.

High-resolution CT may demonstrate inhomogeneous lung opacity, representing the presence of differential pulmonary parenchymal perfusion. The regions of decreased pulmonary parenchymal attenuation represent areas of mosaic perfusion, and the vessels in these regions of lung often are visibly smaller than their counterparts in regions of increased lung attenuation. Although airway diseases may result in a similar pattern of mosaic perfusion, vascular and airway causes of mosaic perfusion may be distinguished using postexpiratory imaging. When due to airway diseases, differences in lung attenuation become accentuated with post expiratory imaging, whereas a proportional increase in attenuation in areas of both increased and decreased attenuation is expected for patients with pulmonary vascular disease.

"A prospective study **(Alhamad EH et al [15])** in which the subjects were 134 patients who underwent rightheartcatheterization and chest CT within 72hours of each other showed that CT-derivedmeasurement of the

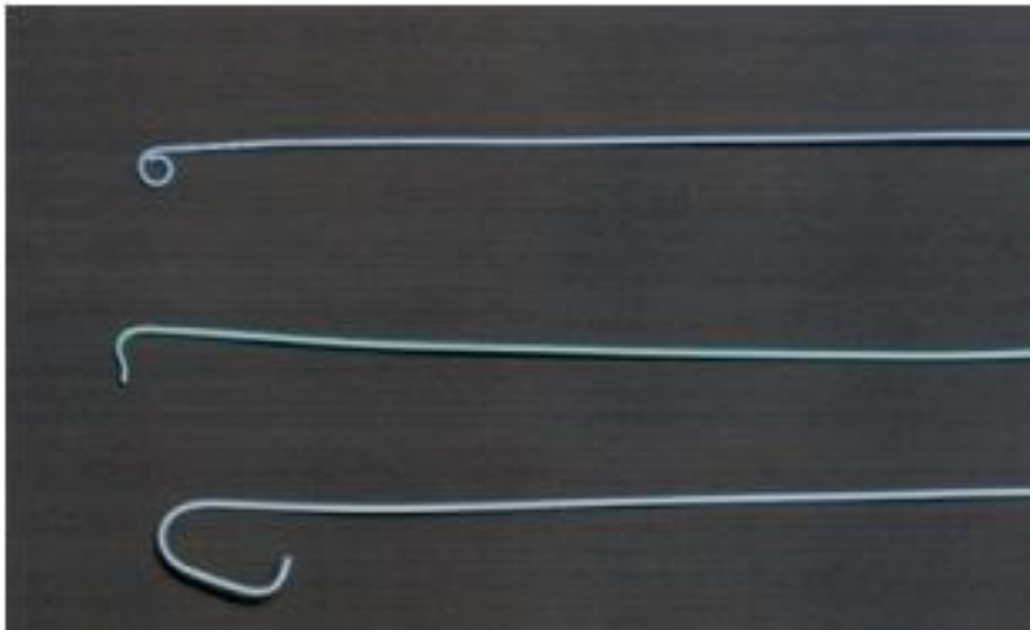
MPA diameter has strongercorrelation with the presence of pulmonaryhypertension in patients without ILD (MPAdiameter > 31.6 mm had a positive predictivevalue of 90.0% and a negative predictive valueof 58.3%) than in patients with ILD (MPA diameter> 25 mm had a positive predictive valueof 46.3% and a negative predictive value of83.8%). In both groups, however, the MPA diameterwas significantly greater in patients withpulmonary hypertension than in those without"

A number of findings on CT have been shown to be useful in evaluating possible PAH. A ratio of pulmonary artery diameter to aortic diameter of >1 has been shown to correlate with elevated mean PAP, although a ratio of <1 does not exclude PAH. In a study by **Kuriyama et al**[15], a **main pulmonary artery diameter of more 29 mm was shown to have a sensitivity of 69% and specificity of 100% for predicting PAH**. Main pulmonary artery diameter has been shown to be useful for detecting PAH in patients with advanced lung disease, with a sensitivity of 87% and specificity of 89%, with the additional finding that a segmental artery-to-bronchus ratio >1:1 increases specificity

RIGHT HEART CATHETERIZATION

It is generally known as pulmonary artery catheterisation or Swan-Ganz catheterisation. It is the gold standard modality in diagnosing and evaluating pulmonary hypertension. Echocardiography is very useful screening tool for the presence of pulmonary hypertension, but it only gives an measure of right ventricular systolic pressure.

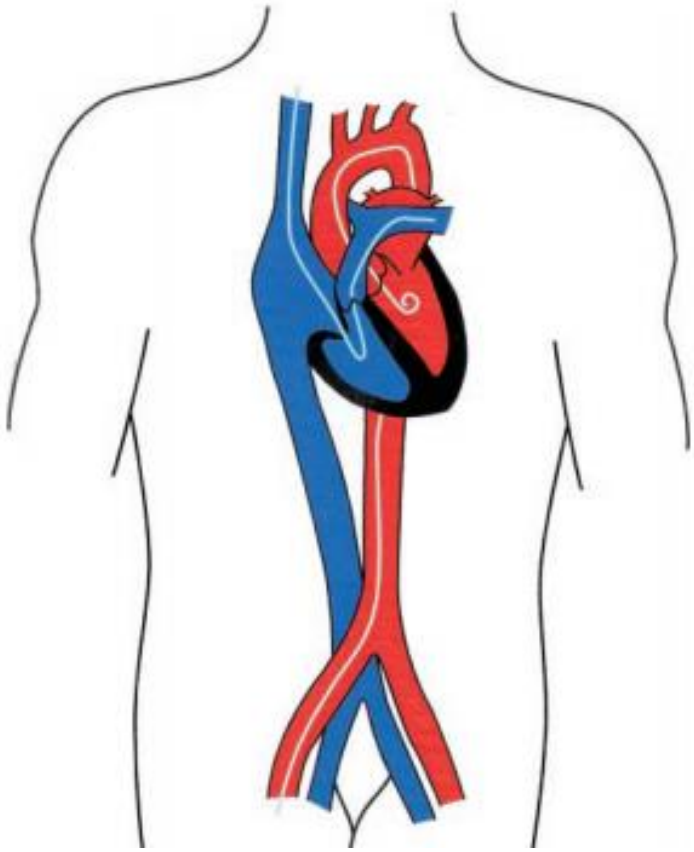
CATHETER



Catheter

- The catheter is usually 120 cm length and has multiple lumens .
- A small inflatable balloon is located at the tip of the catheter. It is inflated so that it can “float” in the direction of blood flow .
- Thermistor (temperature indicator) is present at the tip of the catheter; it detects the changes in blood temperature when performing thermodilution cardiac output measurement

CHOICE OF VENOUS ACCESS SITES



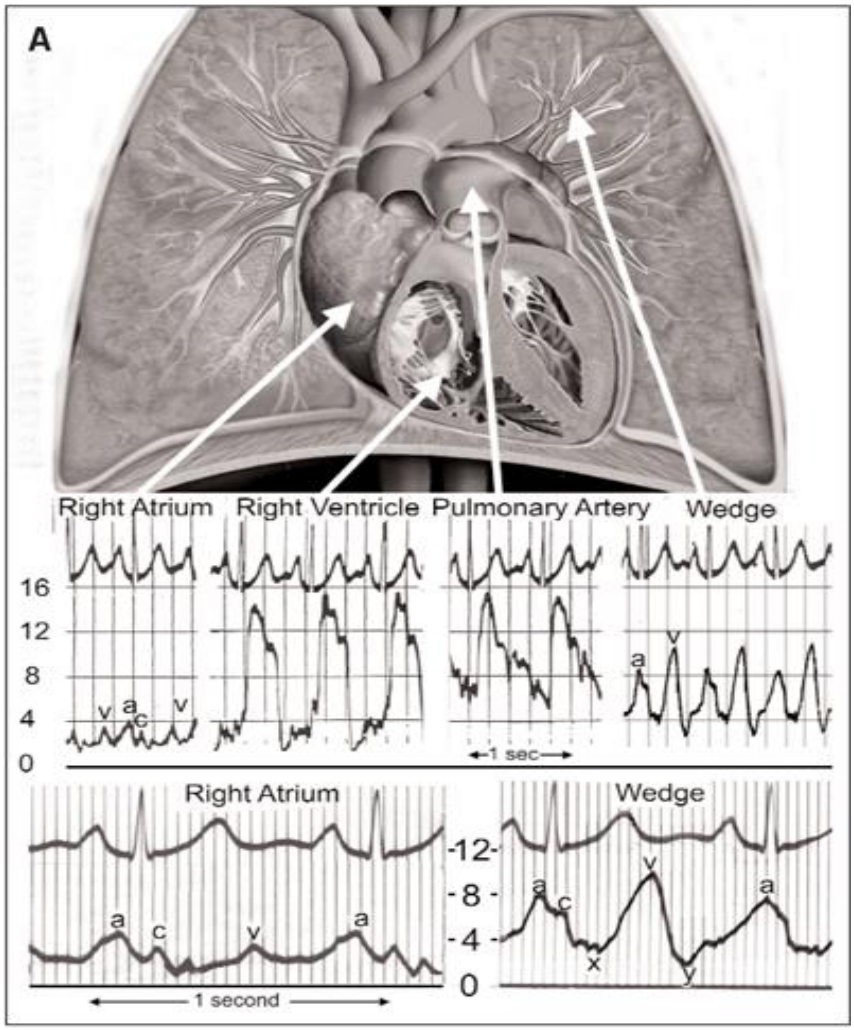
- Right internal jugular vein is used.
- Initially , for a patient's initial catheterization, femoral veins are used for catheterization, as it allowed the greatest flexibility especially for left heart catheterisation.
- In patients for whom it is tough to advance the catheter into the pulmonary artery due to increased pulmonary arterial pressures, the right internal jugular approach may be superior than inferior vena cava approach. This is because this approach allows the catheter to form a natural curve on the floor of the right ventricle it points upward into the main pulmonary artery.

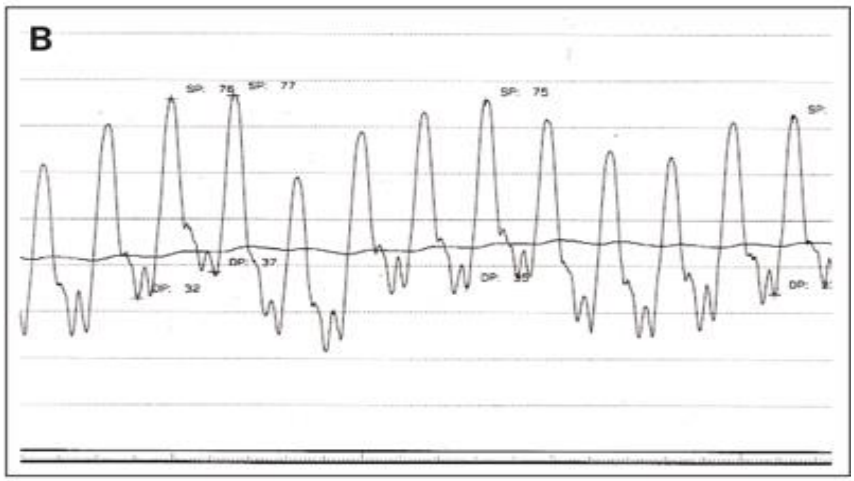
STANDARD RIGHT-HEART CATHETERIZATION MEASUREMENTS

- right atrial pressure (RAP)
- right ventricular pressure (RVP)
- pulmonary arterial pressure (PAP)
- **pulmonary capillary wedge pressure (PCWP)**
- systemic arterial pressure (BP) and heart rate

- cardiac output (CO)
- pulmonary arterial vasoreactivity
- pulmonary arterial (PA) saturation
- superior vena cava (SVC)& IVC saturation
- right atrial (RA)& RV saturation

Normal pressure waveforms in right heart catheterisation





Risks in central venous catheterization :

- It is highly invasive
- As the lung is close to the major veins of the neck. The needle could pierce the lung it can cause pneumothorax and collapse of the lung
- Patient discomfort canresult from placement of the catheter and when it is inserted..
- Any catheter entering the body can be a source of bacteria and infect the patient.
- Bleeding occurs around holes in the veins but it is mild and seals on its own . As the major arteries run alongside the major veins, unknowningly arteries can be punctured . It is usually self limiting but sometimes it can cause hemomediastinum which is a life threatening condition.
- Blood clots can commonly form in catheters inside the veins. Such clots usually do not cause any problems. Sometimes, clots can travel into the lungs causing pulmonary embolism.

- Rarely, air enters the catheter travel through the heart and cause lung injury and cause air embolism.

Some risks are specific to the placement of the catheter through the heart to the pulmonary artery.

- **Rupture of the pulmonary artery** – It is very rare complication. It can cause life-threatening bleeding

- **Heart rhythm abnormalities** - The catheter can accidentally tickle the heart and stimulate, causing arrhythmia.

ROLE OF MRI

MRI is a novel ,non invasive method of imaging heart morphology and its functions. Cardiac MRI helps assessing morphology of the heart like wall movement, valve movement , blood vessels (aorta, pulmonary vein, coronary artery). Function of the heart like blood volume ,flow, cardiac output can be analysed. It measures hemodynamic parameters in pulmonary hypertension patients which helps in diagnosing as well as in monitoring the disease process and its severity.

Cardiac MRI is used to measure the hemodynamic parameters in the pulmonary hypertension patients which helps in the diagnosis as well as post treatment follow up and monitoring of the disease. **(Peacock AJ et al[16])**

MRI is a useful modality for visualising right ventricular anatomy , function which plays important role in the prognosis of patients with pulmonary hypertension.Contrast MRI clearly depicts the myocardial scarring due to infarct, infection and infiltration and infiltrative pathology of myocardium by means of showing delayed enhancement as these pathology can be seen associated with pulmonary hypertension.

Like echocardiography, MRI can also quantify flow velocity with in the vessels by means of phase contrast flow velocity imaging. Advantages of MRI compared with the echocardiography is there is no limitation in choosing the arbitrary plane of imaging, whereas in echocardiography because of inadequate acoustic window we may not be able to choose the arbitary plane of imaging. Thus as we can choose arbitrary plane of imaging in MRI it has a greater accuracy and reproducibility as compared with the echocardiography**(Shah DJ et al[17]).** More over in echocardiography diagnosis of pulmonary hypertension is made with parameter like TRPG (tricuspid regurgitant pressure

gradient) which is the indirect measurement of pulmonary hypertension in echocardiography. Whereas in MRI using phase contrast flow velocity imaging we are able to measure the velocity directly in the pulmonary artery.

MRI is considered as standard modality of choice for assessment of congenital heart disease as it gives very good information about the structure changes , situs , septal defects , atrio ventricular concordance, vessel diameter , wall motion, valvular stenosis / regurgitation**.(Landzberg MJ[18])**

MRI has many advantage over CT and echocardiography. There is no ionising radiation , thus allowing repeated examination whenever necessary without the effect of cumulative radiation exposure. MRI has superior resolution in delineating the soft tissue contrast and has higher spatial resolution than echocardiography. Tailored study of the heart and great vessels using Cardiac MRI and MR angiogram**.(McLure LE et al[19])**

Imaging sequences used for cardiac MR can be broadly divided in to dark blood technique and bright blood technique . Dark blood sequences are basically spin echo sequences that show flowing blood as flow void it includes conventional spin echo, breath hold turbo or fast

spin echo and double inversion recovery fast spin echo. They are static images which show anatomy and myocardial viability. As the signal from moving blood is very low it appears black.

Bright blood sequences are gradient echo sequences that show blood as bright. Gradient echo sequences used for cardiac MRI include turbo FLASH and true FISP. A motion picture loop throughout the various phase of cardiac cycle can also be obtained in gradient sequences. Cine imaging is useful in functional assessment of ventricles. This sequences show signal intensity of moving blood as bright.

Orthogonal planes (axial , sagittal, coronal) used for general chest imaging are not suitable for cardiac imaging because cardiac axes are not parallel to the body axes. For morphology cardiac long axis views are done , function assessment short axis views are done.

ECG gating is essential for motion free images of heart. Images are acquired in the particular phase of the cardiac cycle in every cardiac cycle to avoid images blur and cardiac motion artefacts. The phase of cardiac cycle during which images are acquired is decided by ECG gating. Usually R-wave is used to trigger the acquisition after some time delay such that data is acquired in the diastolic phase. Peripheral pulse can also be used for gating but it is less effective than ECG gating.

Two types of ECG gating**(Joachim Lotz et al[20])**

- ✓ **Prospective gating**
- ✓ **Retrospective gating**

- **Prospective gating techniques** – Before starting data acquisition a signal is required . The time interval between trigger signal is called the arrhythmia rejection window . Example : Breath hold Phase-contrast sequences use this gating.

- **Retrospective gating techniques** –No signal required data acquisition is done throughout the cardiac cycle. Later , this technique retrospectively assign the data from the recorded trigger signals. It can give information of the entire cardiac cycle.

Imaging planes include:

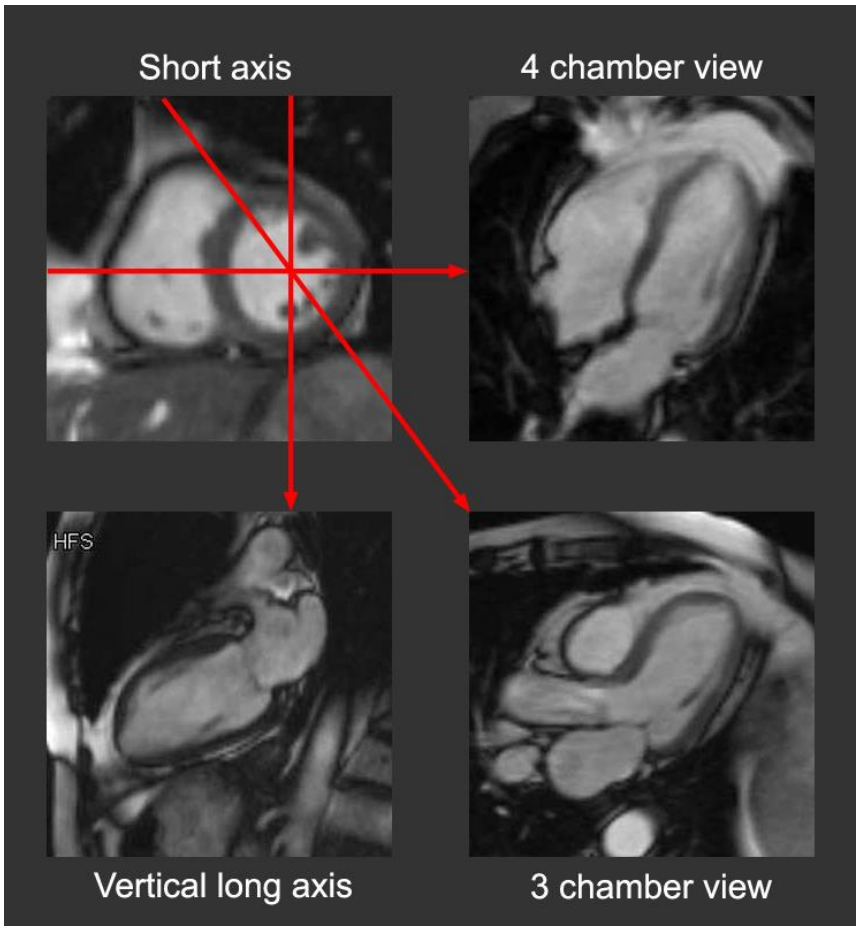
1. Vertical long axis plane (two chamber view)

2. Horizontal long axis view (four chamber view)

3. Short axis plane

4. Five chamber plane

5. Right ventricular outflow tract plane



IMAGING PLANES – CARDIAC MRI

Advantages of MRI (Raymond Benza et al[21])

- ✓ Unparalleled resolution
- ✓ Three-dimensional imaging capacity
- ✓ Noninvasive
- ✓ Ability to depict soft tissues

Disadvantages of MRI

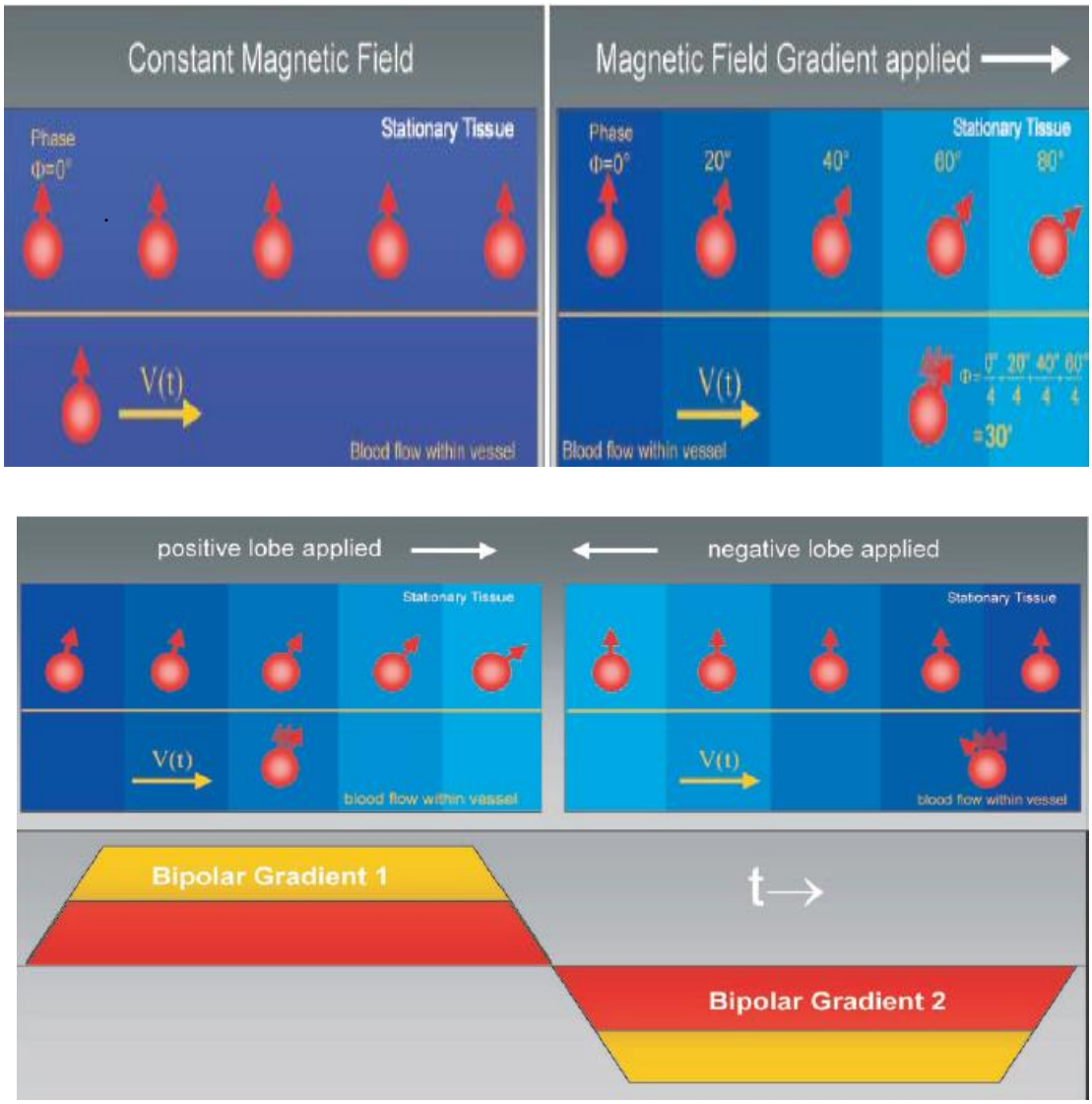
- ✓ Long scan times
- ✓ Artifacts including motion, respiratory, cardiac motion
- ✓ Incompatibility with pacemakers, defibrillators, and aneurysm clips

MR PHASE CONTRAST STUDY

"With the increasing power of MR imaging units and the reduced time needed to complete a cardiac study, more and more indications are evolving for use of phase-contrast flow measurements as an additional source of quantitative functional information in cardiac MR imaging." **(Sakuma H et al[22])**

Phase contrast study includes 'through plane' and 'in plane' imaging. Basic technique behind phase contrast study is contrast between the flowing blood & stationary tissues by manipulating the change in phase of magnetisation, such that phase of magnetisation from stationary spins is zero and phase of magnetisation from spins that are in movement wil be non-zero. Depending on how far the magnetisation ahead from the time it is tipped in to transverse plane until the time it is detected.

"Hunter C. Champion etal[23] conducted studies using the phase-contrast technique and velocityencodedMRI showed the feasibility of estimating right-sidehemodynamics, but the ease of Doppler echocardiographyhas limited the enthusiasm of proceeding with large-scalevalidation studies."



In this technique signal intensity acquired is linearly proportional to the velocity of the spins. Rapidly moving spins will produce higher signal thus spins moving in one direction give bright signal and spins moving in opposite direction give dark signal. By this technique vascular structures can be identified and imaging can be done. Speed and direction of flowing blood is determined qualitatively. Quantitative analysis like velocity of the blood, volume of the flow rate can be measured using

phase difference images. Cross sectional imaging of the vessel allows to measure the area of the vessel.

Volume of flow rate = Area x Average velocity

Velocity measurement in phase contrast study is sensitive to particular range of velocities. So we have to specify particular range of velocity for particular studies. It is assigned by a term called Venc, velocity encoding value. Assigning the Venc value is very critical because if we assign the Venc value too low it will produce aliasing and can be misinterpreted in the image, thus choosing Venc value is very important.

Phase shifts are measured in terms of degree and usually range within -/+ 180 degree.

$$\Delta\phi = \gamma \cdot \Delta m \cdot \nu$$

$\Delta\phi$ - Phase difference

γ - Gyromagnetic ration

Δm– Difference in the first moment of gradient time

Phase difference depends on

- ✓ Gyromagnetic ratio
- ✓ Difference in the first moment of gradient time
- ✓ Velocity

Venc is measured in terms of centimetre/sec. It ranges between highest and lowest detectable velocity for example if Venc = 75 cm/sec describes range of velocity between +/- 75 cm/sec.

Once Phase contrast MR images were acquired they post processed using a software (in Siemens it is called Argus). ECG gating is done for the patient which monitors heart rate also.

The parameters that are measured in Phase contrast study are **(Lotz et al[24])**

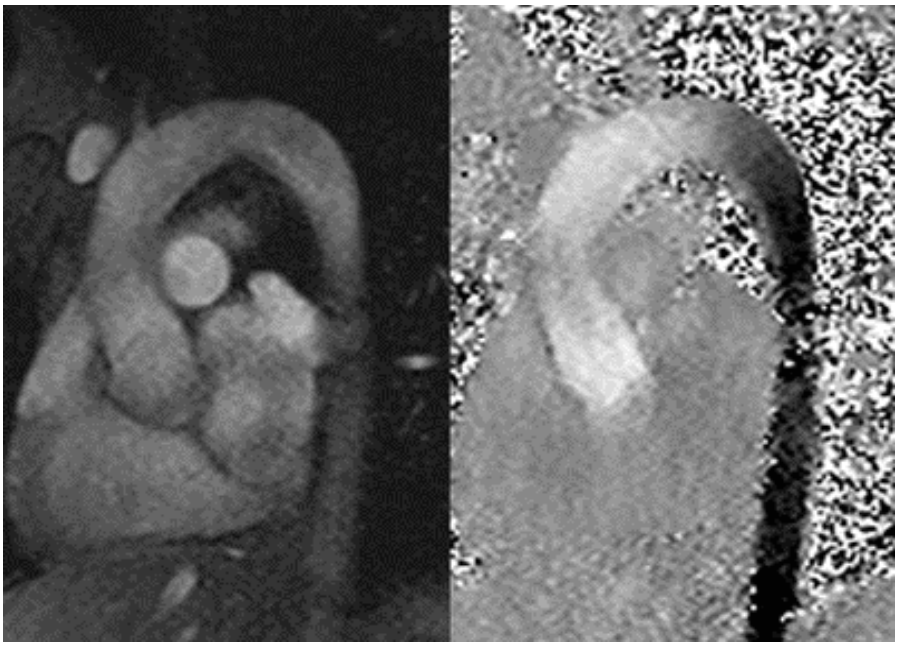
1. Peak velocity
2. Average velocity
3. Maximum area of vessel (in cross section)
4. Minimum area of vessel
5. Average area of vessel
6. Strain

7. Acceleration time (AT)
8. Ejection time (ET)
9. AT/ET ratio
10. Average velocity during AT
11. Average velocity during ETS

PHASE CONTRAST FLOW VELOCITY IMAGING

In plane imaging : Flow qualitative assessment

Through plane imaging : Flow quantification

A.  **B.**

A. In plane imaging
B. Through plane imaging

Though phase contrast MR imaging is very important role in quantitative information on blood flow , there are few common errors like mismatched velocity encoding either too high or too low , devaiation of imaging plane, inadequate spatial and temporal resolution, spatial mis-registration, phase offset errors. However these errors can be reduced by appropriate method and technique . Flow quantification usually done in the plane perpendicular to the vessel and the sequence should be repeated more than once with an higher Venc initially.

"Flow measurements done in vivo are based on results of phantom studies" (**Bakker CJ et al**[25]). "Using phase-contrast measurement, **Evans et al**[26] found a 5% difference between flow in the ascending aorta and flow in the pulmonary artery in healthy volunteers. A deviation from the true flow of 3.5%–4.5% was estimated as the inborn technical error of phase-contrast measurement when a non–breath-hold cine gradient-echo sequence with prospective gating is used". **Kondo et al**[27] "found similar deviations between flow in the aorta and flow in the pulmonary trunk. Using retrospective gating and a breath-hold sequence (FastCine PC; GE Medical Systems, Milwaukee, Wis), we found a 3% difference between flow in the ascending aorta and flow in the pulmonary artery with an intraobserver variability of 2% and interobserver variability of 3%"

Phase-contrast techniques is superior to Doppler ultrasound in terms of measurement of mean flow. However, MR imaging can assess the variation of flow within the vessel in a given spatial resolution. **(28,29, 30)**

ERRORS AND LIMITATIONS INCLUDE

- ✓ Encoding velocity too low
- ✓ Encoding velocity too high
- ✓ Inadequate spatial resolution .
- ✓ Accelerated flow & spatial misregistration
- ✓ Inadequate temporal resolution
- ✓ Deviation of imaging plane
- ✓ Phase offset errors

Encoding Velocity Too High

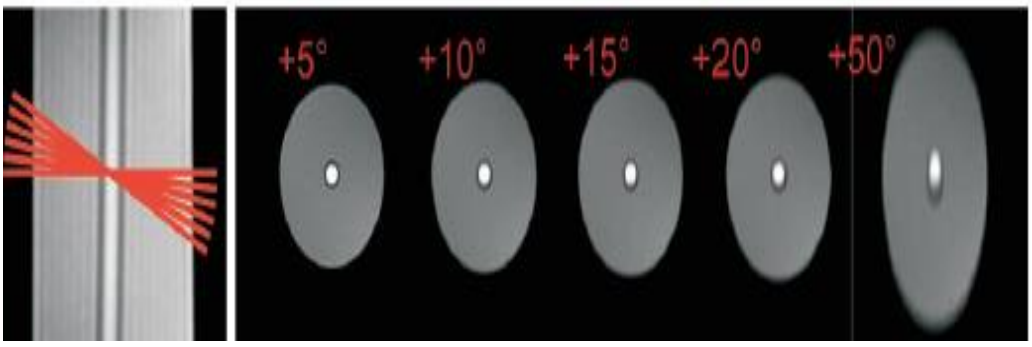
Encoding velocity too high will produce noise. Noise in the velocity depends on velocity encoding value and signal to noise ratio (SNR). It is directly proportional to the velocity encoding and inversely proportional to the signal to noise ratio.

Noise in velocity images increases as Venc values increase.Noise in a velocity decreases with increased signal to noise ratio. An increased SNR is achieved by optimizing parameters like flip angle,section thickness, time to echo. But increasing the time to echo might makes the sequence more prone to movement artifacts.

Encoding Velocity Too low

Encoding Velocity Too low will produce aliasing. It is a wrap around of velocity within a particular voxel. It can affect the results of the flow measurement drastically. But it ie easiest error to be identified and to be corrected. Using some software it can be corrected , however it is more effective to repeat the flow measurtement with a different velocity encoding than to correct the data that is aliased.

Deviation of imaging plane



Measurements of flow are accurate if the imaging plane is orthogonal to the direction of flow. "Quantification of flow is done in through-plane imaging. In-plane imaging is not used for estimation of flow because of increased partial volume effects. Deviation of +/-15° from the orthogonal plane is adjustable for estimation of flow , as the increase in vessel area is compensated by the increase in partial volume effects."

Inadequate Temporal Resolution

"Cardiac-gated flow measurement has a set of frames each of which few show the anatomy and others show the velocity information at a different time in cardiac cycle".

Number of frames to be reconstructed is assigned by the imaging unit and it cannot be altered by the user, like breath hold sequence. Usually a value of 30 frames is sufficient for post-processing time.

Inadequate Spatial Resolution

"When the pixel size exceeds one-third of the vessel diameter Significant partial volume effects must be expected **(Tang C et al[31]).** For a vessel diameter of 3.8 mm for a normal anterior descending branch of the left coronary artery **(Leung WH et al[32]),** an in-plane resolution of 1.2

mm is required". Partial volume effects canunder estimate peak velocity with decreasing spatial resolution. However when flow or peak velocity is measured in the great vessels, this error usually does not occur.

Accelerated Flow and Spatial Misregistration

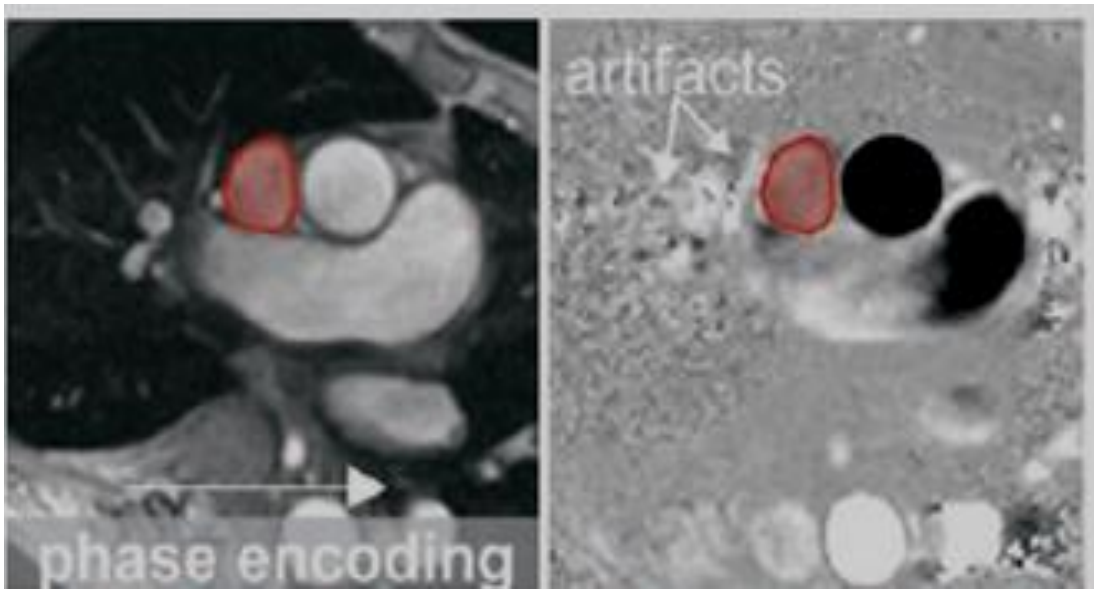
Phase-contrast measurements are generally standardised for linear flow. In case of turbulence or stenotic jets producing accelerated flow causes the precision of the flow measurement to decline **(Hoogeveen RM et al[33]),**however this error can be corrected decreasing echo time **(Mohiaddin RH et al[34]).**

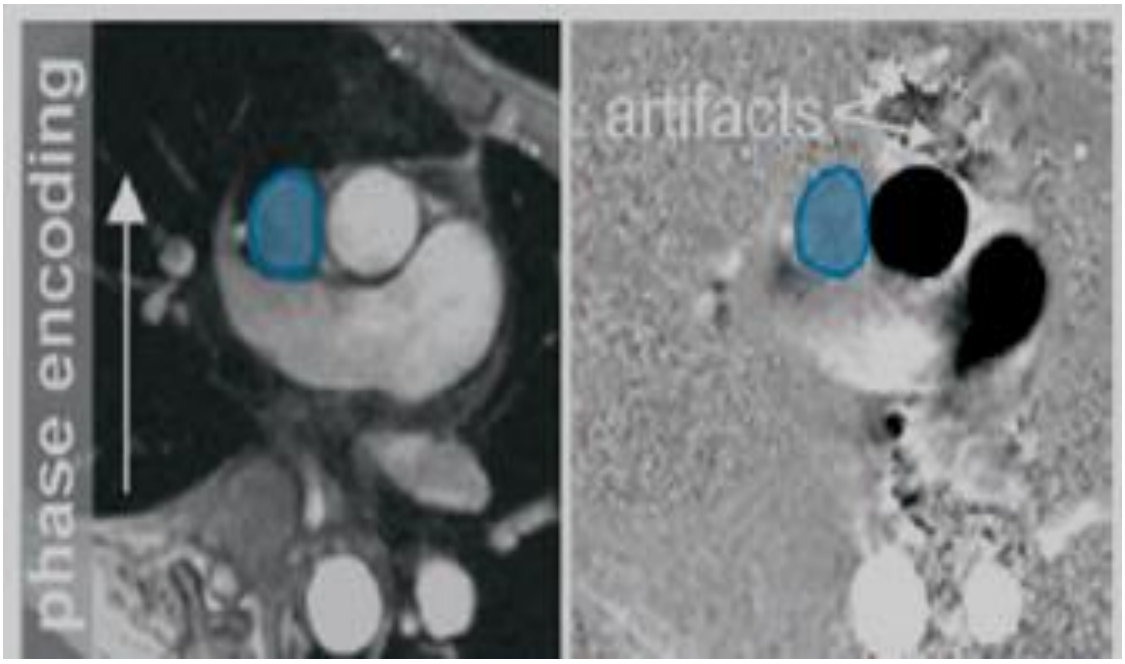
Great vessels like ascending or descending aorta will cause severe problem of constant pulsation artefact along the phase-encoding direction.

Phase Offset Errors

It is due to phase error of moving as well asstationary spins. The phase offset error dependent on gradient imbalance and magnetic field in homogeneities . "It may be detected if the soft-ware for analysis supports a profile representation of the velocity images. In some cases, it may be compensated for by thoroughly applied back-ground subtraction"

PULSATION ARTIFACT





Guidelines forMeasurement and Data Analysis (Lotz et al[35])

As mentioned earlier, flow measurements are more accurate if the flow encoding is set to through-plane flow and if the imaging plane is at right angles to the vessel of interest. Generally the option of reconstructing the components of flow is done separately in each of the three imaging axes s in-stead of producing one summarized data set comprising the net through-plane flow.

"To minimize partial volume effects section thickness of 7 mm used but maintain a good signal to noise ratio in the magnitude image. For small vessels like the coeliac, thickness decreased to adjust the partial volume effects to the small diameter. The choice of the matrixand field of view is related to the anatomy".

"The sequence has to be repeated at least once for precise flow measurements. First measurement, use a high Venc and quick analysis of the data will give an idea of the peak velocities in the vessel. Adding 10% to the high velocity prior will account the physiologic variation of the peak velocity during measurement. This is mandatory for estimation of peak velocities."

In magnitude images the arteries should be round, check for appropriate vessel contour , and look for phase overlap artifacts. Always the images used to prescribe a phase-contrast measurement should be documented, so as to retrospectively look for errors if any.

“Special software programs usually support the drawing of contours (ROIs) into the vessel of interest they automatically copy image the contours from velocity tomagnitude images and vice versa. For large vessels like the aorta or pulmonary vessels the automatic contour detection works reasonably well.”

A small ROI within the vessel is sufficient for estimation of the peak velocity, it will give better results than a ROI encompassing the entire vessel, as arbitrary phase values in the tissue adjacent to the vessel are well avoided **(Hamilton CA et al[36]).**

Background Compensation

- ✓ In phase-contrast flow analysis with availability of commercial software package , we can do background subtraction.
- ✓ The background area should be large , so as to perform background subtraction with out affect the image quality.
- ✓ The mean phase information is subtracted from the area of the vessel.
- ✓ It should be in close proximity to the ROI.
- ✓ These criteria are difficult to maintain within the heart or mediastinum but easy to maintain in the abdomen or extremities imaging.
- ✓ Though it is a powerful tool, it should be used with great caution, so as to avoid phase errors.
- ✓ Generally background compensation is to be avoided and limit its use to the rare cases.

FLOW PARAMETERS AND PULMONARY HYPERTENSION

"In study conducted by **Bouchard A et al**[37] that the average velocity of PA flow was the most useful single parameter in the evaluation of PAH. The presence of slow pulmonary flow in patients with PH was observed in early investigations by using spin-echo MR techniques".

"An **average velocity cut off value of 11.7 cm/s** revealed pulmonary arterial hypertension with 92.9% sensitivity and 82.4% specificity **(Javier Sanz et al**[38]**).**In patients with chronic PAH, a variety of flow measurements in the pulmonary trunk evaluated with phase-contrast MR imaging correlate with the degree of hemodynamic disturbance as determined with the level of pulmonary pressures and vascular resistance. The average blood velocity throughout the cardiac cycle is strongly correlated with pulmonary pressures and resistance and appears to have consistent performance across different subgroups of patients".

"PA area in detecting the presence of PAH (sensitivity and specificity of 92.9% and 88.2%, respectively).However, the use of PA areas had some limitations, including inconsistent performance and more

difficult contour tracing in diastole when proton inflow (and therefore blood signal intensity) was lower .”**(Laffon E et al[39])**

SPECIFIC PROTOCOLS

Sequence and Parameters Used for Phase-Contrast Flow Measurement

Sequence	FastCine PC*
k-space segmentation (views per segment)	4–12†
View-sharing interpolation (reconstructed frames per cardiac cycle)	30
Velocity encoding (cm/sec)	30–550
Field of view (mm)	260–340‡
Matrix	256 × 160
Section thickness (mm)	7
Repetition time (msec)	7§
Echo time (msec)	3.7§
Signals acquired	1
Flip angle (degrees)	40
Imaging time (sec)	<24‖

*GE Medical Systems. Retrospective electrocardiographic gating and view-sharing interpolation are also used.
†Depends on the heart rate and the ability to breath hold.
‡Depends on the size of the patient, the imaging plane selected, and the surface coil used.
§Minimum values are used for all measurements.
‖Depends largely on the pulse rate and the k-space segmentation.

Use of minimum values for time to repeat and time to echo improves the temporal resolution. Therefore, to control excessive noise in the images it i necessary to increase the echo time depending on the MR imaging system used.

SVC and Inferior Vena Cava

“For the first measurement the encoding velocity for flow measurement is 110 cm/sec. The usual velocity for peak velocity is 50–80cm/sec. For the SVC, the imaging plane is selected below the influx of the azygos vein, with the azygos vein seen in the magnitude image.

For magnitude imaging of the inferior vena cava (IVC), imaging plane is selected above the hepatic vein influx. The imaging should cover the ventral wall of the IVC”.

Ascending Aorta

“The encoding velocity for the first measurement for flow measurement is 200 cm/sec. The usual velocity for peak velocity measurement is 100–160 cm/sec choice of the localizer for the pulmonary trunk is crucial. It sometimes helps to choose a plane in the right ventricular outflow tract to visualize the pulmonary valve and pulmonary bifurcation”.

Pulmonary Arteries

"The encoding velocity for the first measurement for flow measurement is 200 cm/sec. The usual velocity for peak velocity measurement is 60–120cm/sec."

"In a healthy adult, 55% of pulmonary flow goes through the right pulmonary artery and 45% goes through the left pulmonary artery"**(Henk CB et al[40]).**

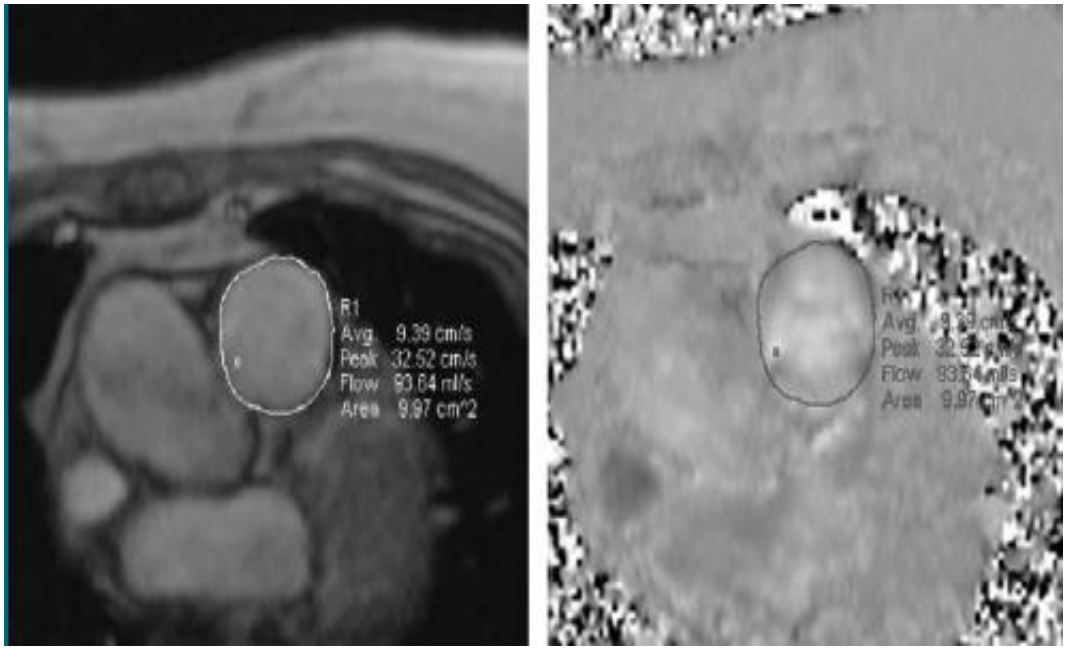
Small Intracardiac Structures

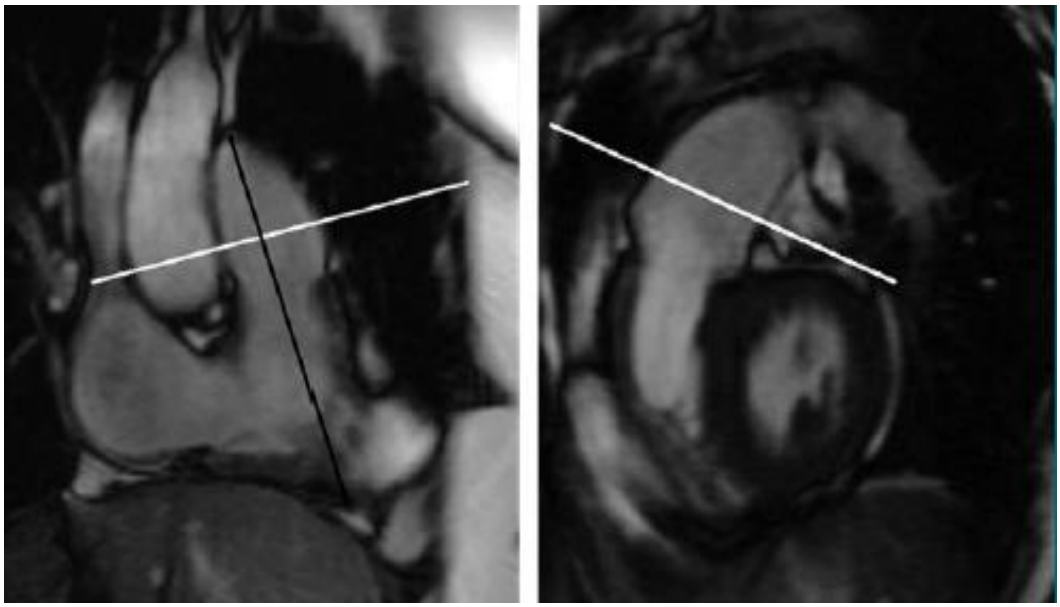
- ✓ Spatial resolution and temporal resolution are the most important sources of errors for small structures like shunts, grafts, bypasses
- ✓ Qualitative flow curve estimation is usually performed for even very small structures.
- ✓ Because of their complex movement of coronary arteries and the coronary sinus pose an additional problem **(Clarke GD et al[41]).**
- ✓ Retrospective cardiac gating and interpolation can produce more than 30 frames per cardiac cycle in a single breath hold.

- ✓ Use of phased-array coil for image reconstruction allows reduction of the field of view to about 22–24 cm without significant artifacts

- ✓ “Whenever measurements are performed in a breath hold, one should keep in mind that a pro-longed breath hold affects hemodynamics and may alter results even for qualitative measurements”.

Main pulmonary artery





IMAGING PLANE FOR MAIN PULMONARY ARTERY

“The encoding velocity for the first measurement for flow measurement is 180 cm/sec. The usualvelocity for peak velocity measurement is 60–120cm/sec.

Use an oblique localizer on an axial or coronal image to visualize the natural bowing of the pulmonary artery . On the resulting image, select an imaging plane above the pulmonary valve but below the pulmonary bifurcation”.

“Phase contrast MRI has been used to study hemodynamic changes associated with PAH. In a recent study of 25 patients with PAH, mean pulmonary artery (PA) peak flow velocity, PA blood flow, and PA distensibility were found to be significantly lower than in a matched group of volunteers (p _ 0.002, p _ 0.002, and p _ 0.008, respectively) (**Schwitter J et al**[42]). This study also documentedreduced time to peak PA velocity and steeper velocity increase gradient among PAH patients. Another study compared fast-gradient echocardiographic phase-contrast MRI and radionuclide lung perfusion for assessing differentialbranch pulmonary blood flow in 12 children with suspected unilateral branch pulmonary artery stenosis.” (**Hundley WG et al**[43]).

Parameters of Pulmonary Hypertension by Noninvasive Imaging Method (Sallach SM et al[44])

Parameter	Noninvasive Imaging Method of Choice	Optimal View or Imaging Window
Anatomy		
Pulmonary artery size	cMRI	High axial image
Qualitative RV size, hypertrophy, trabeculations	Echocardiography	Apical 4-chamber, parasternal long-axis
		4-chamber, short-axis series for volumes, function, and masses
Quantitative RV size, mass, trabecular mass	cMRI	
Function	cMRI	4 chamber shortaxis view
RV free wall motion		
Interventricular septal motion	cMRI	echocardiography
		4-chamber, short-axis
Qualitative systolic function	Echocardiography	4-chamber , parasternal short-axis
Quantitative systolic function	cMRI	Short-axis series
Diastolic function	Echocardiography	AV valve inflow parameters, tissue Doppler
Hemodynamics		
Pulmonary valve motion	Echocardiography	M-mode
RVOT flow	Echocardiography	pulse wave doppler
Pulmonary artery pressure	Echocardiography	TR,PR velocities any flow in to right heart

Current Issues in Cardiovascular Magnetic Resonance Imaging

- Large number of technical parameters that the scanner operator needs to know
- Large number of parameter choices results in different imaging sites using protocols with different parameters, making a comparison of images difficult
- Intensive training is required for scanner operators
- Lack of a standardized viewing and reporting format
- Imaging centers have to create their own viewing tools
- Viewing takes so much time that it limits patient throughput
- Lack of a standardized nomenclature for magnetic resonance sequences and parameters
- Vendors choose different names for the same or similar techniques to differentiate them from their competitors
- Focus on research
- Small number of current procedural terminology codes, each with limited scope

- Current procedural terminology codes have not kept up with technological developments

"In a study conducted by **Sanz et al[45]** the minimum PA area was useful in detecting the presence of PAH (sensitivity and specificity of 92.9% and 88.2%, respectively). And average velocity of PA flow was the most useful single parameter in the evaluation of PAH. The presence of slow pulmonary flow in patients with PH was observed in early investigations by using spin-echo MR techniques **(Bouchard et al[46])** or velocity mapping **(Bogren et al[47]).** In a group of 33 patients with chronic thromboembolic disease, PA peak velocity had a moderate correlation(*r* _ 0.60) with mPAP **(Kreitner KF et al[48])**. There results in a larger group of patients with PAH confirm a significant though weaker correlation between peak velocity and pulmonary pressures and resistance, whereas average velocity had the best correlations with hemodynamic measurements (*r* ranging from _0.73 to0.86). These findings suggest that as pulmonary pressures and resistance increase, the circulation of blood through the pulmonary vascular bed is globally hampered and progressively slows. A potential role for the quantification of average velocity at peak systole in PH evaluation has been previously suggested **(laffon E et al[49]).** However this is the first investigation to evaluate average velocity during the complete cardiac cycle."

"In a study by **Fine et al**[50] on use of RV strain imaging to risk stratify patients with pulmonary hypertension in the absence of pulmonary venous hypertension promises to extend the usefulness of 2D echo in this setting at the present time. However, it remains uncertain whether this will lead to the desired result in the long term: a definitive method for prediction of outcomes in pulmonary hypertension. Nor is it clear whether progress in application of 3DRV or 2D knowledge-based volumetric imaging will super cede it or whether advances in image-based evaluation of RV loading conditions will permit meaningful assessment of RV contractility in the future."

With the advent of cardiac MRI around 1980s which led to rapid development of volumetric 3D imaging for left ventricular (LV) size, function, and mass followed by assessing 3D LV strain which considered as gold standards in the cardiovascular armamentarium. But even so, imaging of RV volume and mass remained more reliable whereas RV free wall strain assessment remained elusive with CMR until now. Now with the emergence of echo RV free wall strain imaging has recognised**.** **(Shehata ML et al**[51]**)**

Metrics that were directly measured in all vessels in phase contrast study were forward-flow volume(FFV) and backward-flow volume (BFV); all other metrics were calculated as follows:

1. Pulmonary forward flow volume (PFFV)=RPA FFV+LPA FFV,

2. Pulmonary backward flow volume (PBFV)=RPA BFV+LPA BFV,

3. Net pulmonary flow volume (NPFV)=PFFV−PBFV,

4. Pulmonary artery regurgitation fraction (PRF)=(PBFV/ PFFV)×100,

5. Tricuspid valve regurgitation fraction (TRF)=(RVSV−PFFV/ RVSV)×100.13

American College of Radiology
ACR Appropriateness Criteria®

Clinical Condition: **Pulmonary Hypertension**

Radiologic Procedure	Rating	Comments	RRL*
US echocardiography transthoracic resting	9	Catheterization and echocardiography are complementary examinations. Both should be performed. Echocardiography is typically performed before catheterization.	0
Right heart catheterization	9	Catheterization and echocardiography are complementary examinations. Both should be performed. Echocardiography is typically performed before catheterization.	☢☢
X-ray chest	8		☢
CTA chest with contrast	8		☢☢☢
Tc-99m V/Q scan lung	7		☢☢☢
MRI heart function and morphology without contrast	6	May be performed with MRA.	0
MRI heart function and morphology without and with contrast	6	See statement regarding contrast in text under "Anticipated Exceptions."	0
MRA pulmonary arteries without and with contrast	6	See statement regarding contrast in text under "Anticipated Exceptions."	0
Arteriography pulmonary with right heart catheterization	6		☢☢☢☢
CT chest without contrast	5	If there is a concern for an occult ILD, HRCT may be appropriate.	☢☢☢
US echocardiography transesophageal	5		0
MRA pulmonary arteries without contrast	2		0
CT chest without and with contrast	1	CT chest with and without contrast does not always provide the same information as a CTA chest.	☢☢☢

Rating Scale: 1,2,3 Usually not appropriate; 4,5,6 May be appropriate; 7,8,9 Usually appropriate

*Relative Radiation Level

AIM OF THE STUDY

1. To assess the pulmonary arterial flow parameters measured using phase contrast magnetic resonance imaging (MRI)

2. To correlate the size of the main pulmonary artery as measured using spiral computerised tomography (CT) with the flow parameters as measured using phase contrast MRI and also with ECHO parameters.

MATERIALS AND METHODS

STUDY DESIGN **:** PROSPECTIVE OBSERVATIONAL

PLACE OF STUDY **:**BARNARD INSTITUTE OF RADIOLOGY,

RAJIV GANDHI GOVERNMENT

GENERAL HOSPITAL, CHENNAI

DURATION OF STUDY : 6 MONTHS

STUDY SIZE : 50 PATIENTS

STUDY POPULATION

INCLUSION CRITERIA:

- Age more than 20years
- The patients who are found to have dilated main pulmonary artery (2.9cm) in CT chest study.
- Those patients who are admitted with pulmonary hypertension of various etiology were evaluated
- Those patients who suspected to have pulmonary hypertension
- subjects with normal pulmonary artery diameter are also included in the study, with no history of chronic lung disease or heart disease

EXCLUSION CRITERIA

- Patients with pace maker, metallics implants, clips for intracranial aneurysm
- Patients having claustrophobia
- Pregnant patients

- Patients who are sick

- Patients with acute pulmonary thromboembolism.

STUDY PROCEDURE

OUTLINE

- After informed written consent, all patients underwent MRI Phase contrast flow velocity imaging including in plane and through plane imaging.

- With Mean VENC value for main pulmonary artery 75-100 cm/sec

- Post processing done with a commercial software ARGUS

- Patients ECHO findings reviewed.

- Control subjects underwent CT chest followed by MRI phase contrast imaging and ECHO

MACHINE :

Studies were performed with

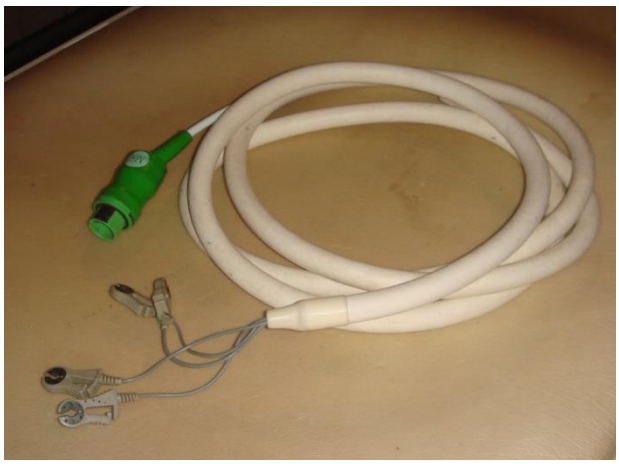
- 1.5-T clinical magnet Magnetom symphony; Siemens Medical Solutions, Erlangen, Germany.
- CT scanner TOSHIBA (Asteion)

After evaluating the pulmonary artery size in contrast enhanced CT chest study, the patients are examined whether they are in sinus rhythm and they are made to liecomfortably in supine position. Phasedarray surface coil is used as a receiver. ECG leads (3 in number) are attached to the patients chest in the posterior aspect. The patients are instructed to perform end-expiratory breath holds precededby brief hyperventilation. After acquiring Standard localizer views , axial four chamber view of the heart is acquired. Then two double-obliqueviews oriented along the main axis ofthe pulmonary trunk were acquiredwith a standard steady-state free precession cine MR sequence . The imaging plane should be perpendicular tothe main PA for the acquisition ofphase-contrast MR images.

Phase-contrast MR images were acquiredwith a segmented fast gradientecho MR sequence , with velocityencoding perpendicular to the imagingplane and with velocity encoding of 75 – 100 cm/sec. In Contrast enhanced CT CHEST transverse diameter of the the main

pulmonary artery is measured in axial sections at about 2cm proximal to its bifurcation in to right and left pulmonary artery.

ECG LEADS

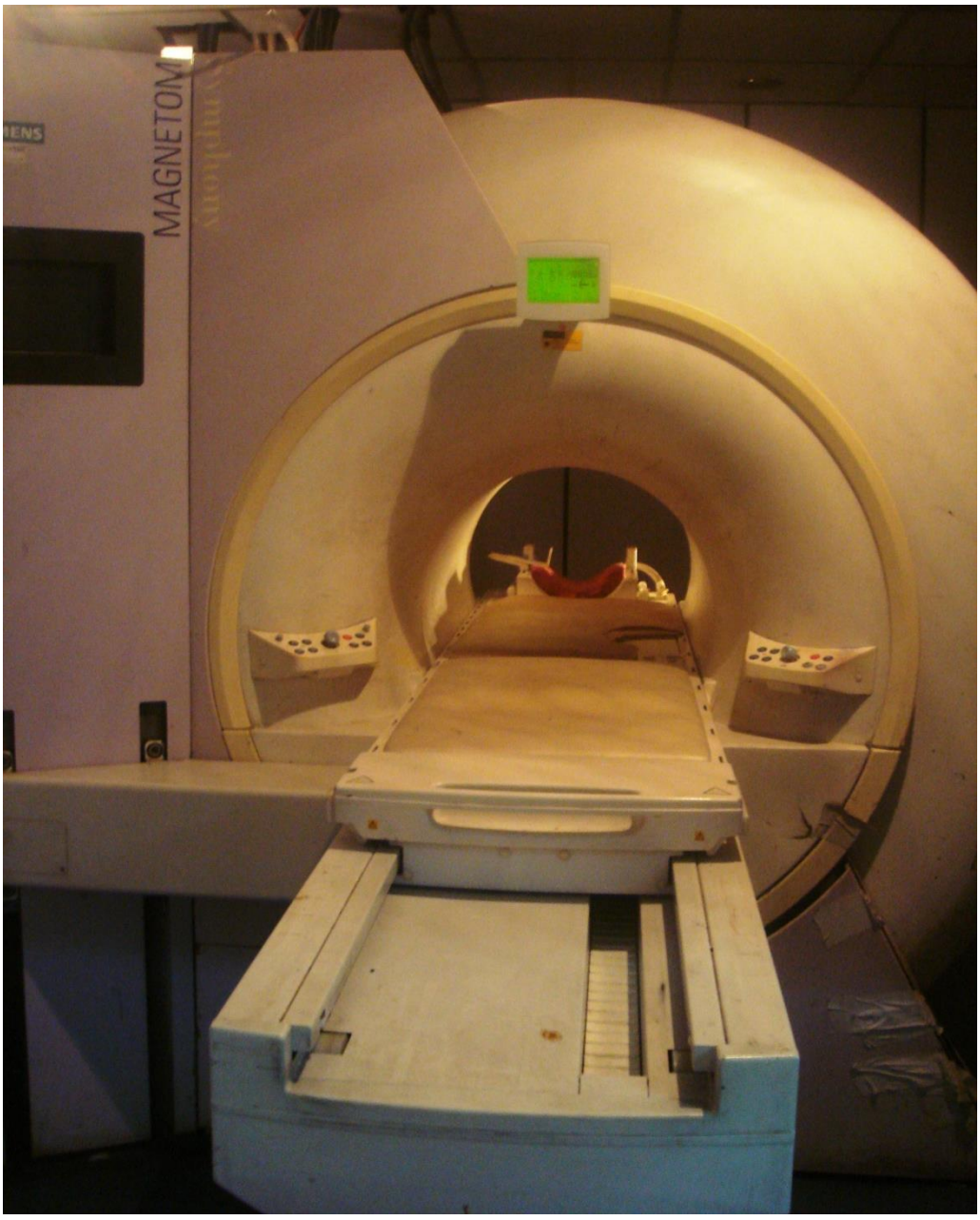


ECG CONNECTOR

PHASED ARRAY COIL

MRI

1.5 TESLA CLINICAL MAGNET, MEGNETOM SYMPHONY (SIEMENS)



DATA INTERPRETATION AND ANALYSIS

MRI PA FLOW PARAMETERS

1. Average velocity

2. Average pulmonary artery area

3. Peak velocity

4. Maximum pulmonary artery area

5. Minimum pulmonary artery area

CT

1. Size of the main pulmonary artery

ECHO

1. TRPG

2. TAPSE

3. Presence or absence of A wave in the M mode.

PARAMETERS	CUT OFF VALUES
ECHO	
1.TRPG	Less than 25mmHg - normal
2. ‘a’ wave	Present - normal
3.TAPSE	More than 20mm- normal
CT	
PA size	Less than 2.9cm - normal
MRI	
1.Average PA velocity	More than 11.7cm/sec - normal
2.Minimum PA area	Less than 10.5cm^2 - normal

CASE 1

Clinical history:

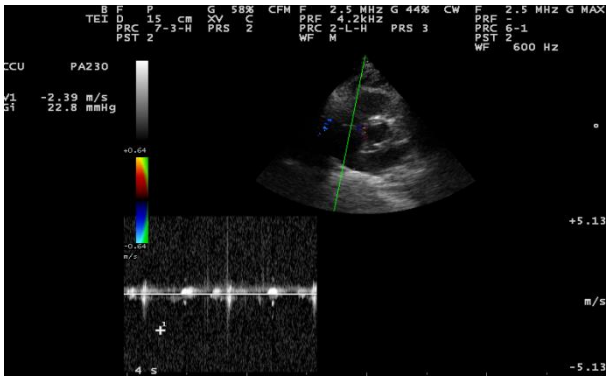
No specific complaints

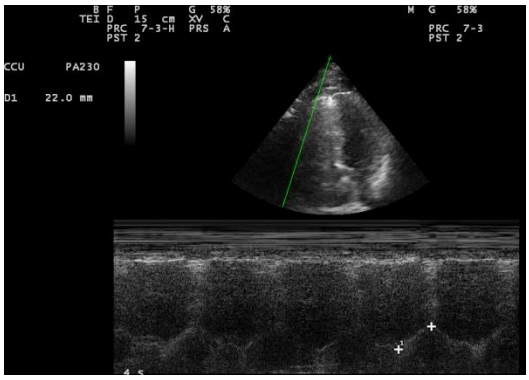
Not a known case of congenital heart disease.

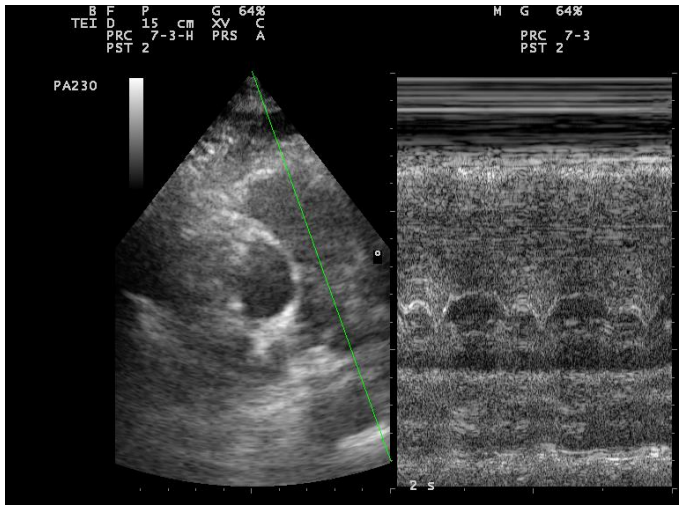
ECHO FINDINGS:

TRPG: 22 mmHg

TAPSE: 2.2 cm



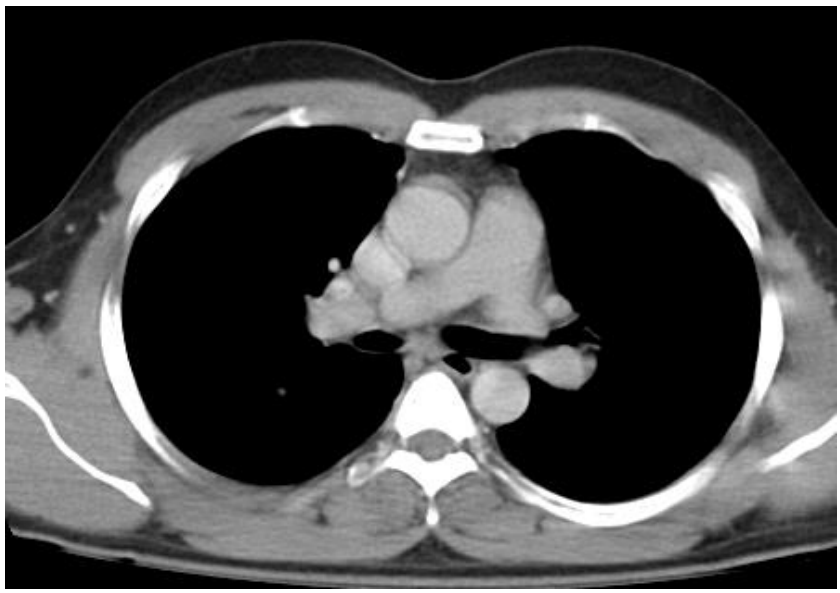




'a' wave present

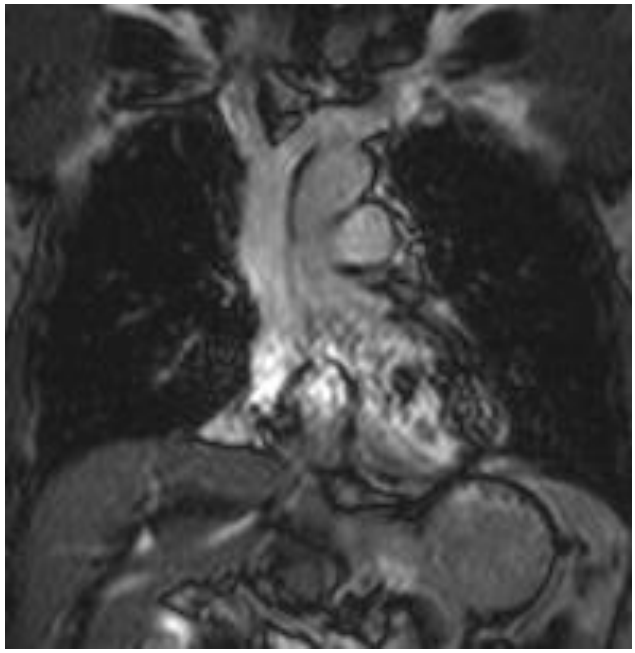
CT:

Main pulmonary artery size: 2.42 cm



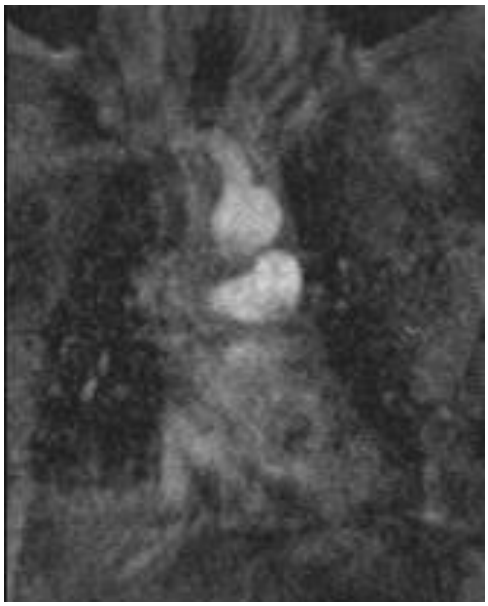
MRI:

Cardiac MRI – TRUFI coronal

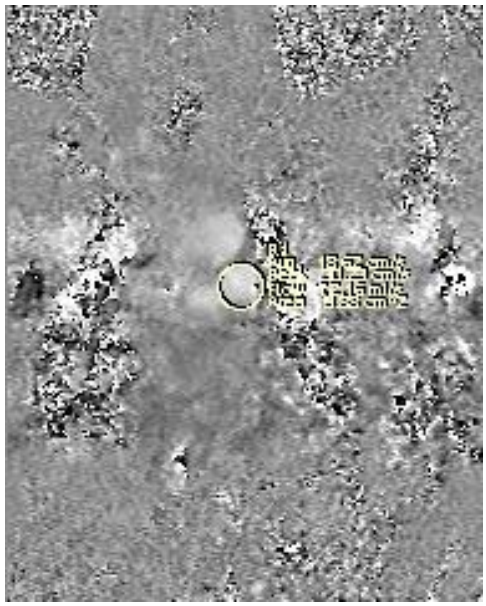


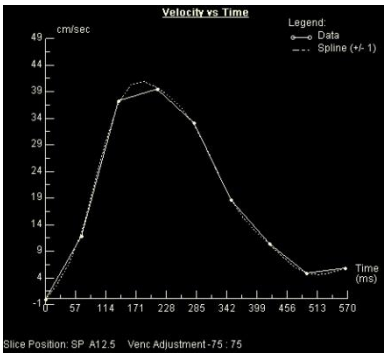
PHASE CONTRAST FLOW STUDY

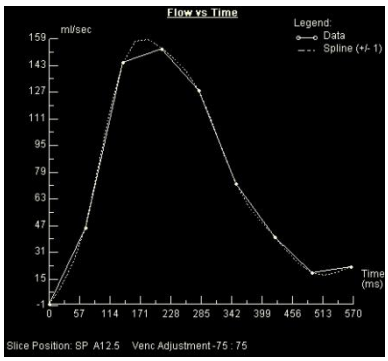
In plane imaging

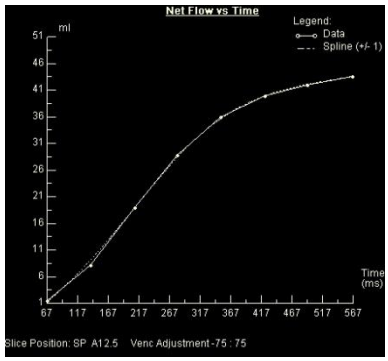


Through plane imaging









PULMONARY ARTERY FLOW PARAMETERS

Slice Position: SP A12.5	Region: 1		
Range,ms: 0 to 565	Venc Adjustment	-75 cm/sec	75 cm/sec
Body Surface Area (BSA):		----	m^2
Velocity			
Peak Velocity:		62.77	cm/sec
Average Velocity:		19.85	cm/sec
Flow			
Average Flow Over Range:		76.95	ml/sec
Average Flow Per Minute:		3.00	l/min
Forward Volume:		43.48	ml
Reverse Volume:		0.000	ml
Net Forward Volume:		43.48	ml
Net Forward Volume / BSA:		----	ml/m^2
Area			
Average Area:		3.88	cm^2
Mininum Area:		3.88	cm^2
Maximum Area:		3.88	cm^2

Average velocity: 19.85 cm/sec

Minimum pulmonary artery (PA) area: 3.88 cm^2

DIAGNOSIS : NORMAL STUDY

CASE 2

Clinical history:

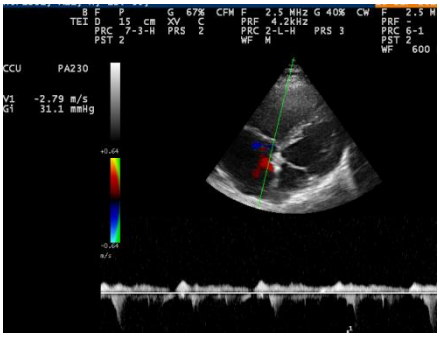
Complaints of breathlessness on & off for past 6 months.

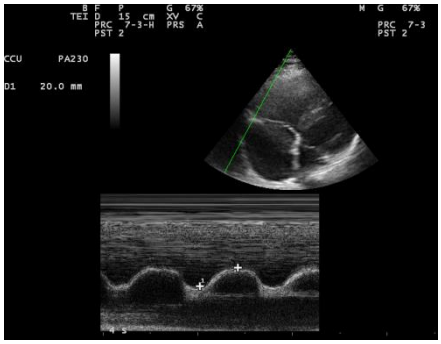
Not a known case of congenital heart disease.

ECHO FINDINGS:

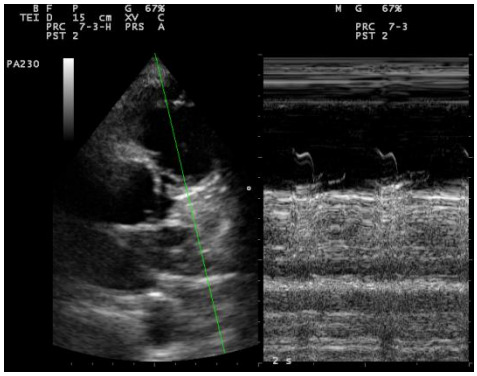
TRPG: 31.1 mmHg

TAPSE: 2.0 cm



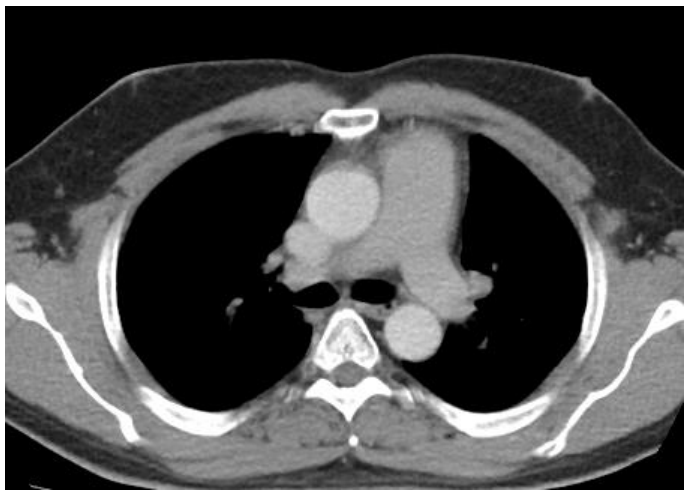


Absent ‘a’ wave



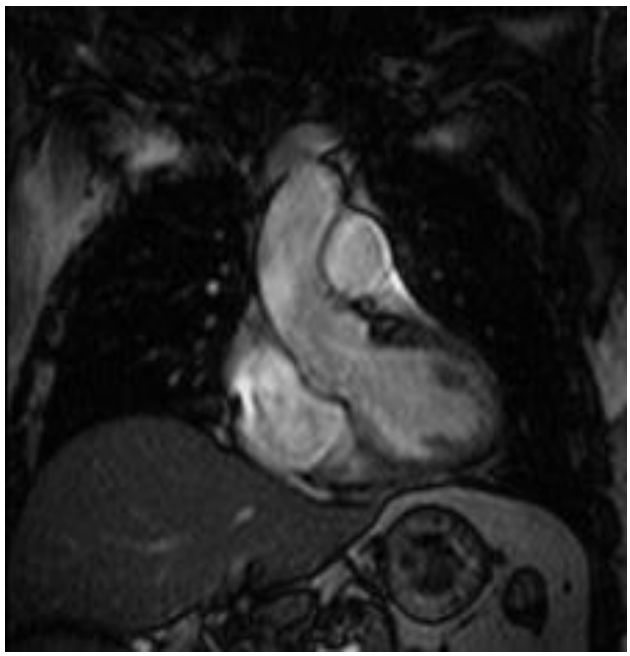
CT:

Main pulmonary artery size: 3.56 cm



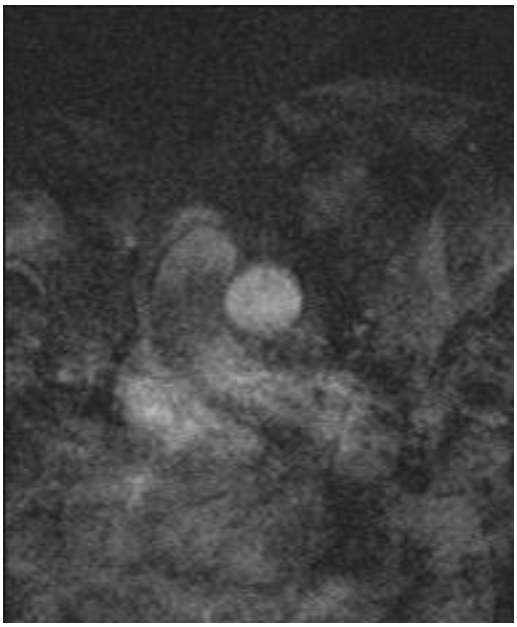
MRI:

Cardiac MRI – TRUFI coronal

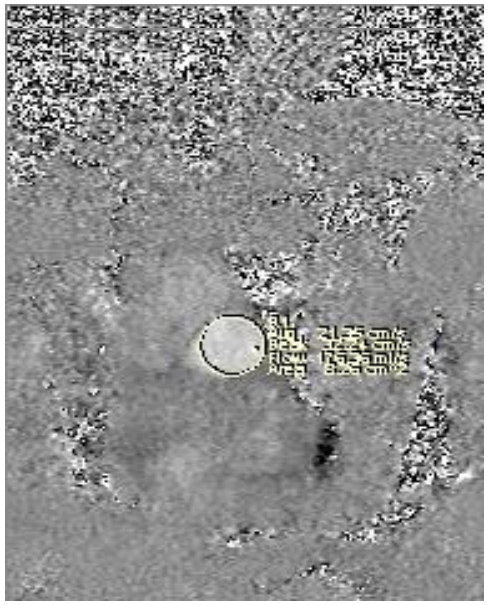


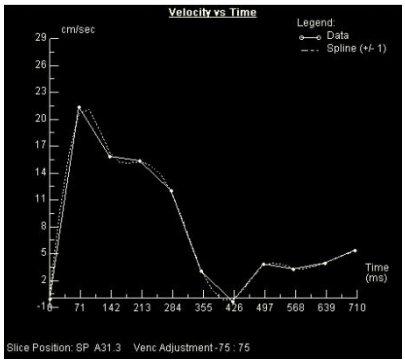
PHASE CONTRAST FLOW STUDY

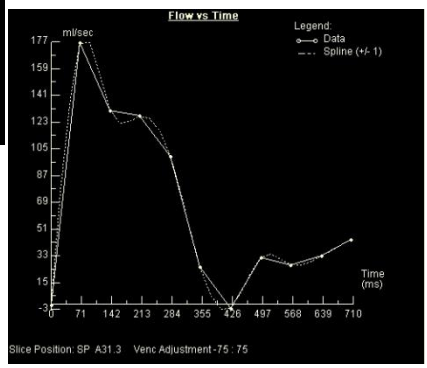
In plane imaging

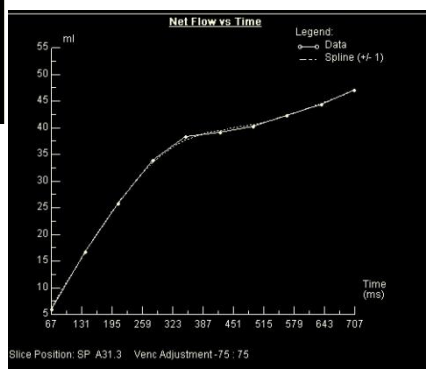


Through plane imaging

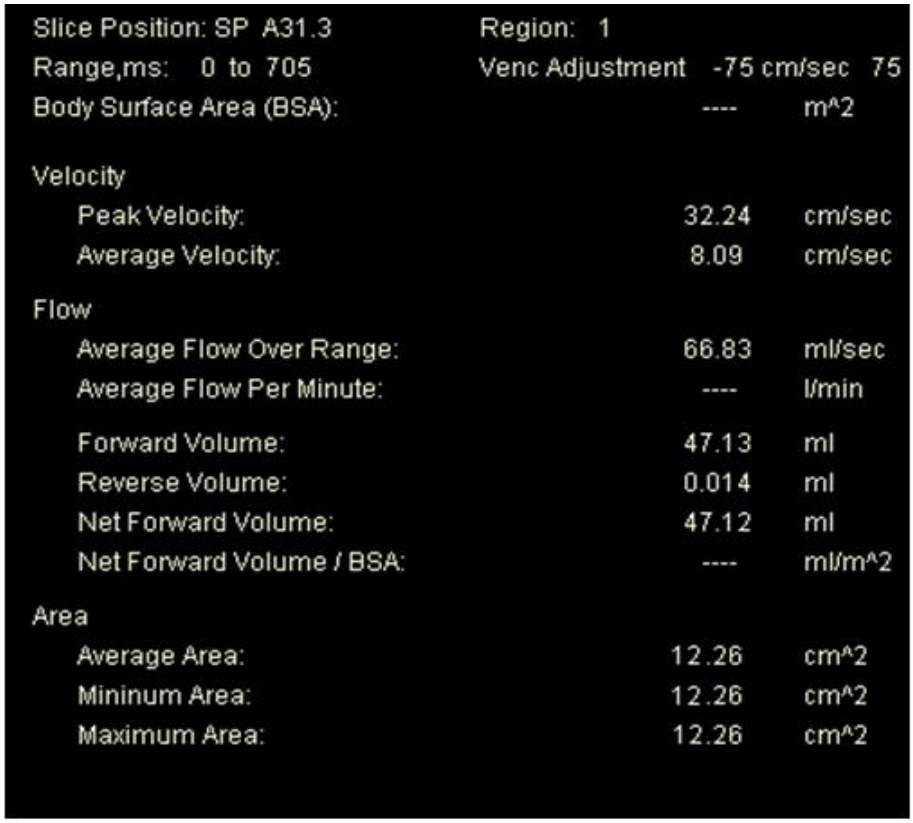








PULMONARY ARTERY FLOW PARAMETERS



Slice Position: SP A31.3	Region: 1	
Range,ms: 0 to 705	Venc Adjustment	-75 cm/sec 75
Body Surface Area (BSA):	----	m^2
Velocity		
Peak Velocity:	32.24	cm/sec
Average Velocity:	8.09	cm/sec
Flow		
Average Flow Over Range:	66.83	ml/sec
Average Flow Per Minute:	----	l/min
Forward Volume:	47.13	ml
Reverse Volume:	0.014	ml
Net Forward Volume:	47.12	ml
Net Forward Volume / BSA:	----	ml/m^2
Area		
Average Area:	12.26	cm^2
Mininum Area:	12.26	cm^2
Maximum Area:	12.26	cm^2

Average velocity: 8.09 cm/sec

Minimum pulmonary artery (PA) area: 12.26 cm2

DIAGNOSIS : PULMONARY ARTERIAL HYPERTENSION

CASE 3

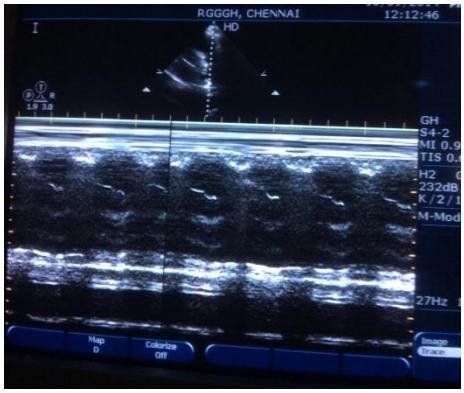
Clinical history:

Complaints of breathlessness on & off for past 6 months.

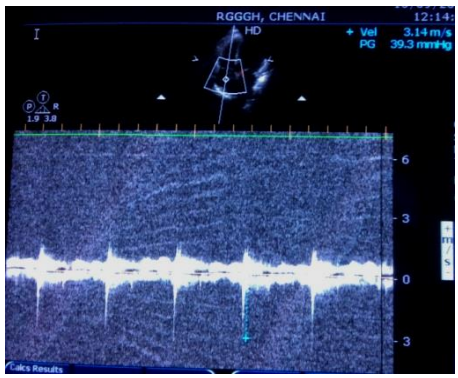
Not a known case of congenital heart disease.

ECHO FINDINGS:

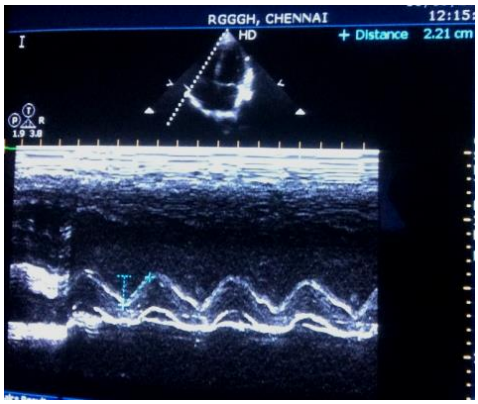
TRPG: 39.3 mmHg



TAPSE: 2.21 cm

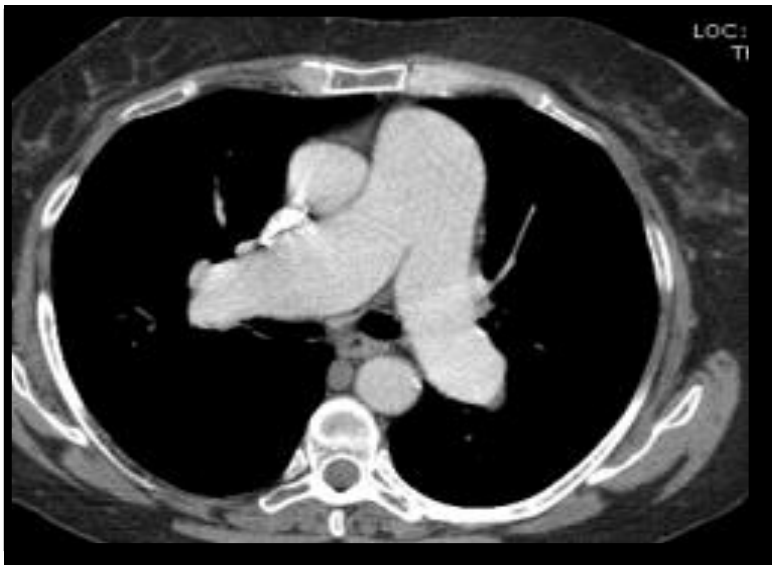


Absent ‘a’ wave



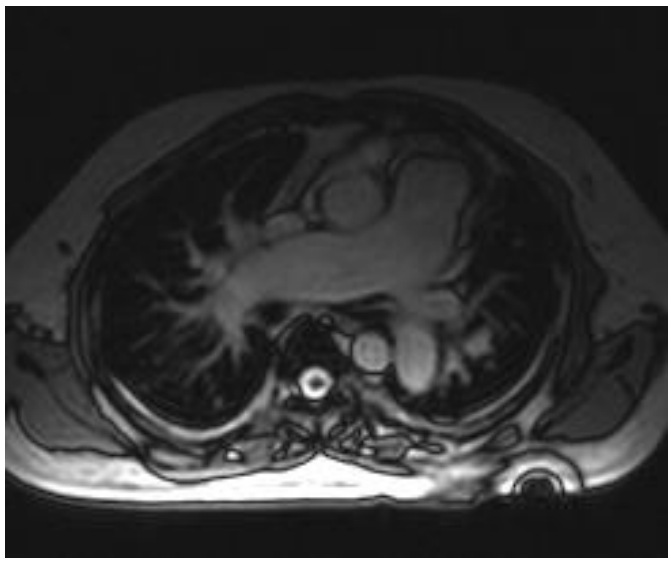
CT:

Main pulmonary artery size: 3.64 cm



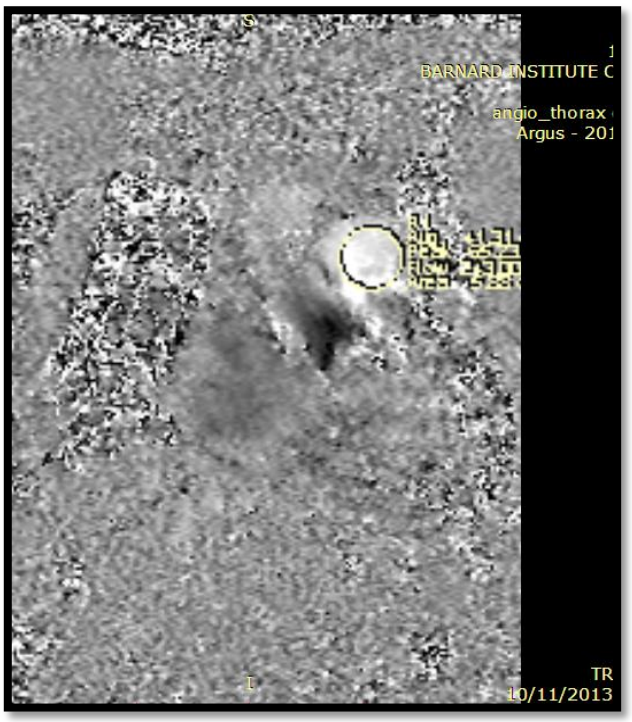
MRI:

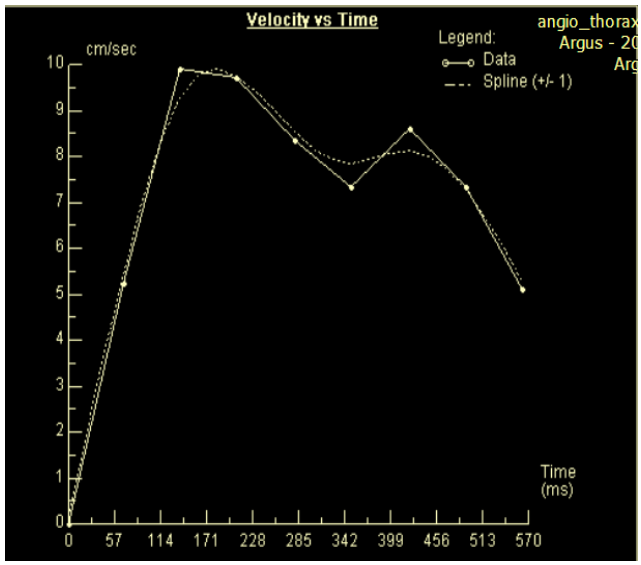
Cardiac MRI – TRUFI coronal



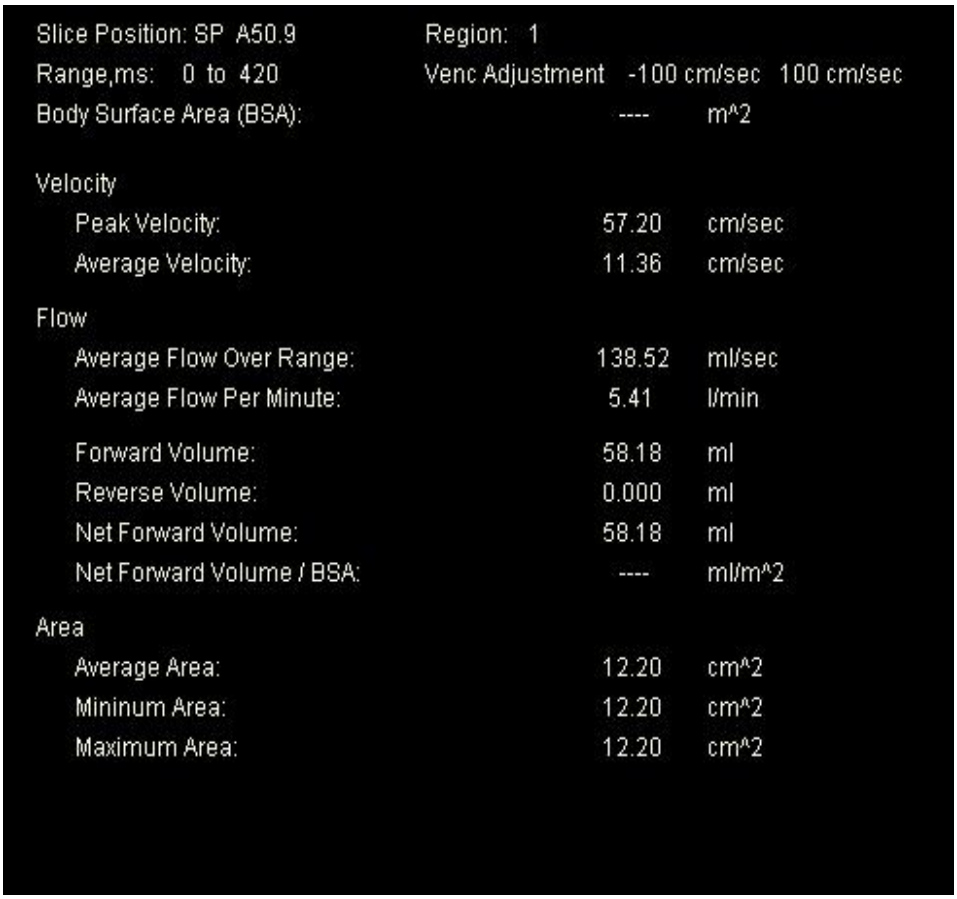
PHASE CONTRAST FLOW STUDY

Through plane imaging





PULMONARY ARTERY FLOW PARAMETERS



Slice Position: SP A50.9	Region: 1	
Range,ms: 0 to 420	Venc Adjustment	-100 cm/sec 100 cm/sec
Body Surface Area (BSA):	----	m^2
Velocity		
Peak Velocity:	57.20	cm/sec
Average Velocity:	11.36	cm/sec
Flow		
Average Flow Over Range:	138.52	ml/sec
Average Flow Per Minute:	5.41	l/min
Forward Volume:	58.18	ml
Reverse Volume:	0.000	ml
Net Forward Volume:	58.18	ml
Net Forward Volume / BSA:	----	ml/m^2
Area		
Average Area:	12.20	cm^2
Mininum Area:	12.20	cm^2
Maximum Area:	12.20	cm^2

Average velocity: 11.36 cm/sec

Minimum pulmonary artery (PA) area: 12.20 cm2

DIAGNOSIS : PULMONARY ARTERIAL HYPERTENSION

CASE 4

Clinical history:

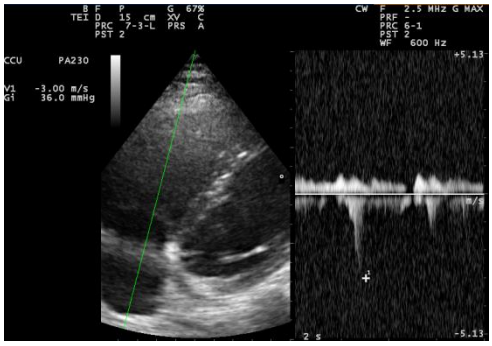
Complaints of breathlessness on & off for past 1 year.

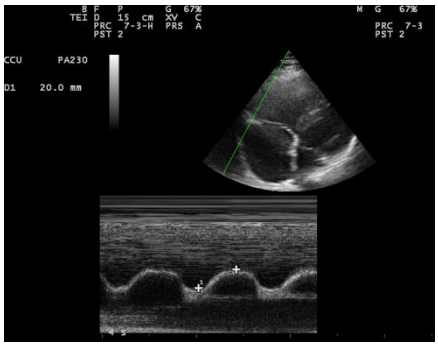
Not a known case of congenital heart disease.

ECHO FINDINGS:

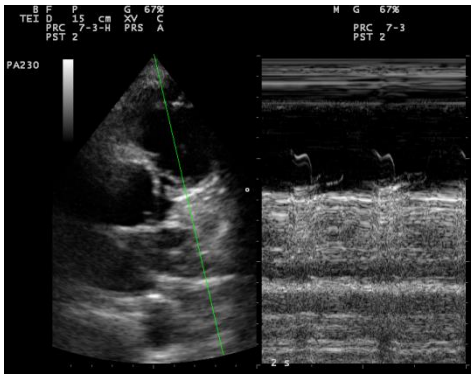
TRPG: 36 .0 mmHg

TAPSE: 2.0 cm



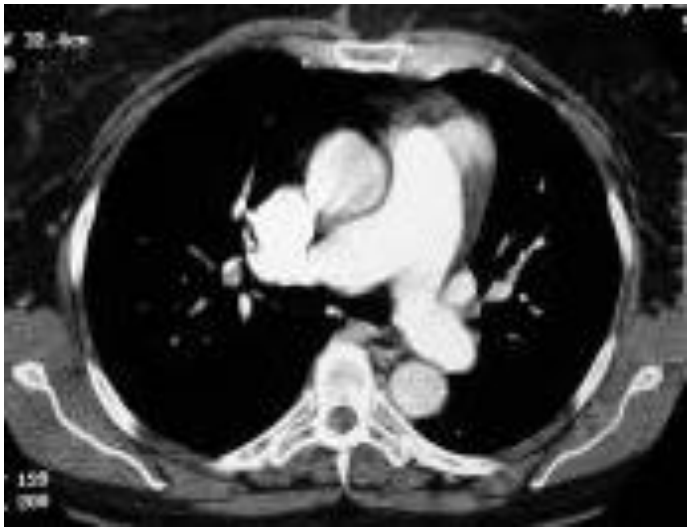


Absent ‘a’ wave



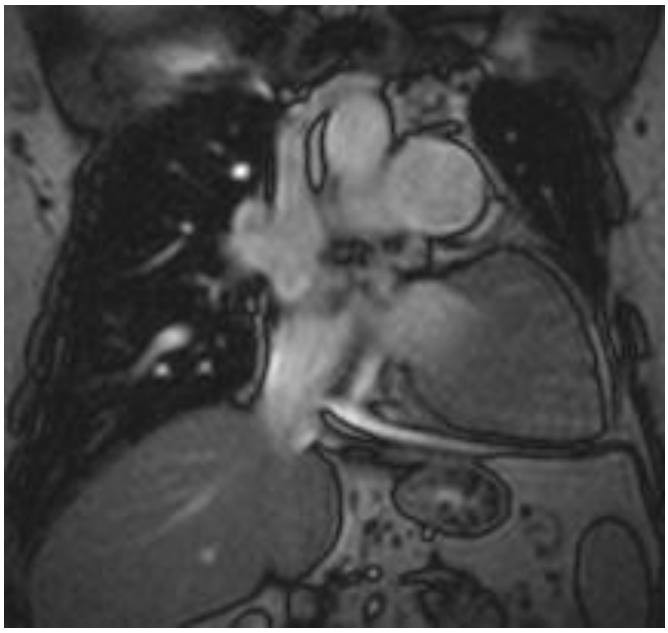
CT:

Main pulmonary artery size: 3.7 cm



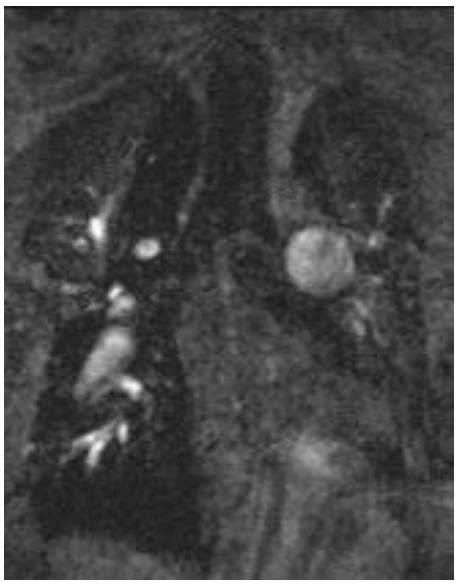
MRI:

Cardiac MRI – TRUFI coronal

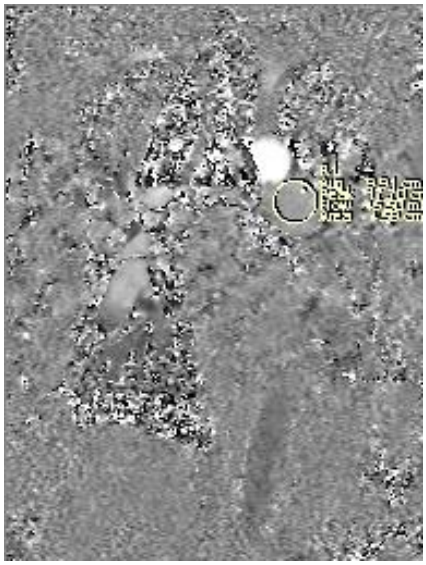


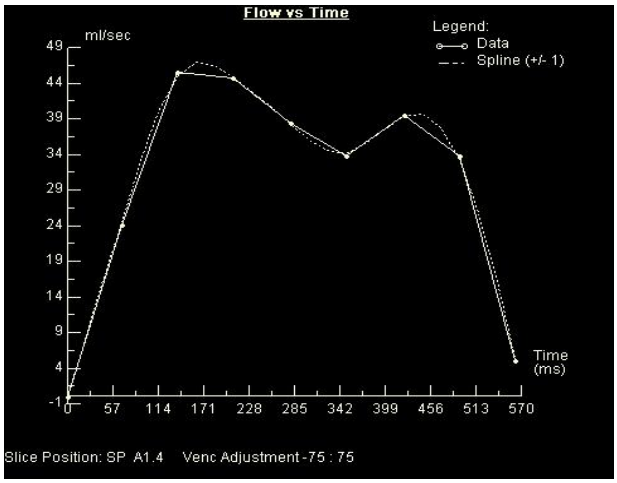
PHASE CONTRAST FLOW STUDY

Through plane imaging

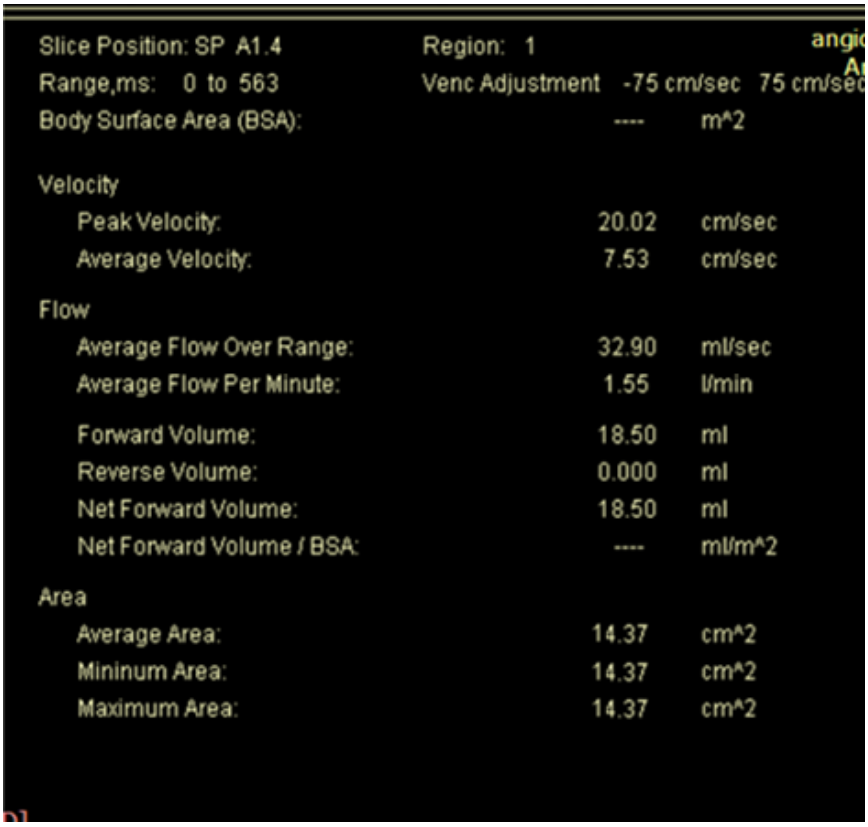


In Plane imaging





PULMONARY ARTERY FLOW PARAMETERS



Slice Position: SP A1.4	Region: 1	angi
Range,ms: 0 to 563	Venc Adjustment -75 cm/sec	75 cm/sec
Body Surface Area (BSA):	----	m^2
Velocity		
Peak Velocity:	20.02	cm/sec
Average Velocity:	7.53	cm/sec
Flow		
Average Flow Over Range:	32.90	ml/sec
Average Flow Per Minute:	1.55	l/min
Forward Volume:	18.50	ml
Reverse Volume:	0.000	ml
Net Forward Volume:	18.50	ml
Net Forward Volume / BSA:	----	ml/m^2
Area		
Average Area:	14.37	cm^2
Mininum Area:	14.37	cm^2
Maximum Area:	14.37	cm^2

Average velocity: 7.53 cm/sec

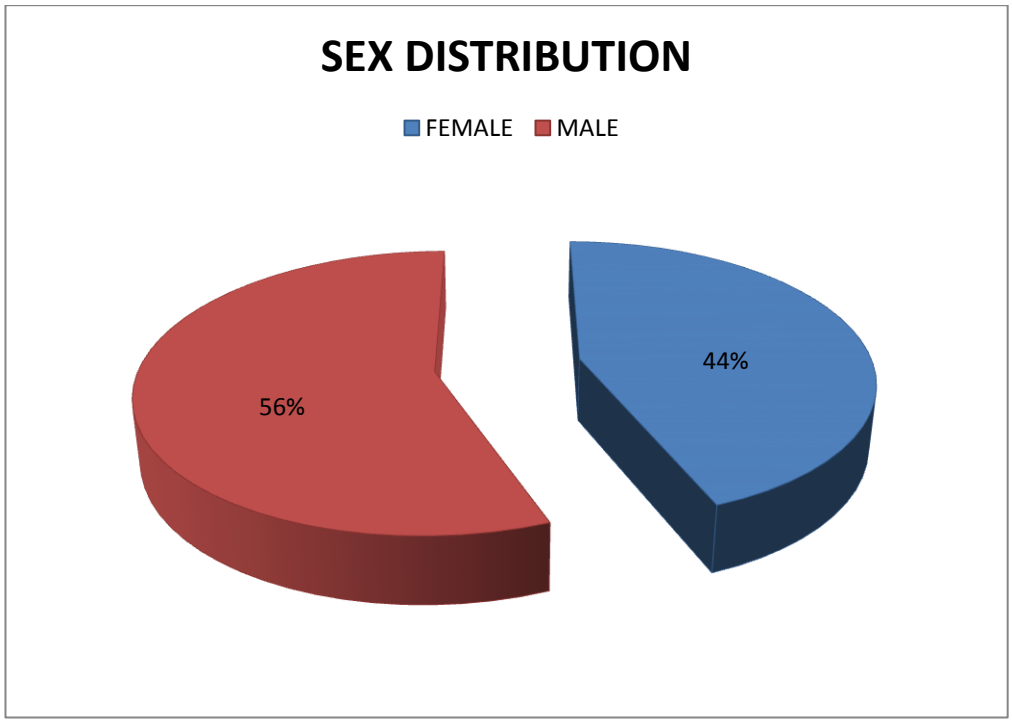
Minimum pulmonary artery (PA) area: 14.37 cm^2

DIAGNOSIS : PULMONARY ARTERIAL HYPERTENSION

STATISTICAL ANALYSIS

TABLE 1

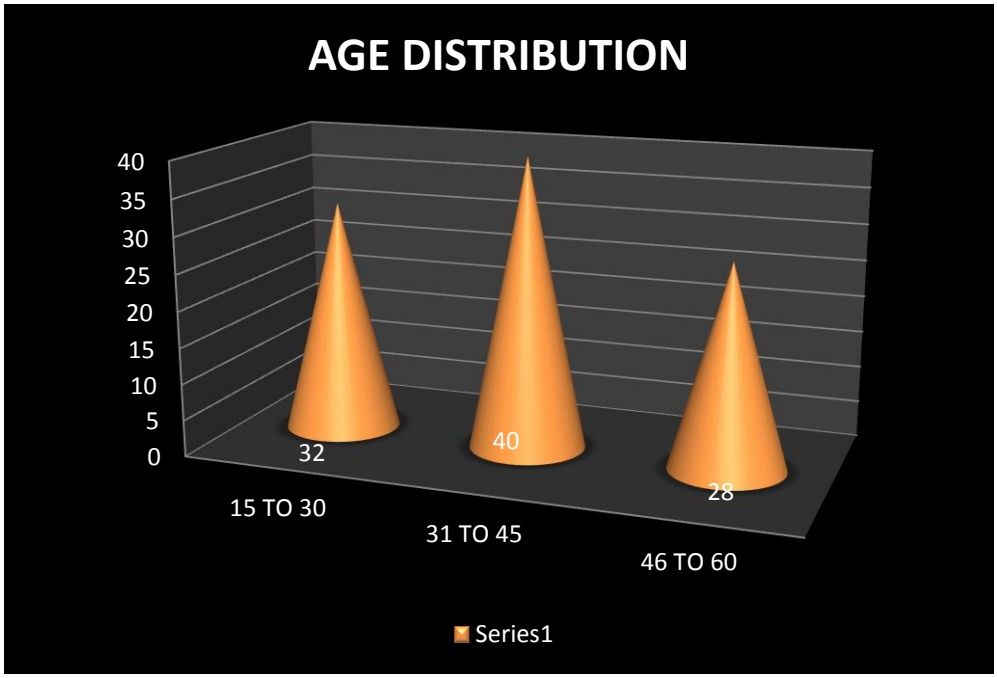
	FREQUENCY	PERCENT
FEMALE	22	44.0
MALE	28	56.0
Total	50	100.0



Among 50 total subjects, 22 subjects were female and 28 subjects were male. That is 44% are female and 58% are male.

TABLE 2

	Frequency	Percent
15 TO 30	16	32.0
31 TO 45	20	40.0
46 TO 60	14	28.0
Total	50	100.0

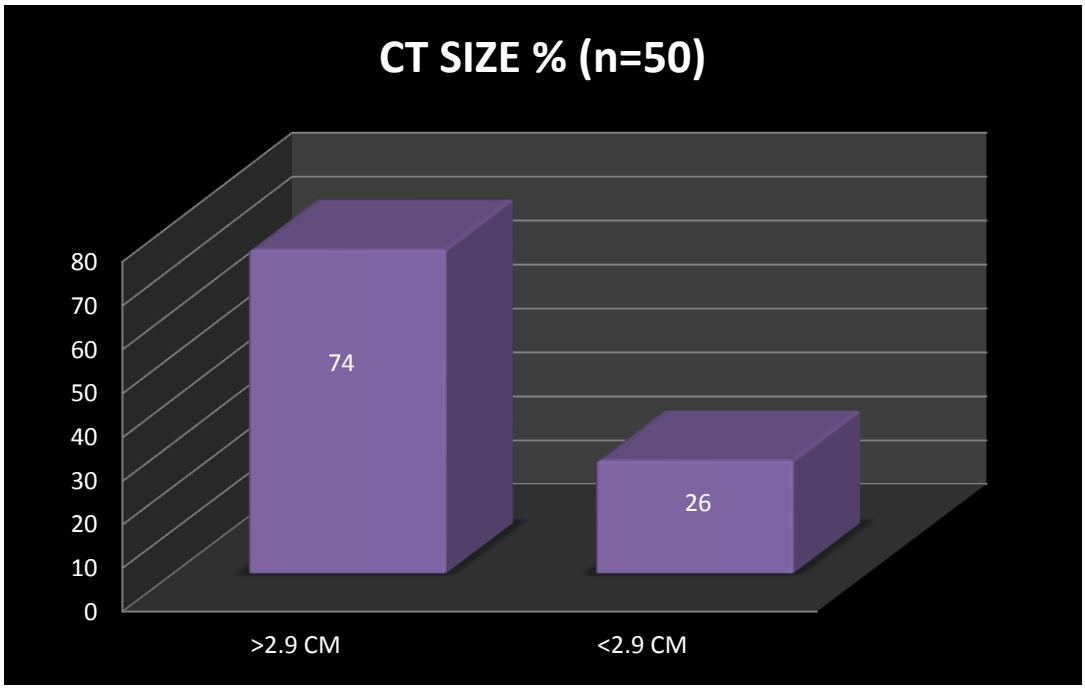


In this study, out of 50 subjects, 16 (32%) subjects were in the age group of 15 to 30, 20 (40%) subjects were in the age group of 31 to 45 remaining 14(28%) subjects fall in the age group of 46 to 60. Thus the frequency is slightly higher in the age group 31 to 45.

TABLE 3

PULMONARY ARTERY SIZE

PA size	Frequency	Percent
< 2.9 cm	13	26
> 2.9cm	37	74
Total	50	100

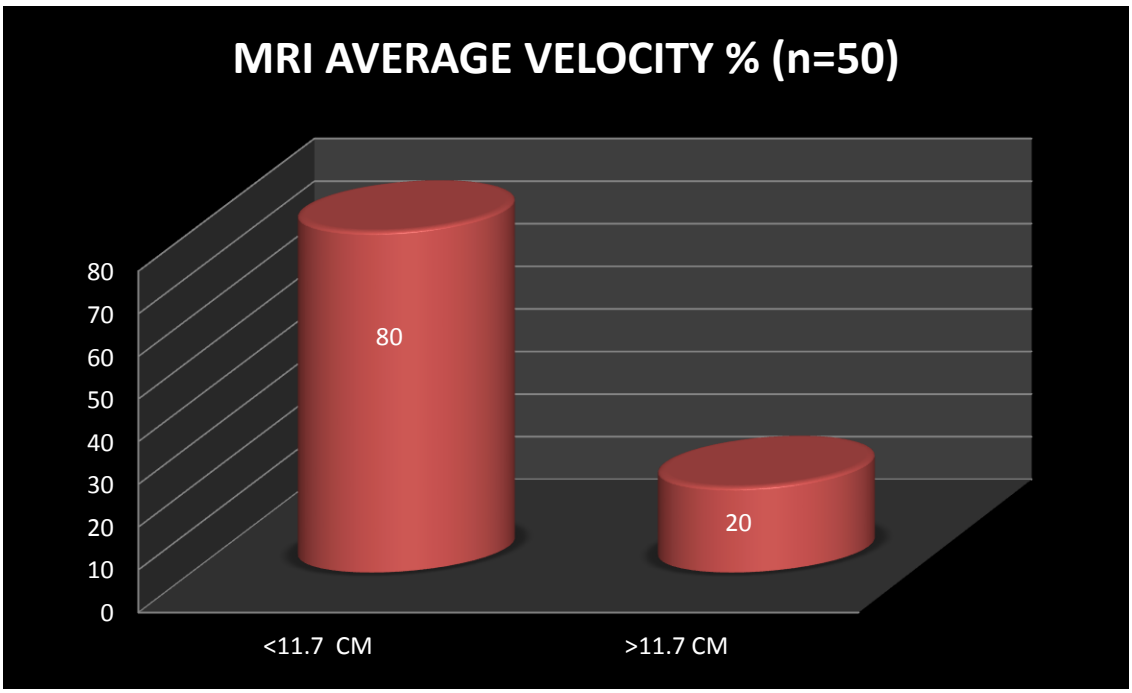


In this study, out of 50 subjects, 37 (74%) subjects were having PA size more than 2.9cm, 13 (26%) subjects were having PA size less than 2.9cm.

TABLE 4

PA AVERAGE VELOCITY

Average PA velocity (cm/sec)	Frequency	Percent
< 11.7	10	20
> 11.7	40	80
Total	50	100

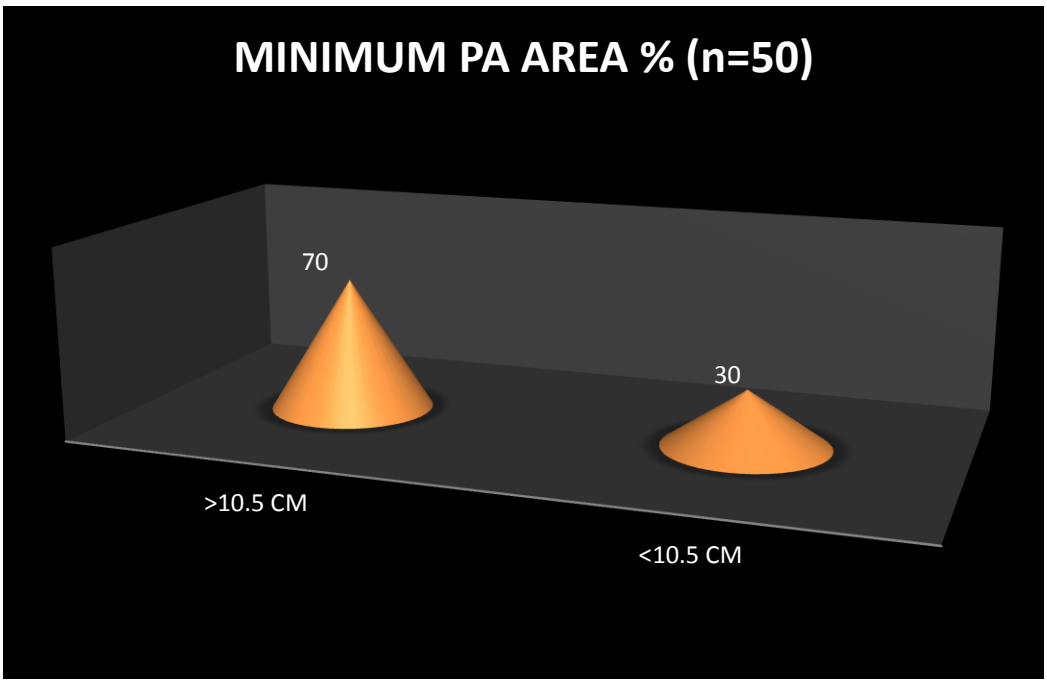


In this study, out of 50 subjects, 40 (80%) subjects were having a velocity of < 11.7 cm/sec, 10 (20%) subjects were having a velocity of >11.7 cm/sec.

TABLE 5

PA MINIMUM AREA

Minimum PA area (cm^2)	Frequency	Percent
<10.5	15	30
> 10.5	35	70
Total	50	100



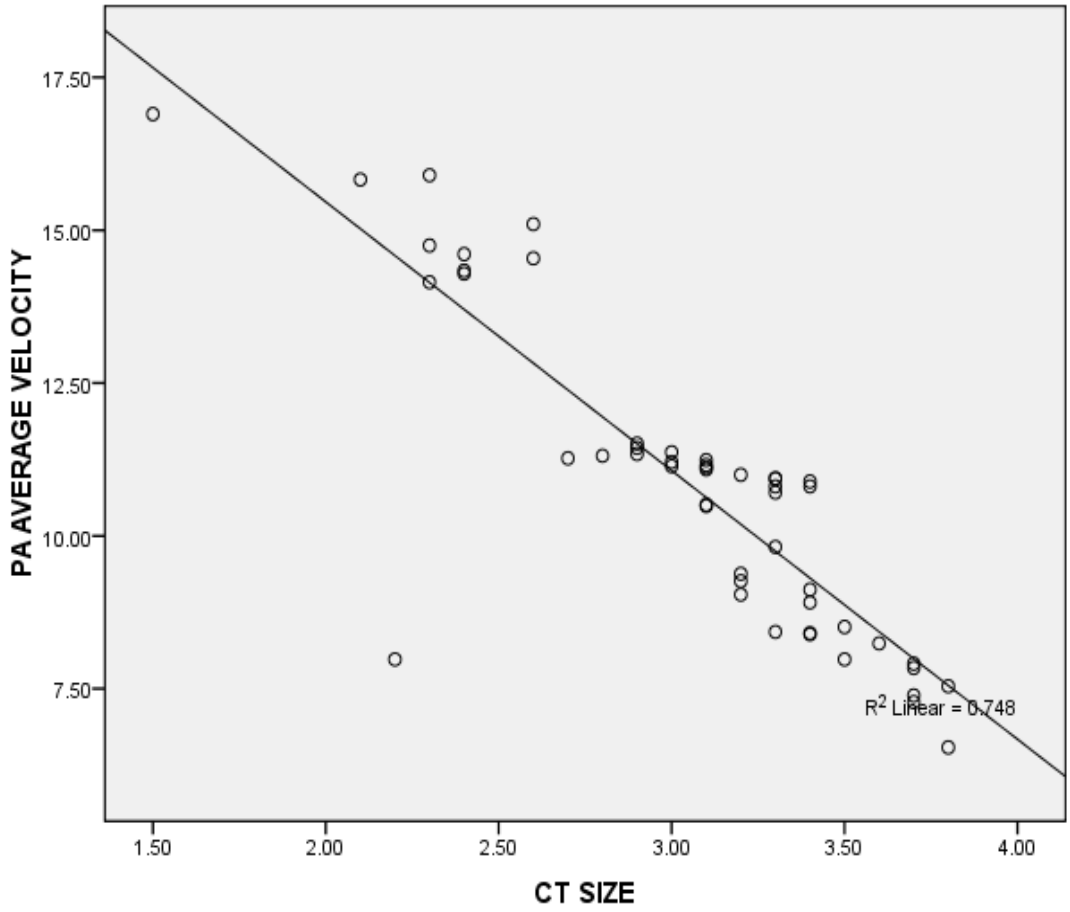
In this study, out of 50 subjects, 35 (70%) subjects were having a minimum area of >10.5 cm^2, 15 (30%) subjects were having a minimum area of <10.5 cm^2.

CORRELATION CHART

		CT SIZE	MRI AVEREAGE VELOCITY	MRI AVERAGE AREA	TRPG
CT SIZE	Pearson Correlation	1	-.865**	.806**	.716**
	Sig. (2-tailed)		.000	.000	.000
	N	50	50	50	50
MRI AVEREAGE VELOCITY	Pearson Correlation	-.865**	1	-.895**	-.810**
	Sig. (2-tailed)	.000		.000	.000
	N	50	50	50	50
MRI MINIMUM AREA	Pearson Correlation	.806**	-.895**	1	.768**
	Sig. (2-tailed)	.000	.000		.000
	N	50	50	50	50
TRPG	Pearson Correlation	.716**	-.810**	.768**	1
	Sig. (2-tailed)	.000	.000	.000	
	N	50	50	50	50

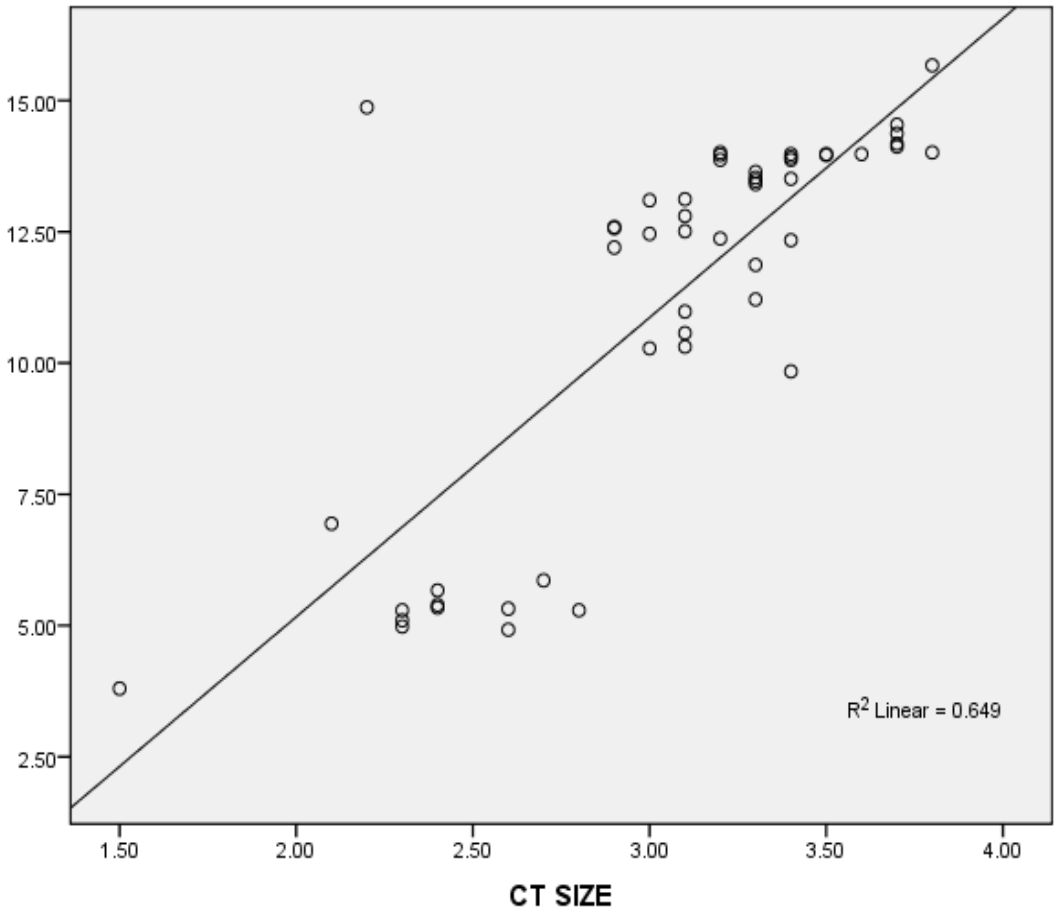
**. Correlation is significant at the 0.01 level (2-tailed).

GRAPH 1



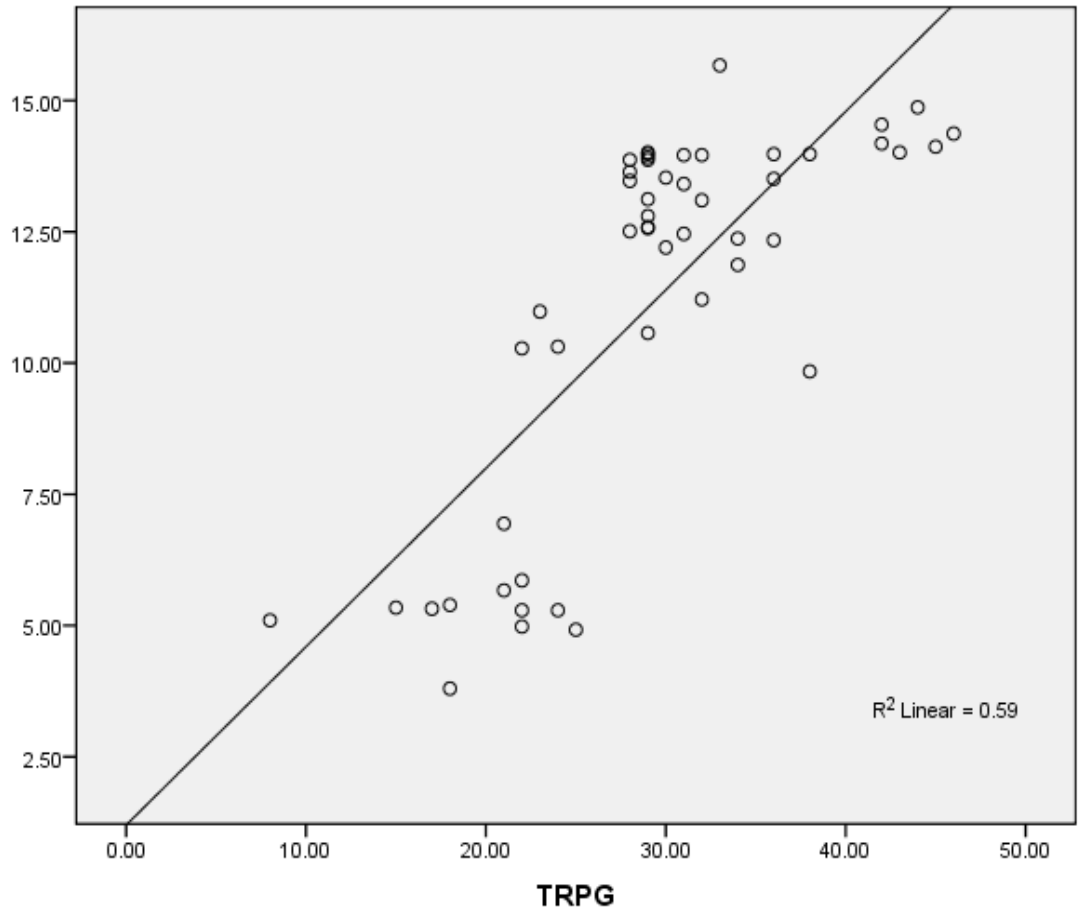
This graph shows correlation between PA Average velocity with pulmonary artery size. It has a negative correlation with Pearson correlation value of – 0.865 with P value 0.001 (P value < 0.05). The percentage of correlation of 86.5%.

GRAPH 2



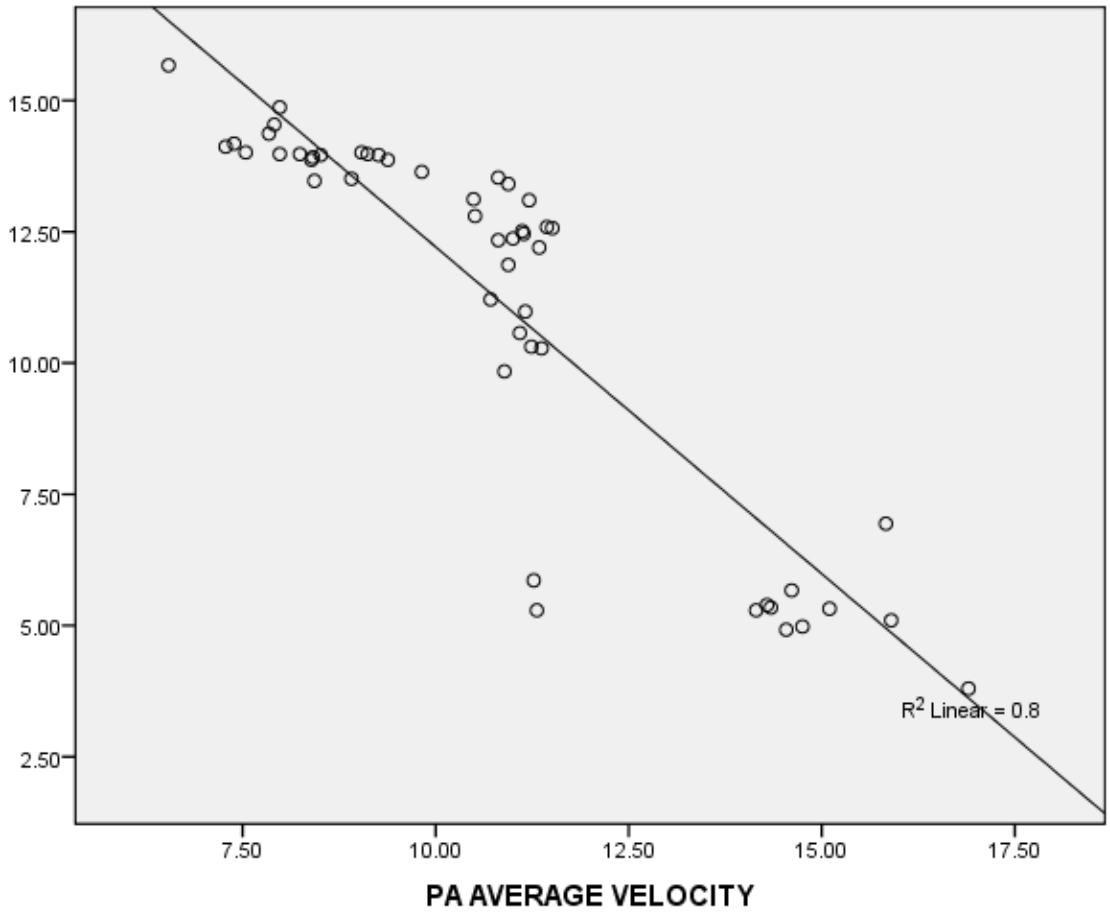
This graph shows correlation between PA area with pulmonary artery size. It has a positive correlation with Pearson correlation value of 0.806 with P value 0.001 (P value < 0.05). The percentage of correlation of 80.6%

GRAPH 3



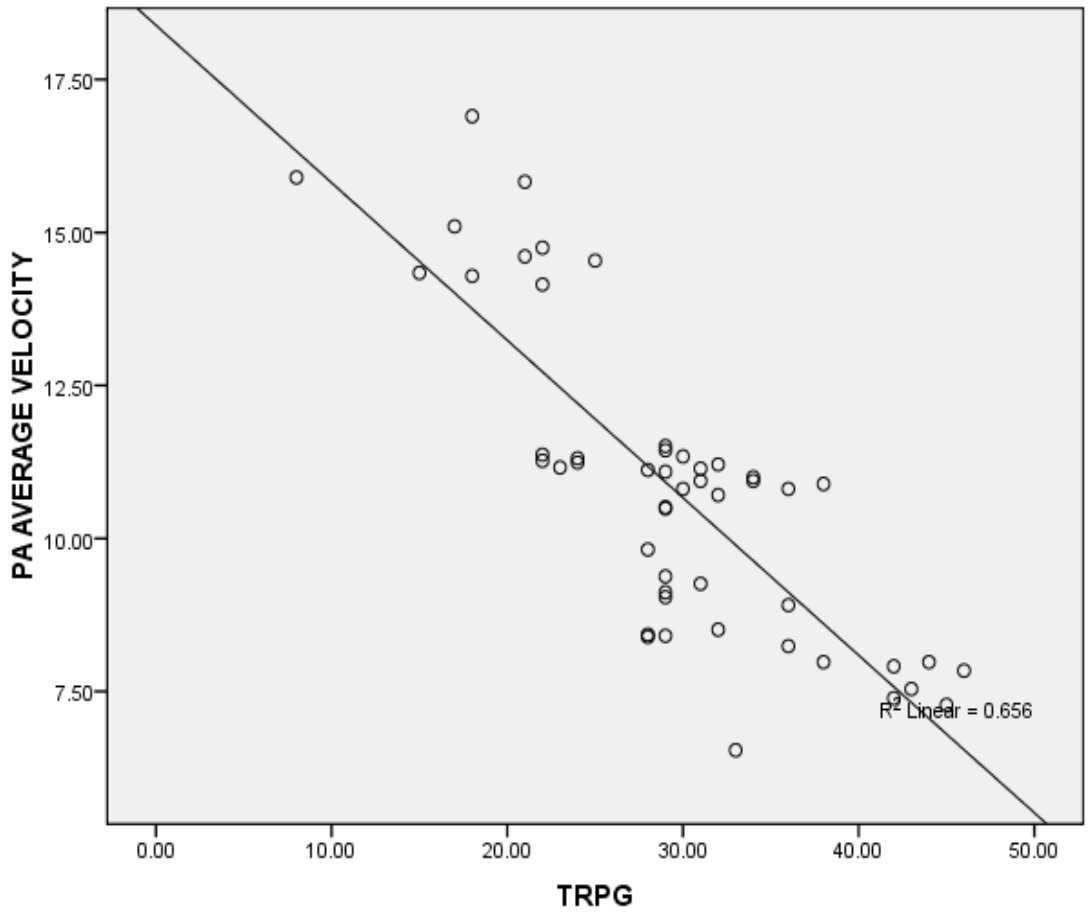
This graph shows correlation between TRPG with pulmonary artery area. It has a positive correlation with Pearson correlation value of 0.716 with P value 0.001 (P value < 0.05). The percentage of correlation of 71.6%

GRAPH 4



This graph shows correlation between PA area with PA average velocity. It has a negative correlation with Pearson correlation value of – 0.895 with P value 0.001 (P value < 0.05). The percentage of correlation of 89.5%.

GRAPH 5



This graph shows correlation between TRPG with PA average velocity. It has a negative correlation with Pearson correlation value of – 0.810 with P value 0.001 (P value < 0.05). The percentage of correlation of 81%.

DISCUSSION

Out of 50 subjects who underwent phase contrast flow velocity imaging 22 subjects were female and 28 were male. With regard to the CT criteria of pulmonary artery size, 37 out of 50 subjects had size more than 2.9cm and remaining 13 subjects had less than 2.9cm. 34 subjects had TRPG value more than 25mmHgand remaining 16 subjects had less than 25mmHg. In MRI phase contrast flow velocity imaging 40 subjects had average velocity less than 11.7cm/sec & minimum area more than 10.5cm^2 and remaining 10 subjects had average velocity more than 11.7cm/sec & average area less than 10.5cm^2. All 37 subjects who had pulmonary artery size more than 2.9cm including 34 subjects who had TRPG > 25mmHg who were diagnosed and treated as pulmonary hypertension depending on clinical symptoms and investigations(ECHO and CT) either in combination or in isolation were having average velocity less than 11.7cm/sec.

All 37 subjects who had pulmonary artery size more than 2.9cmincluding 34 subjects who had TRPG > 25mmHg had average velocity less than 11.7cm/sec and average pulmonary artery area more than 10.5cm^2. Thus pulmonary artery size criteria >2.9cm which is 89% sensitive and 82 specific for diagnosis of pulmonary hypertension is well

correlated with average velocity and minimum area of the pulmonary artery as measured using phase contrast MRI.

Among 50 total subjects, 22 subjects were female and 28 subjects were male. That is 44% are female and 58% are male.

In this study, out of 50 subjects, 16 (32%) subjects were in the age group of 15 to 30, 20 (40%) subjects were in the age group of 31 to 45 remaining 14(28%) subjects fall in the age group of 46 to 60.

In this study, out of 50 subjects, 37 (74%) subjects were having PA size more than 2.9cm, 13 (26%) subjects were having PA size less than 2.9cm. out of which 34 had corresponding ECHO correlation but 3 subjects had a normal echo finding.

In this study, out of 50 subjects, 40 (80%) subjects were having a velocity of < 11.7 cm/sec, 10 (20%) subjects were having a velocity of >11.7 cm/sec. Thus out of 40 subjects positive for pulmonary hypertension in MRI , 37 subjects had corresponding correlation with pulmonary artery size more than 2.9cm suggesting pulmonary hypertension.

In this study, out of 50 subjects, 35 (70%) subjects were having a minimum area of >10.5 cm^2, 15 (30%) subjects were having a minimum area of <10.5 cm^2.

Graph 1 shows correlation between PA Average velocity with pulmonary artery size. It has a negative correlation with pearson correlation value of – 0.865 with P value of 0.001 (P value < 0.05). The percentage of correlation of 86.5%. As PA size increases pulmonary artery average velocity decreases.

Graph 2 shows correlation between PA minimum area with pulmonary artery size. It has a positive correlation with Pearson correlation value of 0.806 with P value of 0.001 (P value < 0.05). The percentage of correlation of 80.6%. As PA size increases PA average area also increases.

Graph 3 shows correlation between TRPG with pulmonary artery size. It has a positive correlation with Pearson correlation value of 0.716 with P value of 0.001 (P value < 0.05). The percentage of correlation of 71.6%. As pulmonary artery size increases TRPG value also increases.

Graph 4 shows correlation between PA minimum area with PA average velocity. It has a negative correlation with Pearson correlation

value of – 0.895 with P value of 0.001 (P value < 0.05). The percentage of correlation of 89.5%. As the PA average area increases PA average velocity decreases.

Graph 5 shows correlation between TRPG with PA average velocity. It has a negative correlation with Pearson correlation value of – 0.810 with P value of 0.001 (P value < 0.05). The percentage of correlation of 81%. As the TRPG value increases PA velocity decreases.

Thus as the pulmonary hypertension develops pulmonary artery size, minimum PA area, TRPG value increases and PA average velocity decrease. And the percentage of correlation (86.5%) is higher between increase PA size and decrease in velocity and also between PA minimum area and PA average velocity (89.5%).

RESULTS

- Out of 50 subjects who underwent phase contrast flow velocity imaging 22 were male and 28 were female patients.

- With regard to the CT criteria of pulmonary artery size more than 2.9cm, 37 out of 50 subjects had size more than 2.9cm and remaining 13 subjects had less than 2.9cm. Out of which 34 subjects had TRPG value more than 25mmHg and remaining had less than 25mmHg

- In MRI phase contrast flow velocity imaging 40 subjects had average velocity less than 11.7cm/sec& minimum area more than 10.5cm^2 and remaining 10 subjects had average velocity more than 11.7cm/sec & minimum area less than 10.5cm^2

- All 37 subjects who had pulmonary artery size more than 2.9cm had average velocity less than 11.7cm/sec and minimum pulmonary artery area more than 10.5 cm^2, thus pulmonary artery size criteria for pulmonary hypertension is well correlated with PA average velocity and PA minimum area of the pulmonary artery as measured using phase contrast MRI.

- Degree of correlation (86.5%) is higher for PA size & PA average velocity and also between PA minimum area & PA average velocity (89.5%)

CONCLUSION

- MRI Phase contrast imaging is a novel non invasive imaging modality that can be used in the diagnosis of pulmonary hypertension by means of measuring flow parameters of pulmonary artery.

- Flow parameters like PA average velocity and PA minimum pulmonary area of well correlated with the pulmonary artery size .

- Thus MRI phase contrast flow velocity imaging can be safe and non invasive imaging modality of choice in pulmonary hypertension.

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MASTER CHART

S. No	Name	Age	CT Size cm	MR PA Velocity Cm/sec	MR PA Area Cm^2	TRPG mmHg
1	Muthu	52	2.4	14.29	5.39	18
2	Saranraj	41	2.3	14.15	5.29	22
3	Ravi M	45	3.3	7.53	14.37	28
4	Madhal	49	3.4	8.41	13.92	29
5	Mufedol	28	3.4	8.39	13.87	28
6	Malliga	32	2.6	15.1	5.32	17
7	Alamelu	45	2.3	15.9	5.1	8
8	Dhanalaskhmi	37	2.4	14.34	5.34	15
9	Madhavan	45	3.1	11.09	10.57	29
10	Nagaraj	51	2.9	11.34	12.2	30
11	Chinathambi	48	3	11.14	12.46	31
12	Natesan	48	3.1	11.12	12.51	28
13	Pattabi	45	3.2	11	12.37	34
14	Vendhan	27	2.4	14.61	5.67	21
15	Veeramani	38	2.3	14.75	4.98	22
16	Praveen	18	1.5	16.9	3.8	18
17	Lakshmi	29	3.3	10.94	11.87	34
18	Mufedol	44	3.4	10.81	12.34	36
19	Muthukumar	30	2.9	11.44	12.59	29
20	Ravi J	43	3	11.36	12.26	22
21	Saranraj	37	3.1	11.24	10.31	24
22	Chitra	19	3.1	11.16	10.98	23
23	Madhavan	54	3.3	10.71	11.21	32
24	Lakshmirani	34	3.4	10.89	9.84	38
25	Sangeetha	32	2.2	7.98	14.87	44
26	Munusamy	47	3.6	8.24	13.98	36
27	Karunakaran	24	3.7	7.91	14.54	42
28	Devan	40	3.7	7.84	14.37	46
29	Mariyammal	48	3.1	10.51	12.8	29
30	Rojapoo	53	3.3	10.94	13.41	31
31	Jhanaki	52	2.1	15.83	6.94	21
32	Vaiyammal	47	2.8	11.31	5.29	24
33	Fiaki	39	2.7	11.27	5.86	22
34	Ellammal	42	2.6	14.54	4.92	25
35	Vadivel	48	3.5	8.51	13.96	32
36	Elumalai	37	3.4	8.91	13.51	36
37	Mythili	29	3.5	7.98	13.98	38
38	Boopalan	34	3.8	7.54	14.01	43
39	Neelambari	25	3.7	7.39	14.18	42
40	Veeraraghavan	26	3.7	7.28	14.12	45
41	Sathyaprakash	24	3.4	9.12	13.98	29
42	Manikandan	51	3.3	8.43	13.47	28
43	Chellan	29	3.2	9.04	14.01	29
44	Ibrahim	28	3.8	6.54	15.67	33
45	Valli	25	3.1	10.49	13.12	29
46	Kumari	29	3.3	10.81	13.53	30
47	Senguttuvan	34	3.2	9.26	13.96	31
48	Sumathi	31	3.2	9.38	13.87	29
49	Renuka	27	2.9	11.51	12.57	29

50	Periyapaiyan	49	3	11.21	13.1	32

patients Information Sheet

Title : PHASE CONTRAST FLOW VELOCITY IMAGING OF MAIN PULMONARY ARTERY

Site : Barnard Institute of Radiology,
Rajiv Gandhi Government General Hospital,
Chennai – 600 003.

PURPOSE OF THE STUDY

Pulmonary hypertension is uncommon disorder characterized by dilated main pulmonary artery. It is usually present as breathlessness and it can be asymptomatic at early stages. These symptoms may last for long period. We want to test the pulmonary artery in this condition. We have obtained permission from the Institutional ethical committee.

The study is a non-invasive. No fasting is required for this procedure. You will be placed in MRI console and scan will be taken on you. It takes 15 mins for the completion of this procedure. No injection will be given to you and CT chest will be performed. If needed ECHO will be done. Single visit to Radiology department is enough.

Before the procedure we will examine your RFT values which are essential for your safety. You must not participate if you are pregnant,

breastfeeding a child, or if you are of childbearing potential and not practicing effective methods of contraception (for studies/procedures which may harm the fetus).

The results of the research may provide benefits to the society in terms of advancement of medical knowledge and/or therapeutic benefit to future patients.

You have the right to confidentiality regarding the privacy of your medical information (personal details, results of physical examinations, investigations, and your medical history). By signing this document, you will be allowing the research team investigators, other study personal, sponsors, Institutional Ethics committee and any person or agency required by law like the Drug controller General of India to view your data, if required.

The information from this study, if published in scientific journals or presented at scientific meetings, will not reveal your identity.

Your decision not to participate in this research study will not affect your medical care or your relationship with the investigator or the institution. You will be taken care of and you will not lose any benefits to which you are entitled.

The participation in this research is purely voluntary and you have the right to withdraw from this study at any time during the course of the study without giving any reasons. However, it is advisable that you talk to the research team prior to stopping the treatment /discontinuing of procedures.

Signature of Investigator **Signature of Participant**

Date **Date**

Patient Consent Form

Title of the Project

PHASE CONTRAST FLOW VELOCITYIMAGING OF MAIN PULMONARY ARTERY

Institution : **Barnard Institute of Radio Diagnosis,**
Madras Medical College,
Chennai-600 003.

Name	:	Date	:
Age	:	IP No	:
Sex	:	Project Patient No	:

The details of the study have been provided to me in writing and explained to me in my own language.

I confirm that I have understood the above study and had the opportunity to ask questions.

I understood that my participation in the study is voluntary and that I am free to withdraw at any time, without giving any reason, without the medical care that will normally be provided by the hospital being affected.

I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purpose(s).

I have been given an information sheet giving details of the study.

I fully consent to participate in the above study regarding mean corpuscular volume as a marker of Alcohol use disorder.

_____	_____	_____
Name of the Subject	Signature	Date

_____	_____	_____
Name of the Investigator	Signature	Date

ABBREVIATIONS

KEY WORDS:

PAH	:	Pulmonary arterial hypertension
CT	:	Computerised Tomography
MRI	:	Magnetic Resonance Imaging
PA	:	Pulmonary Aretry
MPAP	:	Main Pulmonary Artery Pressure
PCWP	:	Pulmonary Capillary Wedge Pressure
TPG/TRPG	:	Tricuspid Regurgitant Pressure Gradient
TAPSE	:	Tricuspid Annular Plane Systolic Excursion
PVH	:	Pulmonary Venous Hypertension
HRCT	:	High Resolution Computerised Tomography
MPA	:	Main Pulmonary Artery
ILD	:	Interstitial Lung Disease
RCH	:	Right Heart Catheterisation
ECG	:	Electrocardiogram
Venc	:	Velocity Encoding
SNR	:	Signal To Noise Ratio
RVOT	:	Right Ventricular Outflow Tract

PROFORMA

Name :

Age and sex :

Weight :

IP/ OP number :

Ward number :

Address :

HISTORY:

PAST HISTORY:

INVESTIGATION:

1.Serum creatinine

2.Blood urea

DATA INTERPRETATION AND ANALYSIS

MRI PA FLOW PARAMETERS

6. Average velocity

7. Average pulmonary artery area

8. Peak velocity

9. Maximum pulmonary artery area

10. Minimum pulmonary artery area

CT

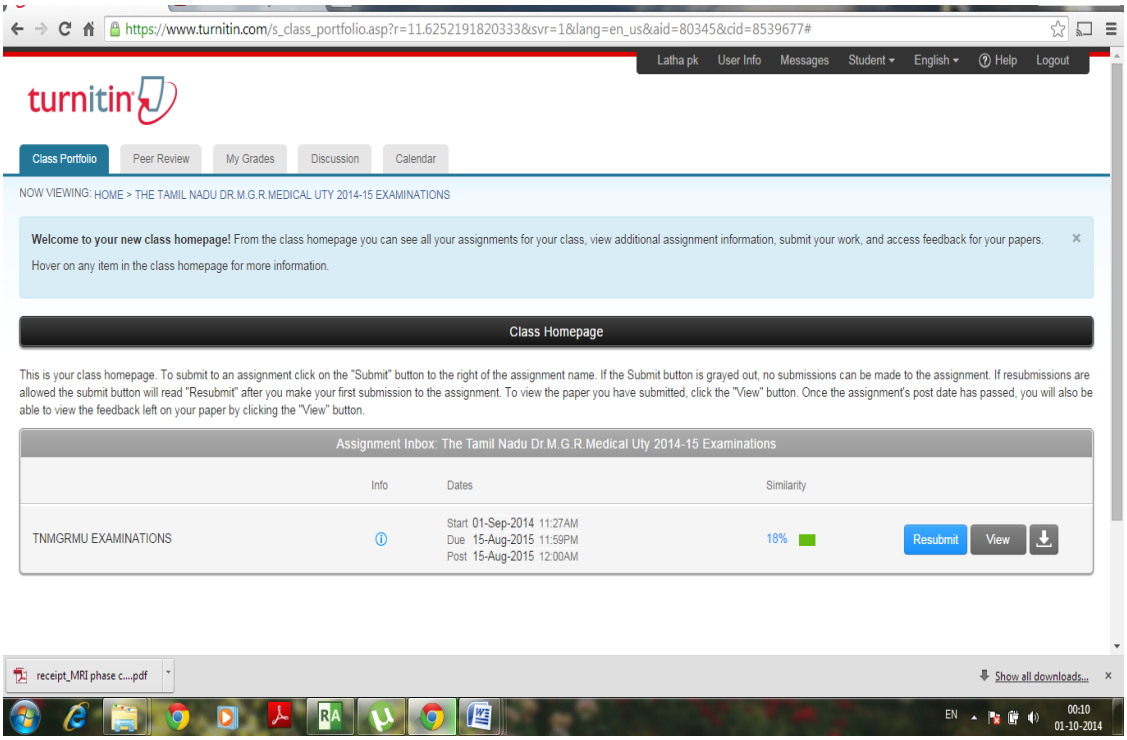
2. Size of the main pulmonary artery

ECHO

4. TRPG

5. TAPSE

6. Presence or absence of A wave in the M mode**.**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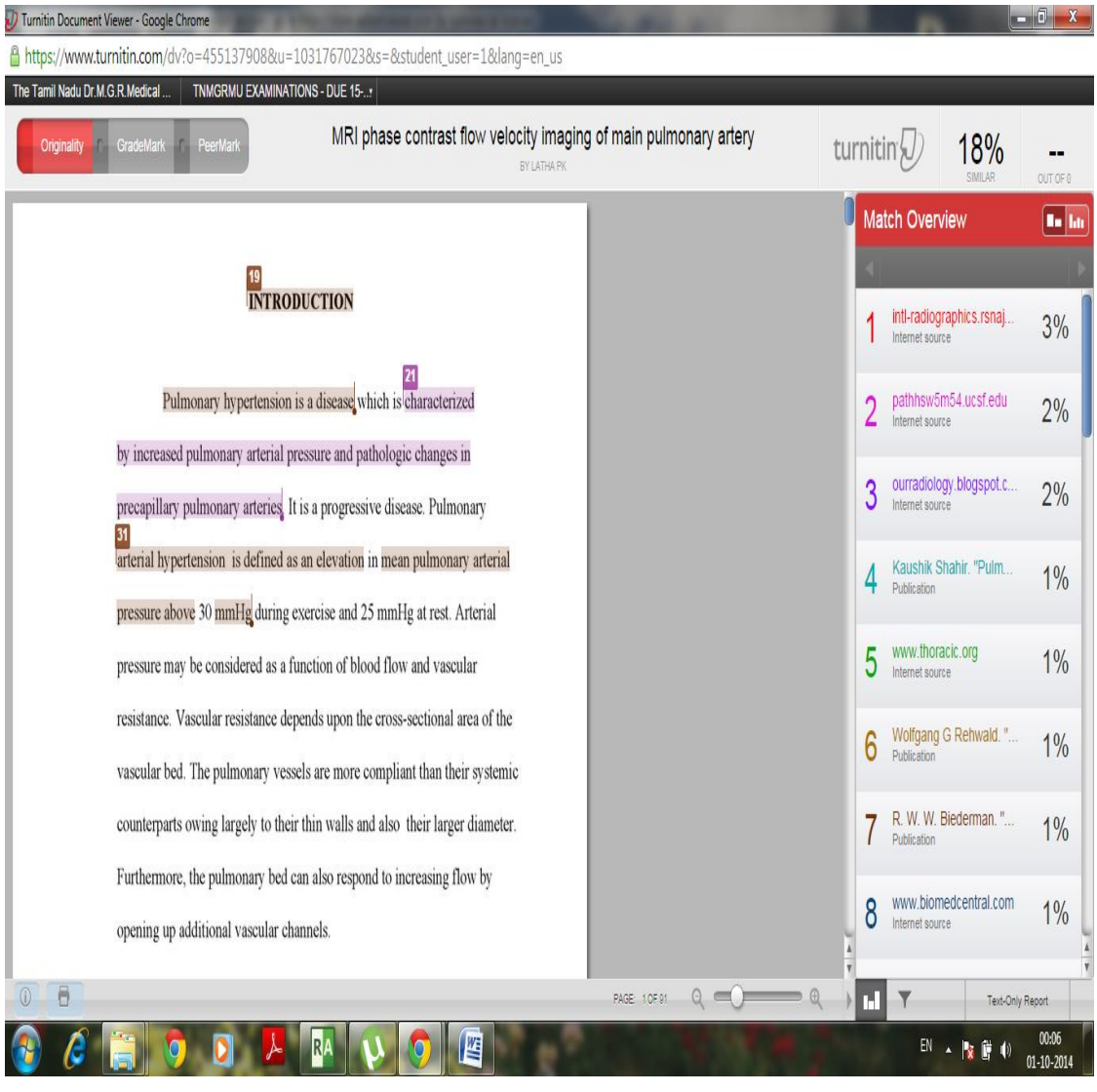
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INTRODUCTION
Pulmonary hypertension is a disease which is characterized by increased pulmonary arterial pressure and pathologic changes in precapillary pulmonary arteries. It is a progressive disease. Pulmonary arterial hypertension is defined as an elevation in mean pulmonary arterial pressure above 30 mmHg during exercise and 25 mmHg at rest. Arterial pressure may be considered as a function of blood flow and vascular resistance. Vascular resistance depends upon the cross-sectional area of the vascular bed. The pulmonary vessels are more compliant than their systemic counterparts owing largely to their thin walls and also their larger diameter. Furthermore, the pulmonary bed can also respond to increasing flow by opening up additional vascular channels.
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