Radiological Magnetic Resonance Imaging anatomy of the Heart; Special emphasis on Valvular lesions

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Abstract

Background: The heart has a central, ventro basal location in the thorax and is bordered bilaterally by the lungs, anteriorly by the sternum, and inferiorly by the diaphragm . It has an oblique position in the thoracic cavity, with the cardiac apex in the left hemi thorax. The long axis of the heart is rotated about 45º to both the sagittal and the coronal planes. In younger or slender individuals, the heart tends to be more vertical, whereas it tends to be more horizontal in obese patients. It is surrounded by the pericardial sac and has no physical connections with the surrounding structures except posteriorly and superiorly where the great arteries and the caval and pulmonary veins drain into the atria. The heart is a double, two-chambered pump "right-sided" and "left sided" chambers. The right chambers are more anteriorly positioned within the chest, the left chambers more posteriorly, and the ventricles are more inferiorly located than the atria.

Keywords: Magnetic Resonance Imaging, heart

Introduction

The heart has a central, ventro basal location in the thorax and is bordered bilaterally by the lungs, anteriorly by the sternum, and inferiorly by the diaphragm . It has an oblique position in the thoracic cavity, with the cardiac apex in the left hemi thorax. The long axis of the heart is rotated about 45º to both the sagittal and the coronal planes. In younger or slender individuals, the heart tends to be more vertical, whereas it tends to be more horizontal in obese patients. It is surrounded by the pericardial sac and has no physical connections with the surrounding structures except posteriorly and superiorly where the great arteries and the caval and pulmonary veins drain into the atria ***(1).***

The heart is a double, two-chambered pump "right-sided" and "left sided" chambers. The right chambers are more anteriorly positioned within the chest, the left chambers more posteriorly, and the ventricles are more inferiorly located than the atria ***(2)***

1--*Right atrium*:

Derived from the right atrial appendage and sinus venosus **,** These two components of the right atrium are separated by the crista terminalis (terminal crest). ***(3)***

The sinus venosus has a smooth wall and receives the inferior vena cava and superior vena cava–coronary sinus. The right atrial appendage is triangular-shaped with a wide opening and contains rough trabeculations or pectinate muscles. The crista terminalis is visualized on both bright and dark blood sequences and appears isointense to the myocardium. A prominent crista terminalis can mimic a mass. However, the location and signal characteristics on MRI can help confirm that this structure is indeed normal cardiac tissue ***(4)***. The valves of the coronary sinus (thebesian valve) and inferior vena cava (eustachian valve) are also readily apparent on MRI.

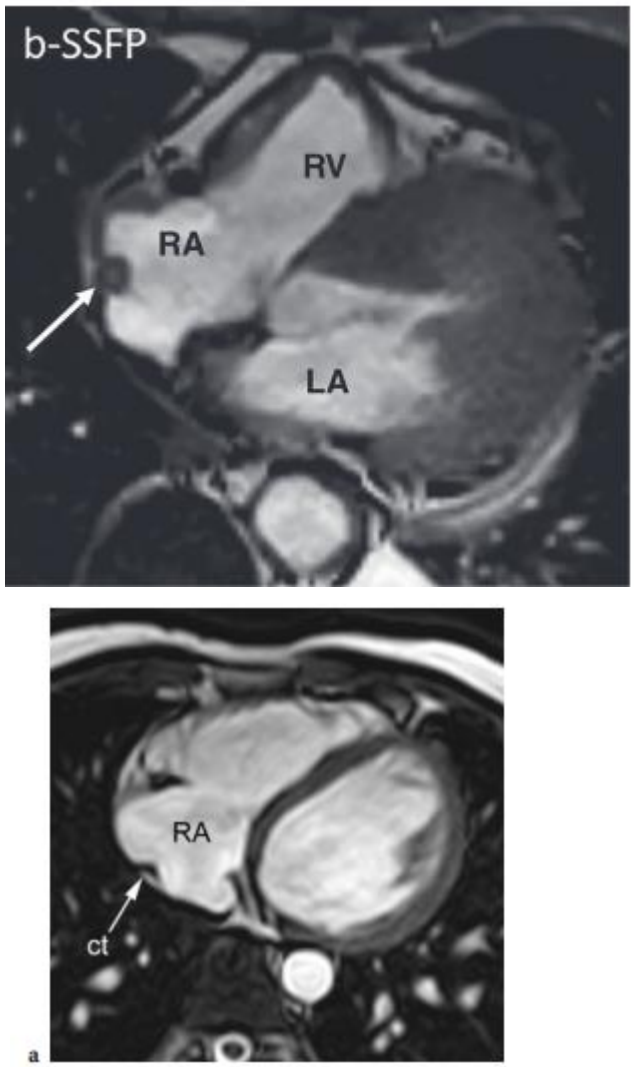
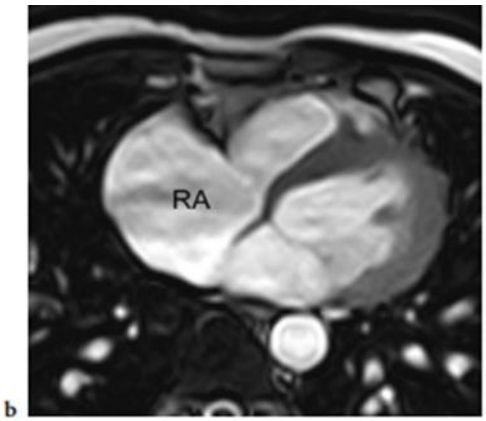


Figure (1): arrow represent Crista terminalis at MRI***. (5)***



Figure(2). Right atrium at end diastole and b end systole. The images are obtained in the transverse axis, using a balanced steady-state free precession (SSFP) technique. The terminal crest (crista terminalis, ct) divides the venous component (postero medially) from the vestibule. Note the important changes in right atrial (RA) volume and shape during the cardiac cycle. ***(5)***

2- Right ventricle: receives blood from the right atrium and consists of inlet, outlet, and apical portions . The inlet contains the tricuspid valves and associated chordal attachments. Attachment of the chords to the septum is characteristic of the tricuspid valve. The apical portion of the right ventricle is trabeculated, whereas the outlet is smooth and leads to the pulmonary artery. The right ventricle also contains the moderator band, which courses from the apical portion at the anterior papillary muscle insertion to the interventricular septum. ***(6)***

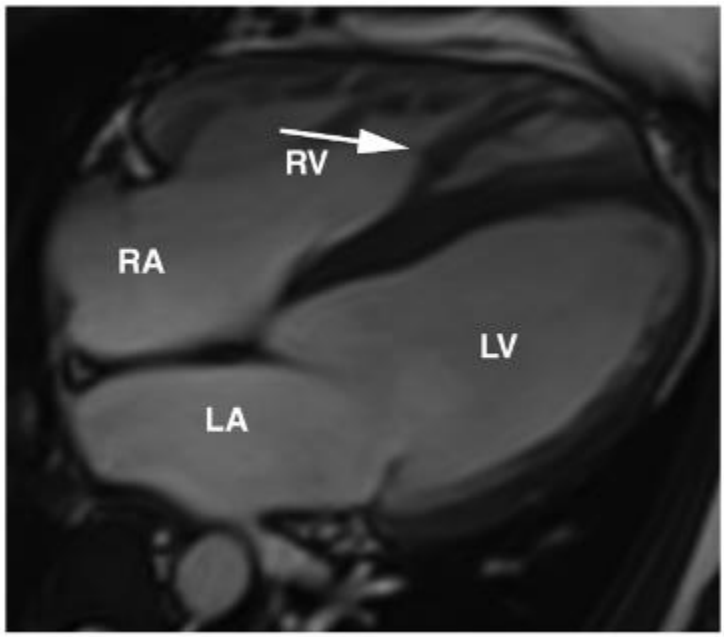


Figure (3): Four-chamber MR image shows the moderator band (arrow) in the mid portion of the RV, extending from the septum to the free wall.***(7).***

3-Left atrium : is the most posterior and superior chamber and receives pulmonary venous return . The left atrium is also attached to the left atrial appendage, which is more elongated and narrower than the right atrial appendage. The left atrium is smaller and contains fewer trabeculations than the right atrium. The interatrial septum separates the right and left atria and features the fossa ovalis, which appears as an area of focal thinning. ***(8)***

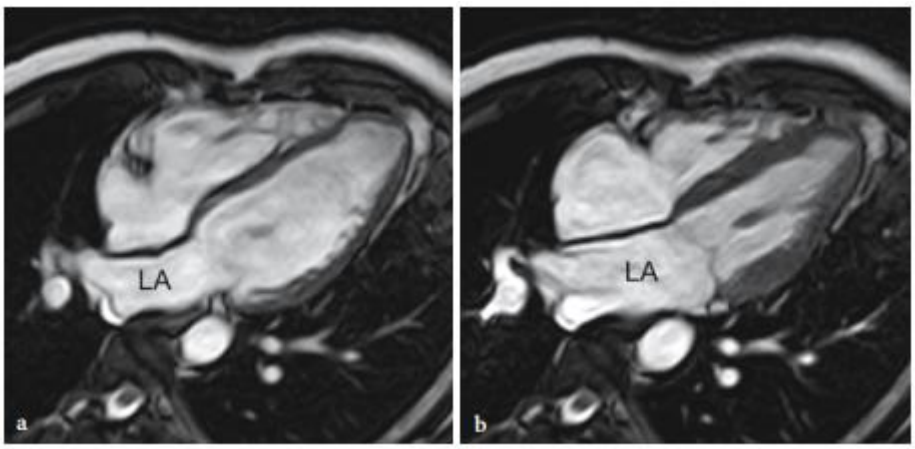


Figure (4) Left atrium at end diastole (a) and end systole (b). Horizontal long-axis images using a balanced SSFP technique. The entrance of the pulmonary vein from the right lower lobe in the left atrium (LA) can be clearly seen ***(9)***.

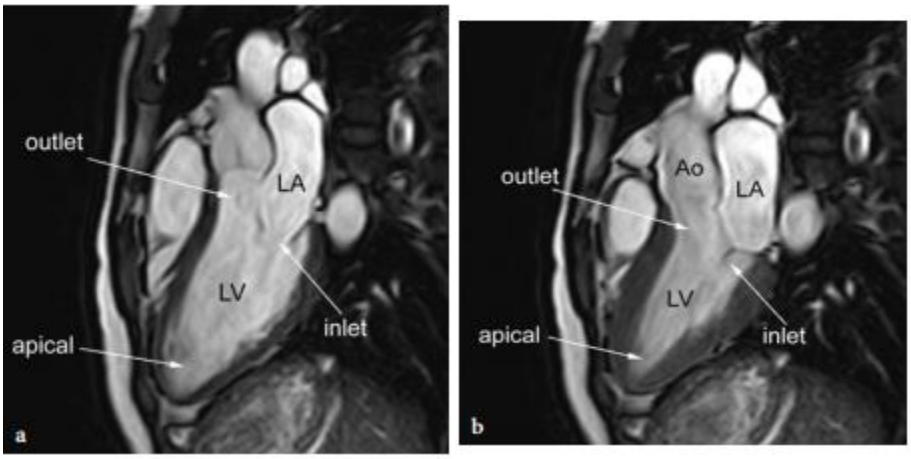


Figure (5) Components of the left ventricle. Left ventricular inflow–outflow tract flow view at end diastole (a) and end systole (b), using the balanced-SSFP technique. Ao, Aorta; LA, left atrium ***(6)***.

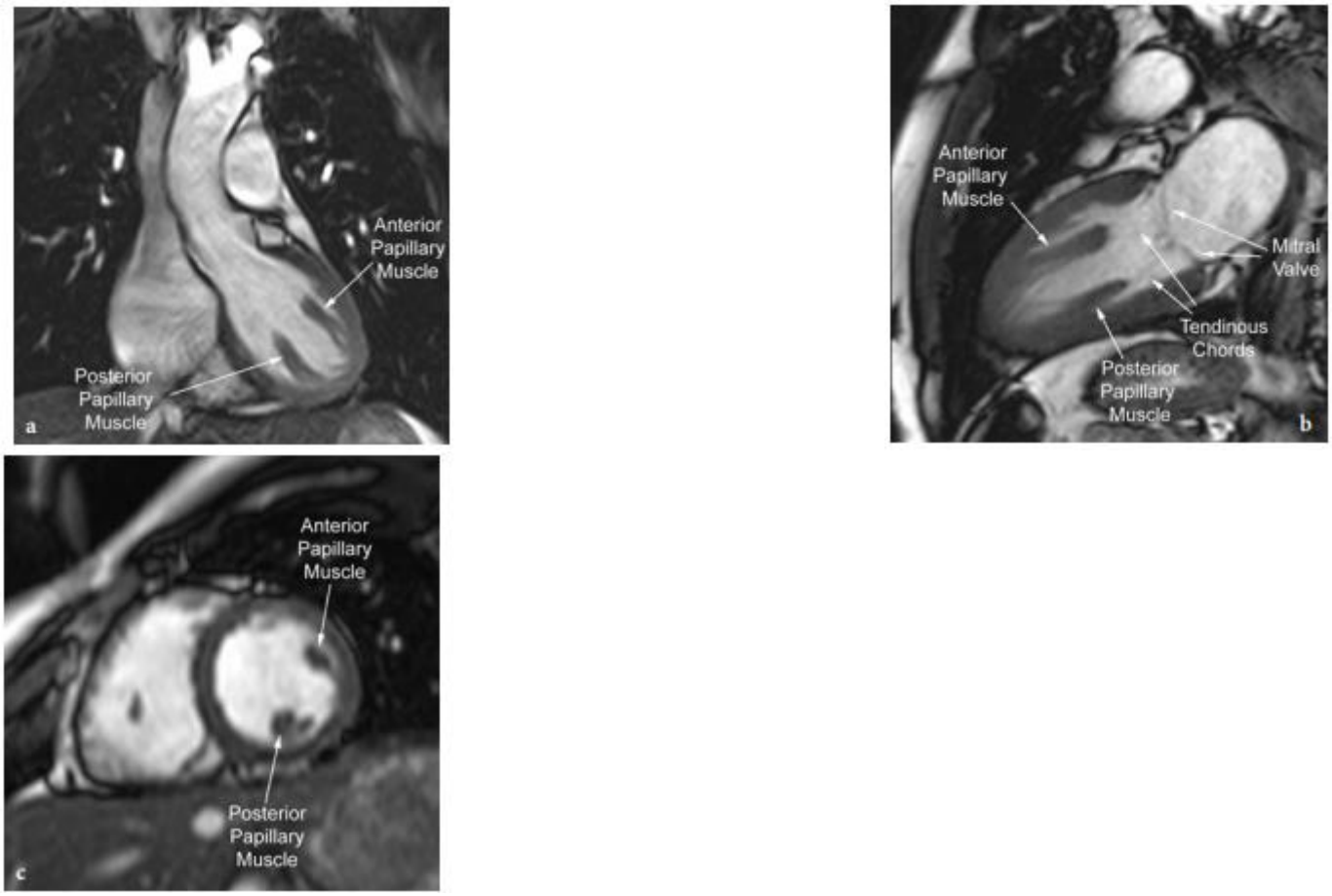


Figure (6). Left ventricular papillary muscles. Left ventricular outflow tract (a), vertical long-axis (b) and mid ventricular short-axis (c) view, using the balanced-SSFP technique. The papillary muscles are clearly depicted as intra cavity structures attached to the posterior (medial) and anterior (lateral) LV wall. Their fibrous extensions, i.e., tendinous chords, towards the mitral valve can be seen on high-quality MRI. Note that the LV septal surface on the short-axis view is free of muscular attachments. ***(3)***

5- **Coronary arteries** : arise from the aortic sinuses, just distal to the aortic valve. The right coronary artery (RCA) courses along the atrioventricular groove and is dominant in approximately 85% of individuals, giving rise to the posterior descending artery (PDA), which courses along the posterior interventricular sulcus toward the apex.

-The RCA also supplies the conal branch, right marginal branches, posterolateral ventricular branch, and frequently the sinus node artery. The left main coronary artery is a short trunk that supplies the left anterior

descending (LAD) and left circumflex arteries. In up to 30% of individuals an additional ramus intermedius artery arises from the bifurcation of the left main coronary artery ***(10)***. The LAD courses along the anterior interventricular sulcus and give rise to the diagonal and septal branches. The left circumflex artery supplies the obtuse marginal branches and sometimes supplies the sinus node artery and PDA in a left-dominant or codominant system.

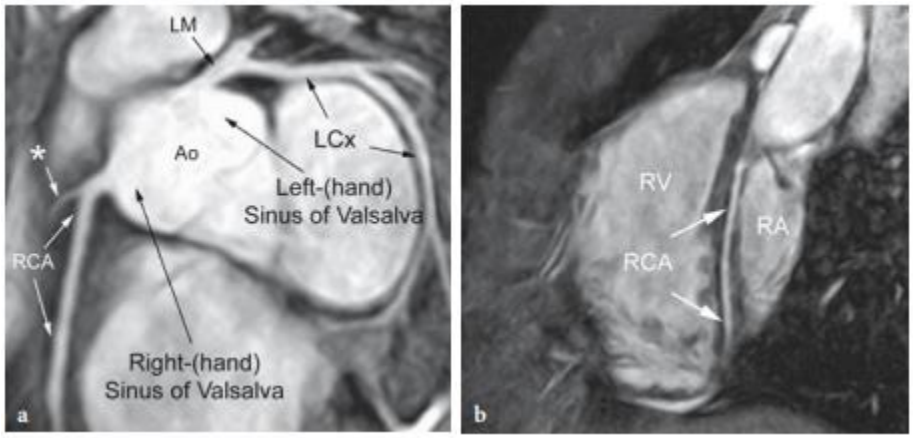


Figure (7) Origin and proximal course of coronary arteries. Short-axis view through the aortic root (a), and oblique view through anterior (or right) atrioventricular groove, using a balanced-SSFP technique with submillimeter spatial resolution. The right coronary artery (RCA) and left coronary artery originate from the right and left sinus of Valsalva, respectively. The major arteries as well as several branches can be readily depicted. This subject has a LCx dominant system. Ao, Aorta; LM, left main stem coronary artery; RA, right atrium; RV, right ventricle; star, conal branch.***(11).***

MRI pulse sequences

- **Dark Blood Imaging**: refers to the low-signal-intensity appearance of fast-flowing blood and is mainly used to delineate anatomic structures. spin-echo (SE) sequences have been used for dark blood imaging. SE has been supplanted by the newer FSE and turbo spin-echo (TSE) techniques in cardiac imaging. Although these techniques have lower signal-to-noise ratio than SE, they enable rapid imaging, which minimizes the effects of respiratory and cardiac motion.

- Basic FSE and TSE sequences : consist of radiofrequency pulses with flip angles (α) of 90° and 180° followed by acquisition of 1 or 2 signals. and can be T1- or T2-weighted acquired over a series of single or double R-R intervals.

- A potential source of artifact is slow-flowing blood, which can appear bright and can blend in with anatomic structures. As well as, the presence of gadolinium can interfere with nulling of blood signal and should be administered after dark blood imaging. However, real-time non gated black blood sequences with gadolinium contrast administration have proven to be effective for evaluating myocardial ischemia ***(12)***

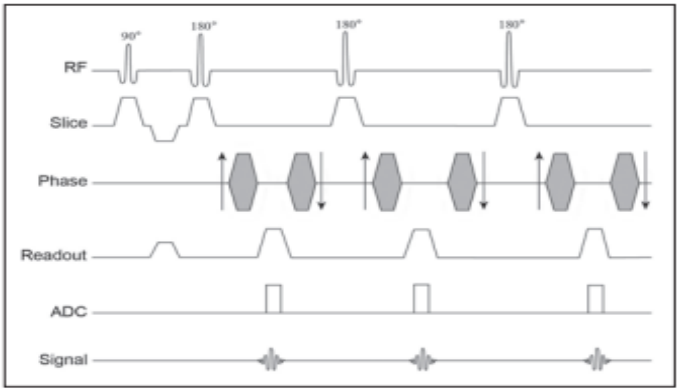


fig. (8)—Diagram shows fast spin-echo pulse sequence. RF = radiofrequency pulse, ADC = analog-to-digital converter

Bright Blood Imaging: describes the high signal intensity of fast-flowing blood and is typically used to evaluate cardiac function. The main pulse sequences used for bright blood imaging include GRE and the more recently introduced, but related, technique termed "steady-state free precession" or "SSFP." GRE sequences (i.e., spoiled gradient recall [SPGR], turbo FLASH, turbo field echo, and fast-field echo [FFE]) are produced by emitting an excitation radiofrequency pulse that is usually less than 90°, followed by gradient reversals in at least two directions, which create an echo signal that can be detected.

-SSFP sequences (i.e., fast imaging employing steady-state acquisition [FIESTA], fast imaging with steady-state precession [FISP], balanced FFE) are similar but incorporate a short TR with gradient refocusing that is less vulnerable to T2* effects compared with standard GRE. SSFP sequences can be executed rapidly while providing greater contrast-to-noise and signal-to-noise ratios than GRE sequences. ***(12)***

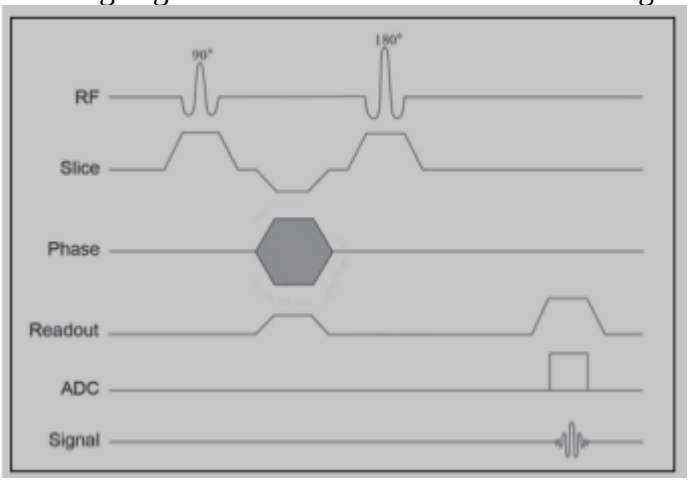


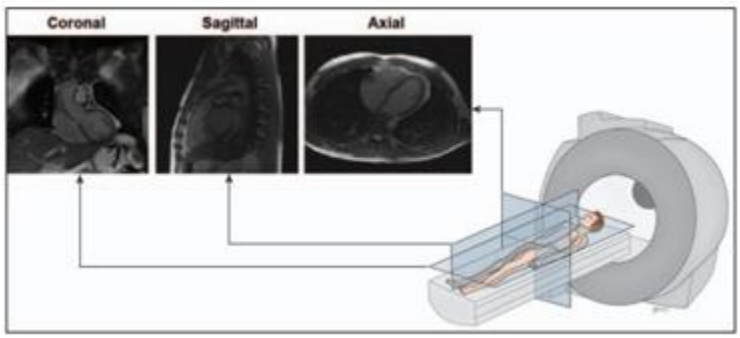
figure (9) diagram shows steady state free precession sequence. RF =radiofrequency pulse, ADC=analogue-to-digital converter. ***(12)***

Imaging Planes:

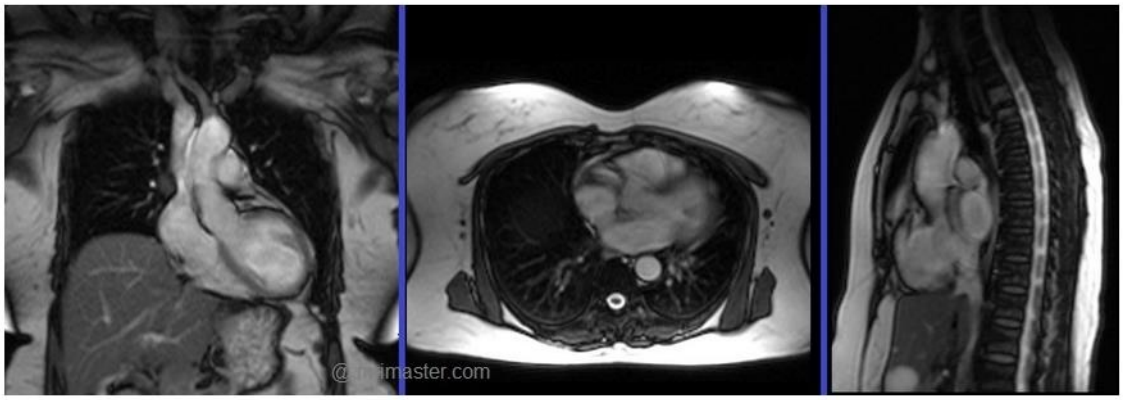
The two main coordinate systems used for cardiac MRI include the body (scanner) planes and the cardiac planes.

-Body planes are oriented orthogonal to the long axis of the body and consist of axial, sagittal, and coronal planes **.** These planes are used to derive the scout images and provide a qualitative overview of cardiac morphology.

- The ***sagittal plane*** can show the great vessels arising in continuity from the ventricles.
- The ***coronal plane*** can be used to assess the left ventricular outflow tract, the left atrium, and the pulmonary veins. However, the obliquity (≈ 45°) of these planes to the walls of the heart precludes accurate anatomic and functional characterization. Rather, such information should be obtained from the specialized cardiac planes.
- The ***axial plane*** can depict the four chambers of the heart and the pericardium simultaneously.



Figure(10) show orientation of major body planes with respect to patient and their corresponding appearance on bright blood imaging sequences.



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-The standard **cardiac planes** are established using the scout images and include short axis, horizontal long axis (four-chamber view), and vertical long axis (two-chamber view) ***(13)***.

-These planes are prescribed along a line extending from the cardiac apex to the center of the mitral valve (long axis of the heart) using the axial body plane images.

- ***The short-axis plane***: extends perpendicular to this true long axis of the heart at the level of the mid left ventricle.

- ***The horizontal long axis*** is generated by selecting the horizontal plane that is perpendicular to the short axis.

-***The vertical long axis*** is prescribed along a vertical plane orthogonal to the short-axis plane.

- Ventricular volumetric measurements are routinely derived from the short-axis views ***(14)***.

-Other commonly used planes include ***the oblique sagittal plane*** (parallel to the axis of the aorta), "***three-points" plane*** (connects three points along a selected coronary artery), ***left ventricular outflow view*** (three-chamber view), and ***right ventricular outflow track view (13).***

General techniques Left ventricular (LV) structure and function:

1. Scout imaging: trans axial, coronal and sagittal, these are in general single heartbeat acquisitions acquired in 1 breath hold.

2. Transaxial (8–10 mm) set of bSSFP or fast spin echo (FSE) images through the chest. These are single-shot, single heartbeat images with a set acquired in 1–2 breath holds.

3. Scout to line up short axis images – cine acquisitions are preferable to single shot as long axis motion and inflow should be visualized ***(9).***

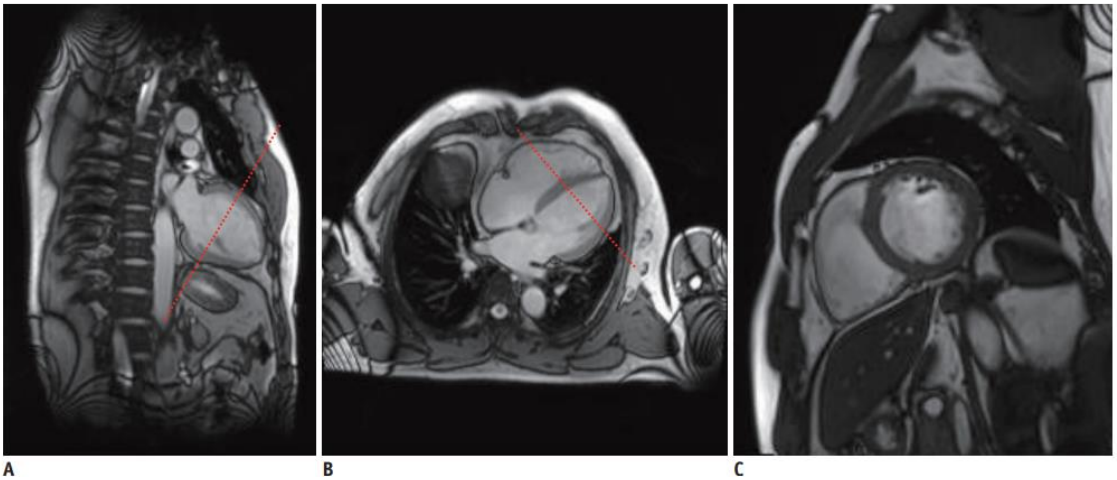

Fig (11). How to obtain short axis image. A. Vertical long axis image. B. Horizontal long axis. C. Short axis image. ***(15).***

Cine images

- Used for the assessment of cardiac function. During cine acquisition several individual images are acquired in different phases of cardiac cycle. These are then played as movie to provide the user with a video of the cardiac activity.
- True SSFP sequences are highly recommended for cine imaging due to high temporal and spatial resolution and improved blood tissue contrast.
- bSSFP is the method of choice for cine imaging as it provides high SNR and excellent contrast between myocardium and blood pool.
    - At SSFP cine images may be compromised by artifact and spoiled gradient-echo sequences can be considered as an alternative
    - Strategies to reduce or move banding artifact include shimming, reducing the TR, and adjusting the RF frequency (frequency 'scout' sequence can be helpful for this)
    - Cine images are acquired during a breath-hold. Breath-hold on expiration provides more consistent positioning but inspiratory breath-hold may be more comfortable and easier to sustain for some patients.

- ***bSSFP short axis cine images***
    - Acquired from the base of the LV through the apex.
    - The first short-axis cine plane should be planned using the 4- and 2-chamber long-axis views, and it should be perpendicular to the long-axis of the body of the LV. This plane might not always be parallel to the mitral valve plane.

- Slice thickness 6–8 mm, with or without 2–4 mm interslice gaps (to make a total of 10 mm).
- Temporal resolution ≤45 ms between phases to optimize evaluation of wall motion e. Parallel imaging or compressed sensing used as available to shorten scan time.

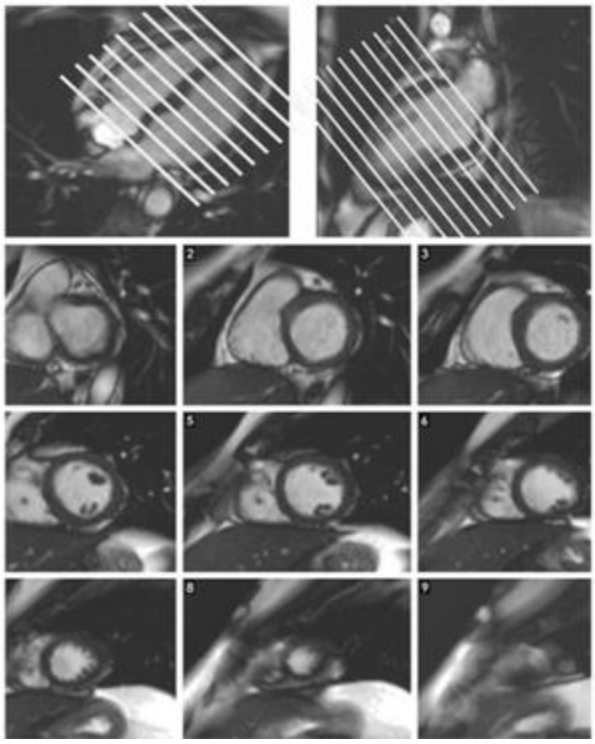

Fig. (12) Top – Planning of the short axis image plane parallel to the mitral valve in the 4 chamber long axis plane (left) and 2 chamber long-axis plane (right). Bottom panel – 9 short axis cine slices shown from base (top left) to apex (bottom right) ***(9)***.

- ***bSSFP long axis cine images:***
    - The 4-chamber long-axis view is prescribed from the 2-chamber long-axis view through the apex and center of the mitral and tricuspid valves. This can be modified and/or cross checked on basal short-axis views, to have the plane cross the acute margin of the right ventricular (RV) free wall and perpendicular to the interventricular septum.

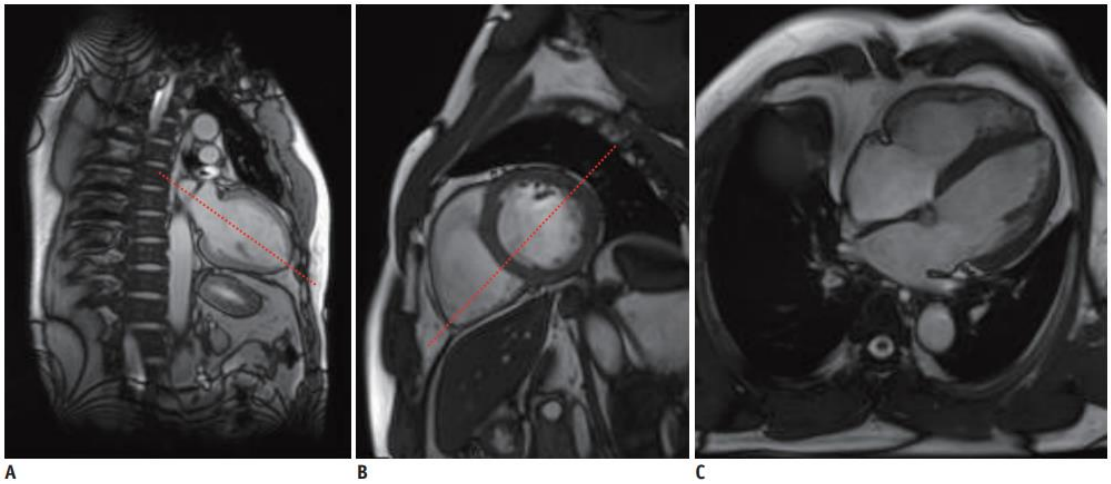

Fig. (13) How to obtain 4-chamber view. A. Vertical long axis image. B. Short axis image. C. 4-chamber view. ***(15).***

-***The 2-chamber LV view*** is prescribed from the vertical long-axis scout already acquired with modification to pass through the anterior and inferior myocardial walls.

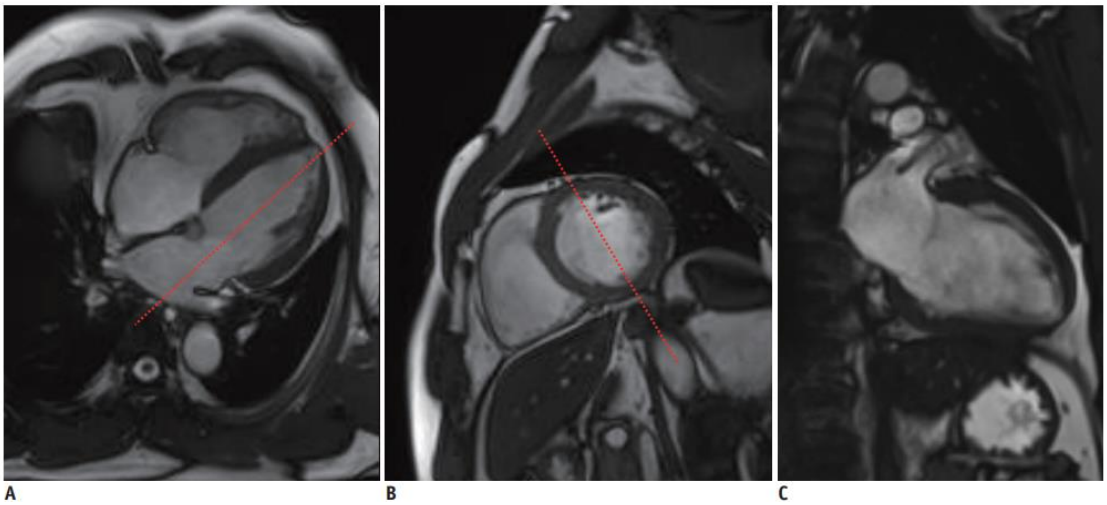

Fig. (14) How to obtain 2-chamber view. A. 4-chamber view. B. Short axis image. C. 2-chamber view. ***(15).***

-The 3-chamber LV view is prescribed passing through the apex, the center of the mitral valve and aligned with the center of LV outflow tract (LVOT) to aortic valve, as seen on a basal short axis cine.

Right ventricular (RV) structure and function

1. ***RV short-axis views*** can be obtained in a similar fashion to LV structure and function. If the short axis is used for quantification, it is important to place the basal short axis slice immediately on the myocardial side of the RV.

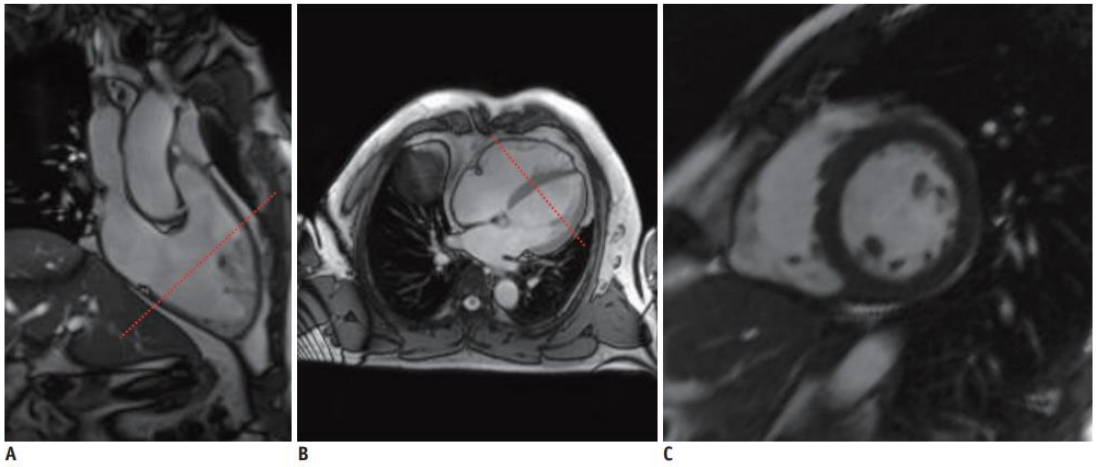

Fig. (15) How to obtain right ventricle (RV) short axis view. A. RV 2-chamber view. B. 4-chamber view. C. RV short axis view. ***(15).***

2. ***Long-axis images*** should include an RV vertical long-axis view aligned with tricuspid valve inflow and an RV outflow tract view (sagittal or oblique sagittal plane through the pulmonary valve).

3.***Transaxial stack of cines*** covering the RV can be considered as an alternative for RV volume try.

Left ventricular outflow tact:

Plan the left ventricular outflow tract coronal cine on the 3-chamber images. Angle the position block parallel to the line along the center of the aortic valve and ascending aorta .

Planning for Aortic valve:

Plan the Aortic valve axial cine on the 3-chamber images. Angle the position block parallel to the aortic annulus .Check the position in the Left ventricular outflow tract coronal cine .An appropriate angle must be given in the LVOT coronal cine (parallel to aortic annulus).

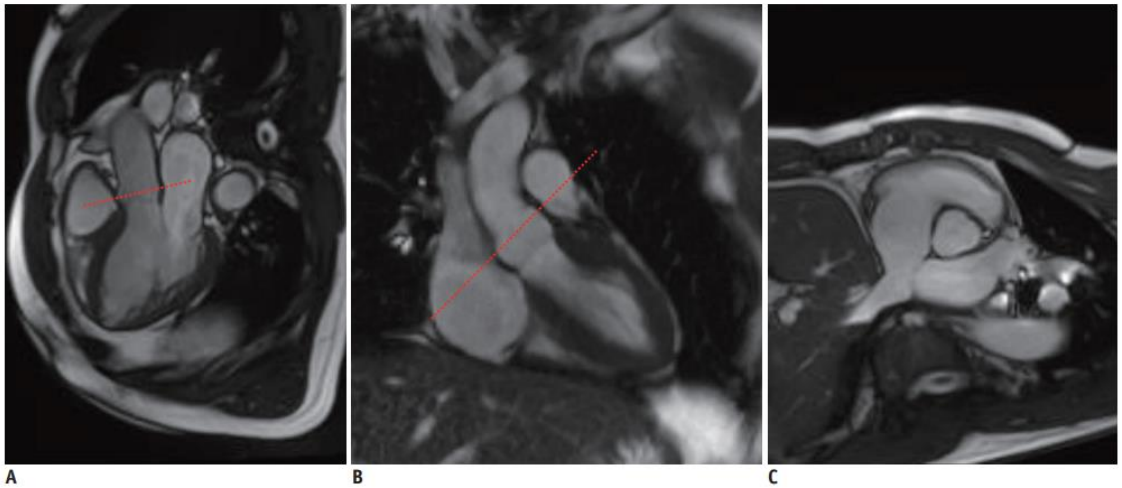

Fig. 16. How to obtain aortic valve (AV) view. A. 3-chamber view. B. Left ventricel outflow tract view. C. AV view. ***(15).***

Right ventricular out flow tract:

Plan the Right ventricular outflow tract cine on the axial bright images. Angle the position block parallel to the line bisecting the pulmonary trunk, pulmonary valves and right atrium. Check the position in the other two planes. An appropriate angle must be given in the 2-chamber localizer (parallel to the line bisecting the pulmonary outflow and pulmonary valves).

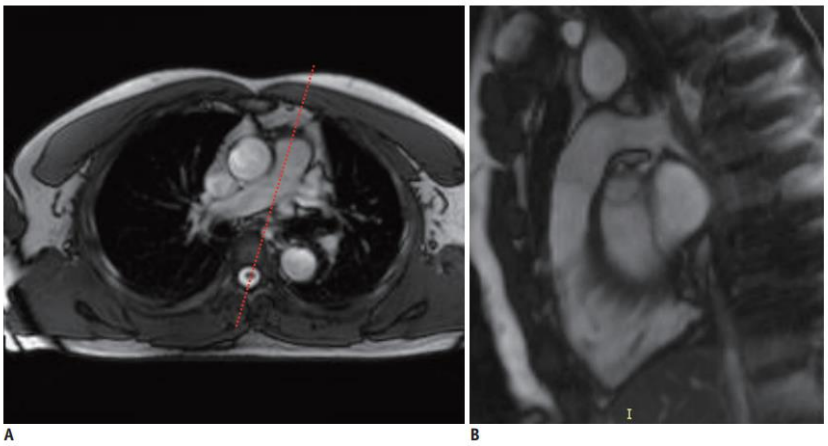

Fig. (17) How to obtain right ventricle outflow tract (RVOT) view. A. Axial scout image. B. RVOT view ***(15).***

Planning for pulmonary flow:

- Plan off the RVOT and PA cine views. Place the plane perpendicular to the PA on both these images. This gets the PA as circular as possible.

Planning for mitral valve

- Can be obtained from the 2-chamber view and 4-chamber view.
- Plane is parallel to the MV in the middle of the MV.

Planning for Tricuspid valve

-Can be obtained from the 2-chamber view and 4-chamber view.
- Plane is parallel to the TV in the middle of the TV.

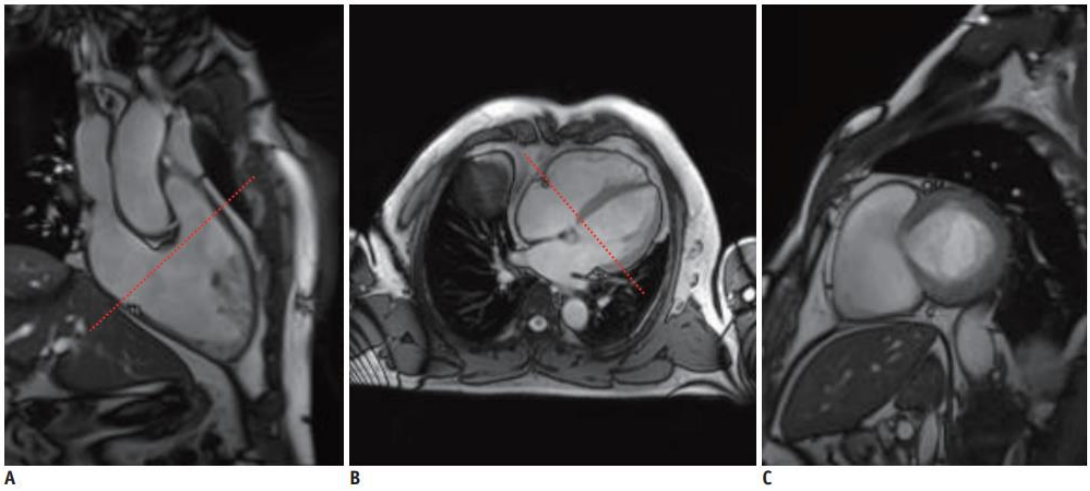

Fig. (18) How to obtain tricuspid valve (TV) view. A. Right ventricle 3-chamber view. B. 4-chamber view. C. TV view. ***(15).***

Flow imaging—conventional phase contrast cine MRI (PC-MRI):

-Flow quantification can be performed using specific CMR sequences and post-processing software. Phase-contrast velocity encoding is a technique that utilizes velocity-encoding (VENC) gradients to generate a phase shift in the magnetic resonance imaging signal, which is proportional to the velocity of the moving protons. Phase velocity images display the velocity of spins (positive and negative) in the direction of the applied velocity encoding gradients for each voxel. The VENC is an operator-controlled parameter which defines the maximum velocity that can be encoded without aliasing ***(16).***

In phase-contrast cine, the stationary tissue appears intermediate signal intensity on the phase contrast image, while the flowing blood would appear white or black signal intensity, depending on the direction of the flow relative to the velocity-encoding gradients,These two-dimensional phase velocity images can have velocity

encoded in the "in-plane" or "through-plane direction." The in-plane sequence allows for visualization of the stenotic or regurgitant jet and allow for planning of the "through-plane" phase-contrast image to quantify blood velocity and flow. These images can be processed using specialized software to calculate the velocity, volume, and direction of flow, thus allowing for quantification of stenosis or regurgitation.

- There are some limitations to phase-contrast velocity imaging such as: ***(16).***

(1) limited temporal resolution of 20–25 ms,. but, the temporal resolution is usually sufficient for most velocity and flow measurements.

(2) Another limitation is that the dephasing due to turbulent motion leading to loss of signal resulting from high-velocity turbulent flows. This limits the accuracy for velocities greater than 3.5 m/s. This may be problematic when assessing severity of aortic stenosis, or peak mitral regurgitation velocity.

-For quantification of peak velocities, it is important to ensure that the VENC direction is collinear with the direction of flow, or peak velocities may be underestimated.

- *An advantage of quantifying flow* with VENC imaging over Doppler-based flow measurements is that it does not require any geometric assumptions, as the velocity is known at each pixel location, and the number of pixels within a region of interest (ROI) is known. And CMR flow measurements have less angular dependence than Doppler techniques for quantifying flow. One other caveat for quantifying flow is that the slice location is fixed in space, and the valve moves relative to the slice, meaning that the velocities are not measured at the same anatomic location throughout the cardiac cycle. This may lead to some challenges when directly quantifying tricuspid or mitral flow but is usually not problem for quantifying flow in the proximal aorta and pulmonary artery.

CMR Quantification of AR Severity:

-The preferred method to quantify AR severity by CMR is to quantify the RVol which can be assessed even in the presence of coexisting valvular lesions. Flow through the aortic valve can be measured with phase-contrast velocity mapping in a plane perpendicular to the aorta. The imaging slice should be halfway between aortic annulus and sinotubular junction, near the leaflet tips in the sinuses orthogonal to aortic flow in two imaging planes. In the absence of obvious stenosis, a maximum VENC of 150 or 200 cm/s is typically sufficient. If aliasing occurs, then the VENC can be increased by 50 cm/s until aliasing does not occur. This allows for a more accurate assessment of peak velocity in the aorta.

-Aortic Rvol is calculated from the area under the retrograde diastolic flow curve.

-Regurgitant fraction (RF) is calculated with the following equation: RF = (regurgitant volume/forward volume) × 100%. ***(16).***

-As per the 2014 ACC/AHA guidelines, current values for echoc ardiographic-defined severe AR are RF > 50% and RVol > 60, which are the values commonly used today for CMR quantification.

CMR Quantification of Aortic stenosis

-CMR can be used for aortic valve anatomy assessment and to determine the degree of stenosis. Assessment of AS severity by CMR utilizes two parameters: planimetry of the valve area and peak velocity/gradient across the aortic valve ***(17)***.

Mitral valve

-Stenosis: Long-axis views of the ventricles usually demonstrate the restricted leaflets, and direct measurement of the orifice area can be performed by placing an imaging plane at the mitral or tricuspid valve tips during diastole **(18)**.

-**Regurge :** Cine imaging can be performed through the different scallops of the mitral valve (MV) leaflets to help assess the mechanism of MR, Secondary MR can be identified by assessing LV functional impairment and LV enlargement.

The jet of MR can be visualized by either SSFP or GRE cine images, but qualitative assessment is problematic as the appearance of signal void on cine images may be dependent on pulse sequence parameters.

Current guidelines recommend RVol and RF as the best method for quantification of MR severity. Measuring mitral regurgitation with phase-contrast imaging of the mitral valve is difficult due to the movement of the mitral valve annulus during ventricular systole, and due to the high velocity and turbulence of the regurgitant jet .

-Quantification of MR should be performed using SSFP imaging to quantify LV stroke volume (SV) and phase-contrast imaging to quantify forward flow volume across the aortic (or pulmonic) valves to quantify MR RVol and RF. The currently preferred method is to calculate the total LV SV and subtract this from the aortic forward flow volume.
-Alternative techniques include using pulmonic artery flow in place of aortic flow, or calculating the difference in LV and RV SV quantified by SSFP imaging, or by measuring the difference between mitral inflow SV and aortic SV by phase-contrast images. RF can be calculated by dividing RVol by LV SV in the volume in the first two methods and by mitral inflow SV for the third method ***(19).***

Pulmonary valve

-Qualitative assessment of pulmonic stenosis (PS) can be performed with "through-plane" phase-contrast imaging at the level of maximal stenosis at valvular, supravalvular, or subvalvular positions. Using post-processing software, a peak velocity can be obtained through the stenotic region.
-Pulmonic regurgitation (PR) can be quantified by assessing the regurgitant fraction using a through-plane phase-contrast image acquired at the level of the pulmonary artery just above the pulmonic valve ***(16).***

Tricuspid valve

-Quantification of flow across the tricuspid valve by phase velocity imaging is difficult due to the Trans annular motion of the valve, the regurgitant volume is **indirectly** measured by subtracting the main pulmonary artery forward volume from the RV systolic volume calculated using a stack of short-axis images ***(19).***
-***The cut-off values*** for severity grading were based mainly on the echocardiographic standards. For the TR fraction, recent guidelines suggest using the same severity cut-off used for mitral regurgitation (a regurgitant fraction ≤ 15% for mild TR, 16%–25% for moderate TR, 26%–48% for moderately severe TR, and > 48% for severe TR) ***(20).***
-PC-MRI is used for flow quantifcation of through cardiac valves and within great vessels . The blood flow of a vessel can be determined by the product of averaged velocity within the vessel lumen and the lumen area. The technique allows for quantifcation of cardiac output, valve regurgitation, severity of vascular and valvular stenosis, pulmonary to systemic flow ratio (i.e., QP/QS ratio, which reflects shunting of blood) and shunting flow **in congenital heart disease.** ***(21)***.

Pathophysiology of valve lesions:

1-Tricupid valve lesions:

- **Tricuspid regurgitation:**

Tricuspid regurgitation is comparatively a common anomaly. Structural modifications of any or all of the tricuspid valve apparatus may cause the development of tricuspid regurgitation ***.(22)***

Etiology:

TR can be divided into two types, primary and secondary, according to their pathophysiology:
(1) *Primary* : where the intrinsic abnormalities involving the valve apparatus are responsible.
(2) *Secondary* : where the right ventricular dilatation causes tricuspid regurgitation. Secondary disorders like tricuspid annular dilation and/or leaflet tethering or tenting, malcoaptation caused by RV dilatation, dysfunction right ventricular pressure and/or volume overload are largely responsible for tricuspid regurgitation as compared to primary disorders. ***(23).***

- ***Primary Tricuspid Regurgitation***

In adolescents and young adults, the cause of tricuspid regurgitation is usually congenital, but in adults, conditions that directly affect the tricuspid valvular apparatus are rare

Causes of primary TR include

- Direct valve injury from a permanent pacemaker or implantable cardioverter-defibrillator lead placement or removal or endo myocardial biopsy in cardiac transplant recipients.
- Chest wall or deceleration injury trauma
- Infective endocarditis
- Ebstein anomaly is the most common form of congenital disease affecting the tricuspid valve: The posterior and the septal leaflets of the tricuspid valve are displaced apically in the right ventricle resulting in its atrialization. ***(22)***

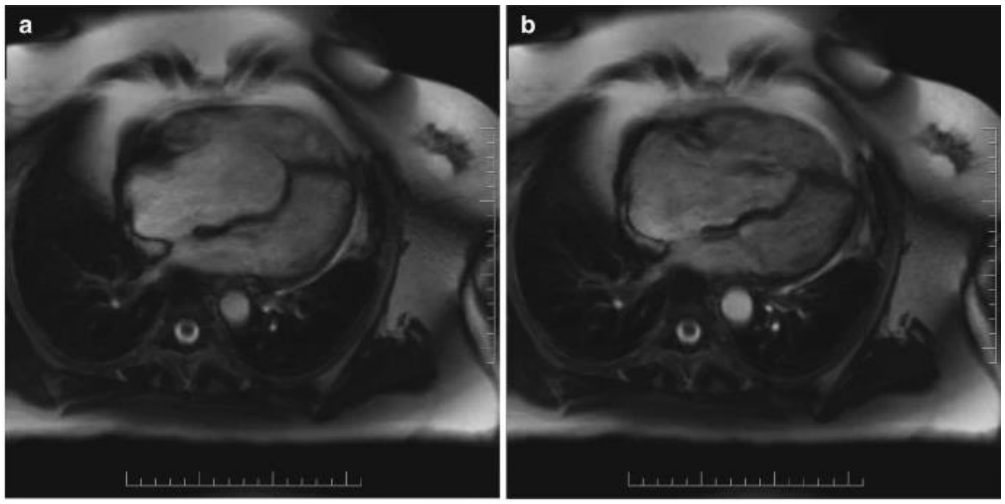

Figure (19) :Cardiac magnetic resonance imaging of severe tricuspid regurgitation in Ebstein anomaly; (**a**) four chamber view in diastole, (**b**) four chamber view in systole.***(24)***

- Rheumatic valve disease due to damage of the tricuspid leaflets. The valves undergo fibrous thickening without commissural fusion, fused chordae, or calcific deposits.
- Carcinoid syndrome: the valve leaflets adhere to the right ventricular wall owing to the fibrous plaques on the valve leaflets and the endocardium. Thus, the tricuspid cusps do not coapt appropriately during systole causing tricuspid regurgitation. ***(25).***
- Myxomatous degeneration associated with tricuspid valve prolapse.
- Connective tissue disorder as Marfan syndrome, Ehlers-Danlos: due to mildly dilated tricuspid annulus without floppy leaflets and floppy tricuspid valve leaflets.

Secondary Tricuspid Regurgitation

In adults, tricuspid regurgitation is most commonly secondary with probable normal anatomical leaflets and chords.

-This may result due to conditions affecting the right ventricle or may be due to an increase in right ventricle systolic pressure often with pulmonary hypertension. The valves are anatomically normal, but because of the enlarged right ventricular cavity and dilatation of the annulus, the leaflets fail to coapt appropriately.

- Conditions affecting the right ventricle that cause tricuspid regurgitation include (1) cardiomyopathies (2) ischemic heart diseases involving the right ventricular myocardium and tricuspid papillary muscles. ***(26)*** .
- The conditions that induce pulmonary hypertension and secondary RV dilatation include the following:

❖ Left-sided heart failure
❖ Mitral stenosis or regurgitation
❖ Primary pulmonary disease: Cor pulmonale, pulmonary embolism, pulmonary hypertension of any cause
❖ Stenosis of the pulmonic valve or pulmonary artery
❖ Hyperthyroidism.

Pathophysiology

Tricuspid regurgitation is defined by the backflow of blood from the right ventricle into the right atrium during systole. In mild to moderate cases of tricuspid regurgitation, no major hemodynamic consequences are noted due to the comparatively compliant nature of the right atrium. However, in severe cases, right ventricular volume overload develops eventually lead to right-sided congestive heart failure presenting with peripheral edema, ascites, and hepatic congestion. ***(22)***

The severity of tricuspid regurgitation is increased during inspiration. The right ventricle widens during inspiration, which further enlargement of the tricuspid valve annulus, leading to increase the effective regurgitant orifice area. ***(22)***

Tricuspid Stenosis

Tricuspid stenosis (TS) is even more rarely described. It most often co-exists with mitral valve pathology especially in patients with rheumatic heart disease **.**

-Etiology:

-Rheumatic heart disease is one of the most common causes of TS and almost always occurs in conjunction with mitral stenosis ***(22)***

-Large vegetations in infective endocarditis can cause relative stenosis **.**

-Carcinoid syndrome may cause isolated TS or mixed with the regurgitant lesion ***(20)***.

- Systemic diseases like systemic lupus erythematosus (SLE), antiphospholipid antibody (APLA) syndrome and the presence of permanent pacing and fusion of implantable cardioverter defibrillator leads to sub-valvular structures can cause tricuspid stenosis ***(23)***.

- Benign tumors like atrial myxomas can cause functional TS ***(23)***.

-Blunt trauma , Renal and ovarian tumors can grow into the tricuspid orifice causing stenosis .

Pathophysiology

-The rheumatic tricuspid disease is characterized by diffuse fibrous thickening of the leaflets and fusion of 2 or 3 commissures. Leaflet thickening usually occurs in the absence of calcific deposits, and the antero septal commissure is most commonly involved. ***(27)*** **.**

-Incompletely developed leaflets, shortened or malformed chordae, a small annulus, or an abnormal number or size of papillary muscles may result in TS.

-Genetic or acquired/environmental causes that disrupt the normal organization and composition of the extracellular matrix and communication between valve endothelial cells (VECs) and interstitial valve cells (VICs) alter valve mechanics and interfere with the valve leaflet function, culminating in heart failure .

-The primary result of TS is right atrial pressure elevation and consequent right-sided congestion.

Mitral valve

Mitral regurgitation (MR):

-MR is the most common valvular abnormality, is caused by the retrograde flow of blood from the left ventricle (LV) into the left atrium (LA) through the mitral valve (MV) ***(28).***

-Mitral regurgitation can subdivide into primary and secondary causes.

Primary Mitral Regurgitation

Also called degenerative or organic, Any MR resulting from structural deformity of or damage to the leaflets, chordae, and/or papillary muscles causing leaflets to close insufficiently during systole ***(28).***

Mitral valve prolapses (MVP)

Etiology:

either as a primary or secondary caused by Marfan syndrome, Ehlers-Danlos syndrome, systemic lupus erythematosus (SLE), osteogenesis imperfecta, and pseudoxanthoma elasticum.

- Mitral leaflet perforation.
- Isolated cleft of the mitral valve, double orifice mitral valve, and parachute mitral valve (PMV), which is a congenital valvular anomaly where the chordae tendineae are attached to a single papillary muscle, have been linked to causing MR. While extremely rare.

Secondary Mitral Regurgitation

-Also called functional or ischemic. No structural problems with the valve itself **,**Leads to mitral annular dilatation or displacement of papillary muscles causing retrograde flow from improperly closed mitral valve leaflets. ***(29).***

-Etiology :

- Papillary muscle rupture: leads to severe mitral regurgitation due to dysfunction of the papillary muscle. ***(29).***
- Ischemic cardiomyopathy: ischemia of the segments underlying the papillary muscles results in remodeling. Causing papillary muscle displacement, which results in a more apical position of the leaflets known as a “seagull sign. ***(29).***
- Dilated cardiomyopathy.

- Atrial fibrillation (AF): cause increased atrial and valve annular size, resulting in functional MR.
- Hypertrophic Cardiomyopathy: severe left ventricular hypertrophy, which causes increased papillary muscle mass, bringing them closer together. Causing the mitral valve leaflets to become elongated and floppy and pulls the leaflets closer to the left ventricular outflow tract regurgitant retrograde flow ***(29).***

Carpentier's Classification

- Type 1**:** Normal leaflet motion
  - Caused by annular dilation or leaflet perforation

Regurgitation jet directed centrally (***30).***

- Type 2: Excessive leaflet motion
  - Caused by papillary muscle rupture, chordal rupture, or redundant chordae

Eccentric jet directed away from the involved leaflet (***30).***

- Type 3: Restricted leaflet motion
  - IIIa: Leaflet motion restricted in both systole and diastole
    - Caused by rheumatic heart disease commonly
    - Normal papillary muscles
    - Jet may be centrally or eccentrically directed
  - IIIb: Leaflet motion restricted in systole
    - Caused by papillary muscle dysfunction or left ventricular dilation
    - Abnormal papillary muscles
    - Jet may be centrally or eccentrically directed.

Pathophysiology

The definition of mitral regurgitation is a retrograde flow from the left ventricle into the left atrium. Mitral regurgitation leads to left ventricular volume overload due to increased stroke volume, caused by an increase in blood volume within the left atrium and leads to increased preload delivered to the left ventricle during diastole. In chronic progressive MR, ventricular remodeling occurs, allowing maintenance of cardiac output, and an initial increase in ejection fraction (EF) is usually observed. However, depending on the regurgitant fraction, the effective EF can be considerably lower. Over time, there is a positive feedback loop by which volume overload from MR causes ventricular dilatation, widening of the mitral annulus, and diminished coaptation of leaflets, leading to further worsening of MR. Eventually, volume overload becomes so severe that excitation-contraction coupling becomes impaired and wall stress-related afterload on the left ventricle leading to dilatation and decreased contractility, resulting in a reduction of EF ***(30).***

Mitral stenosis

Mitral stenosis (MS) is a form of valvular heart disease. Mitral stenosis is characterized by narrowing of the mitral valve orifice.

-Etiology:

- Rheumatic fever: The most common cause of mitral stenosis.
- Calcification of the mitral valve leaflets
- Congenital heart disease.
- Infective endocarditis.
- Endomyocardial fibroelastosis.
- Malignant carcinoid syndrome.
- Systemic lupus erythematosus, Whipple disease, Fabry disease, and rheumatoid arthritis (***31).***

Pathophysiology

The mitral valve is a bi leaflet valve positioned between the left atrium and left ventricle. The normal mitral orifice area is 4 to 6 square centimeters. Under normal physiologic conditions, the mitral valve opens during the left ventricular diastole to allow blood to flow from the left atrium to the left ventricle. The pressure in

the left atrium and the left ventricle during diastole are equal. The left ventricle gets filled with blood during early ventricular diastole. Only a small amount of blood remains in the left atrium. This small amount of blood fills the left ventricle with the contraction of the left atrium (the "atrial kick") during late ventricular diastole ***(32)***.

-Mitral valve areas less than 2 square centimeters cause an impediment to the blood flow from the left atrium into the left ventricle. This creates a pressure gradient across the mitral valve. As the gradient across the mitral valve increases, the left ventricle requires the atrial kick to fill with blood ***(32)***. -Mitral stenosis causes an increase in left atrial pressure. The normal left ventricular diastolic pressure is 5 mmHg. A pressure gradient across the mitral valve of 20 mmHg due to severe mitral stenosis will cause a left atrial pressure of about 25 mmHg. This left atrial pressure is transmitted to the pulmonary vasculature resulting in pulmonary hypertension.

-As left atrial pressure remains elevated, the left atrium will increase in size. there is a greater chance of developing atrial fibrillation. If atrial fibrillation develops, the atrial kick is lost.

Thus, the left ventricular filling depends on the atrial kick in severe mitral stenosis. With the loss of the atrial kick, there is a decrease in cardiac output and sudden development of congestive heart failure ***(32)***.

Aortic valve

I-Aortic stenosis

Aortic stenosis is a common valvular disorder, especially in the elderly population, causing left ventricular outflow obstruction

Etiology

Congenital

Etiology:

-Congenital:

- The bicuspid aortic valve is the most common cause of aortic stenosis in patients less than the age of 70 years in developed countries.
- A congenitally abnormal valve with superimposed calcification can cause aortic stenosis ***(33).***

- Acquired:

- Rheumatic valve disease is the most common cause in developing countries ***(33).*** The commissures of the leaflets fuse to leave a small central orifice.
- Calcification of the tri-leaflet valve,
- alkaptonuria, systemic lupus erythematosus, ochronosis, irradiation, homozygous type II lipoproteinemia, and metabolic diseases such as Fabry disease **.**
- Mineral metabolism disturbances, such as end-stage renal disease, have also shown to contribute to the calcification of the valve ***(33).***
- Hypertrophic cardiomyopathy : can cause dynamic subvalvular stenosis.

Pathophysiology:

- Left ventricular (LV) obstruction caused by the stenosis of the valve increases LV systolic pressure. It also results in increased LV ejection time (LVET), decreased aortic pressure, and increased LV end-diastolic pressure.

-The increased afterload, in addition, to an increase in LV volume overload, leads to an increase in LV mass, leading to LV dysfunction and failure.

-Myocardial oxygen consumption increases with increased LV systolic pressure, LV mass, and LVET, while myocardial perfusion time decreases with increased LVET. leading to LV function further deteriorates with increased myocardial oxygen consumption and decreased myocardial oxygen supply **(34)**.

Aortic regurgitation

Aortic regurgitation is the inadequate closure of the aortic valve during diastole that results in reverse blood flow through the aortic valve. This condition can occur as a native valvular disease or as a result of aortic root dilatation.

Etiology:

AR results from mal-coaptation of the aortic leaflets due to abnormalities of the aortic leaflets, their supporting structures such as the aortic root and annulus, or both. ***(35).***

Primary valve disease:

- Congenitally bicuspid aortic valve (BAV) may cause AR due to incomplete closure or prolapse of the valve, although AS is a more common complication of BAV.
- Congenital AR due to unicommissural and quadricuspid valves, or rupture of a fenestrated valve are less common.
- Prolapse of an aortic cusp occurs in some patients with ventricular septal defect (VSD).

Secondary valve disease *(35).*

- Rheumatic disease results in fibrous infiltration of AV cusps leading to retraction that prevents proper opening during systole and closure during diastole. The associated fusion of the commissures may lead to combined AS and AR. Rheumatic aortic valve disease is often associated with rheumatic mitral valve disease.
- Calcific aortic valve disease, which is usually associated with aortic stenosis (AS) but can be associated with some degree of AR.
- Infective endocarditis, which alters the anatomy of the leaflets; tear or laceration in the ascending aorta which leads to prolapse of the aortic cusp due to loss of commissural support
- Myxomatous degeneration of the aortic valve can also lead to progressive AR.
- Membranous subaortic stenosis may lead to thickening and scarring of the AV leaflets resulting in secondary AR.
- AR is complication of percutaneous aortic balloon valvotomy and transcatheter aortic valve replacements. ***(35)***
- Traumatic rupture or avulsion of an aortic cusp is an uncommon cause of acute AR.
- Other less common causes of AR occur in association with systemic lupus erythematosus, Takayasu disease, Whipple disease, rheumatoid arthritis, ankylosing spondylitis, Jaccoud arthropathy, syphilis, Crohn disease, and appetite-suppressing drugs

Pathophysiology

AR causes left ventricular volume overload. An increase in LV end-diastolic volume causes dilation and eccentric hypertrophy of the LV. This allows ejection of a larger stroke volume. In patients with AR, the total stroke volume ejected by the LV is the sum of effective stroke volume and the regurgitant volume Thus, AR is associated with increased preload. LV dilation increases the LV systolic tension in accordance with the law of Laplace. This, in combination with the elevated systolic blood pressure that results from the increase in total forward stroke volume, leads to increased afterload. ***(36).***

-LV function is compensated due to the combination of LV dilation and hypertrophy. Over time, but , wall thickening fails to keep pace with the hemodynamic load resulting in a decline in systolic function and ejection fraction.

-Decompensation of the LV results in decreased compliance and increased LV end-diastolic pressure and volume. In advanced stages, left atrial, pulmonary artery wedge, pulmonary arterial, right ventricular (RV), and right atrial pressures rise, and the effective (forward) cardiac output falls. Symptoms of heart failure develop, including dyspnea, orthopnea, and paroxysmal nocturnal dyspnea due to pulmonary congestion. ***(36).***

-An increase in LV mass leads to increased myocardial oxygen requirements. Also, coronary perfusion pressure is reduced. This causes myocardial ischemia and exertional chest pain.

-In patients with acute severe AR, compensatory mechanisms of the LV do not develop rapidly enough to handle the regurgitant volume load. LV diastolic pressures rise rapidly and may lead to acute pulmonary

edema and cardiogenic shock. Even diastolic mitral regurgitation can occur as a result of the sudden severe increase in LV volume and pressure, ***(36).***

Pulmonary stenosis

Pulmonic stenosis is a defect of the pulmonic valve in which the valve is stiffened, causing an obstruction to flow.

Etiology:

- Congenital structural cardiac syndromes, including tetralogy of Fallot and Noonan syndrome(**37) .**
- Maternal rubella syndrome is also a common cause of congenital pulmonic stenosis (**37) .**
- Carcinoid syndrome (**37) .**
- Rheumatic heart disease.
- Previous cardiothoracic surgeries, or a cardiac tumor may acquire pulmonic stenosis.

Pathophysiology

-Isolated pulmonic stenosis is divided in to valvular, subvalvular, and supravalvular obstruction.

(1)-Valvular pulmonic stenosis is the most common type. In typical valvular disease, the commissures are partially fused, and leaflets are thin. This structural anomaly results in a dome-shaped or conical outlet seen during systole. Less commonly, the pulmonic valves can be dysplastic, thickened, and without fusion. The associated hypoplastic annulus and proximal pulmonary artery are common in atypical presentations and are also associated with Noonan syndrome The atypical presentation generally requires intervention in the early years of life. Bicuspid valves are associated with tetralogy of Fallot. ***(38).***

(2)-Subvavular pulmonic stenosis: is a defect obstructing the infundibular region. A primary cause of fibromuscular narrowing of the right ventricular outflow tract may be associated with a double-chambered right ventricle or tetralogy of Fallot ***(38).***

Secondary causes of subvalvular pulmonic stenosis may be a result of primary valvular stenosis, inducing hypertrophy of the right ventricle. Secondary stenosis often regresses with valvotomy or valvuloplasty ***(38).***

(3)Supravalvular pulmonic stenosis: also known as peripheral pulmonary stenosis, is a functional obstruction originating from the pulmonary artery. The blockage may occur at the main artery, bifurcation point, at distal branches of the pulmonary artery, or any combination of there . Supravalvular stenosis is associated with structural defects of atrial septal defects, ventricular septal defects, patent ductus arteriosus, and tetralogy of Fallot. Supravalvular stenosis may also be a result of surgical repair of transposition of the great arteries.

Pulmonary regurgitation

The pulmonary valve directs blood from the right ventricle (RV) towards the pulmonary arteries during systole. Equally important is its closure during diastole to prevent the reversal of flow into the right ventricle driven by the drop in right ventricular pressure.

Etiology

- Surgical valvulotomy and balloon valvuloplasty are the most common causes of iatrogenic pulmonary valve regurgitation and pathological pulmonary regurgitation overall such as tetralogy of Fallot ***(39).***
- Pulmonary arterial hypertension.
- Carcinoid disease.
- Infective endocarditis.
- Rheumatic heart disease.
- Congenital pulmonary regurgitation.

Pathophysiology

-Defective closure of the pulmonary valve prevents forward flow and causes blood to regurgitate back into the right ventricle during diastole.

-Flow in the heart is determined by pressure gradients. Pressure in the right ventricle during diastole, right ventricular end-diastolic pressure (RVDP), is initially lower than the pressure in the pulmonary arteries in diastole.

-Physiologically the volume of blood ejected during systole into the pulmonary circulation is prevented from regurgitating back into the right ventricle despite the drop in its pressures due to a functioning pulmonary valve.
-In pathological pulmonary regurgitation, the valve is defective and allows blood to regurgitate into the right ventricle.
The ventricles experience volume overload, which is initially accommodated for. progressively over time, the ventricles undergo eccentric hypertrophy , resulting in ventricular dilation ***(40)*** .
-The purpose of this remodeling is to accommodate for the increased stroke volume and to maintain cardiac output in the next cycle of systole ***(40)*** .
-If pulmonary regurgitation is left untreated, chronic volume overload to the right ventricle can lead to right heart failure.
-The eccentric ventricular remodeling can no longer compensate and results in systolic dysfunction of the heart.
- There are two consequences that occur as a result.:
1- Systolic dysfunction can result in a reduced cardiac output leading to symptoms such as lightheadedness and syncope.
2-Right ventricular pressure and volume increase and are transmitted to the jugular veins and the venous system of the heart, presenting as the classical signs and symptoms of right heart failure ***(40)*** .

References values of heart ventricles

CMR provides highly accurate assessments of both ventricles, including volumes, mass, and function, typically involving a stack of contiguous slices from base to apex.

Left ventricular dimensions and functions:

The primary method used to assess the LV is balanced steady-state -free precession (bSSFP) technique at 1.5 or 3 T CMR , Papillary muscle mass has been shown to significantly affect LV volumes and mass ***(41).***

- LV volumes and mass were indexed by body surface area and measurements of LV diameter obtained on cine bSSFP images at diastole and systole on a 4 chamber view and short axis view. ***(41).***

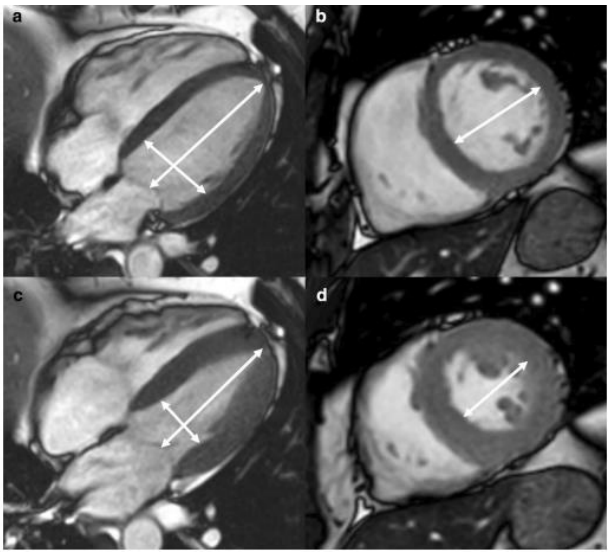

Figure (20) Measurements of LV diameters obtained on cine bSSFP images during diastole (a, b) and systole (c, d) on the 4 chamber view (a, c) and short axis view (b, d). The longitudinal diameter of the LV was measured on the 4 chamber view as the distance between the mitral valve plane and the LV apex (a, c). On the 4 chamber view the transverse diameter was defned as the distance between the septum and the lateral wall at the basal level. On the short axis view the transverse diameter was obtained at the level of the basal papillary muscles (b, d) ***(41).***

-Maximal LV wall thickness was measured in end diastole at (septal, septo-apical, apical, inferolateral). ***(42).***
- Left ventricular parameters for adult (papillary muscles included in LV volume) according to reference ***(42).***

Right ventricular dimensions and functions:

-For measurement of right ventricular (RV) volumes, a stack of cine bSSFP images is acquired either in the short axis plane or trans axial plane, analysis of the RV is usually performed on a per slice basis by manual contouring of the endocardial and epicardial borders ***(43)***.
- The RV volumes and mass are significantly affected by inclusion or exclusion of trabeculations and papillary muscles ***(43)***.

Right ventricular parameters for adult athletes (papillary muscles included in right ventricular volume) according to reference ***(43)***.

Conflicts of Interest: The authors declare no conflict of interest.

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