Right heart on multidetector CT

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ABSTRACT. Right ventricular function plays an integral role in the pathogenesis and outcome of many cardiovascular diseases. Imaging the right ventricle has long been a challenge because of its complex geometry. In recent years there has been a tremendous expansion in multidetector row CT (MDCT) and its cardiac applications. By judicious modification of contrast medium protocol, it is possible to achieve good opacification of the right-sided cardiac chambers, thereby paving the way for exploring the overshadowed right heart. This article will describe the key features of right heart anatomy, review MDCT acquisition techniques, elaborate the various morphological and functional information that can be obtained, and illustrate some important clinical conditions associated with an abnormal right heart.

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The body of literature illustrating the use of multidetector row CT (MDCT) in the assessment of cardiac diseases has exponentially increased with advances in CT technology. Although electrocardiogram (ECG) gating is invaluable in the evaluation of cardiac structures, in the setting of undiagnosed cardiorespiratory symptoms, non-gated thoracic CT is more commonly adopted. With modern scanners, even these studies offer the opportunity to assess the heart for diseases that may otherwise go undetected.

Evaluation of coronary artery disease is perhaps the mainstay of a gated cardiac CT examination. MDCT has been shown to be complementary and, in some cases, a reliable alternative to other non-invasive imaging modalities in the evaluation of non-coronary cardiac abnormalities. The author acknowledges that the emphasis of most gated cardiac CT examinations is mainly placed on the left heart, with optimisation of injection protocols to achieve this aim. This usually results in varying levels of enhancement of the right heart. However, by altering the volume and rate of contrast medium injection, it is possible to achieve good opacification of the right-sided cardiac chambers, thereby paving the way for exploring the overshadowed right heart.

This article will review the right heart anatomy and MDCT acquisition parameters with emphasis on contrast medium protocols and appropriate imaging planes to delineate the right side, elaborate the various morphological and functional information that can be obtained and illustrate some important clinical conditions associated with an abnormal right heart.

Right heart anatomy

Right atrium

The right atrium (RA) is a complex structure with distinctive topographic features that not only define

the anatomy but also carry practical implications for interventional procedures. The RA chamber begins at the venoatrial junctions and terminates at the fibrofatty tissue plane of the atrioventricular junction. It is larger than the left atrium, with thinner walls measuring up to 2 mm. A vertical muscular ridge, the crista terminalis, is located at the junction between the RA and the superior vena cava and separates the smooth venous component of the RA from the rough appendage wall. The RA appendage is a triangular anterior projection with its apex pointing superiorly. An extensive array of muscular fibres called pectinate muscles fan out at right angles from the crista, forming a series of interconnecting parallel ridges within the appendage (Figure 1). The sinus node lies in the musculature of the terminal crest, usually at the anterolateral junction with the superior vena cava. A critical zone of tissue located between the tricuspid annulus and the inferior vena cava, the cavotricuspid isthmus, facilitates perpetuation of the re-entrant circuit, and hence is an ideal target for radio-frequency ablation procedures (Figure 2) [1]. The extent of the pectinate muscle from the atrioventricular junction is the most accurate criterion for determining the laterality of the atrium, but this cannot be delineated on CT owing to insufficient spatial resolution [2]. The superior vena caval orifice is valveless. A rudimentary Eustachian valve is present at the inferior vena caval orifice and directs flow towards the foramen ovale. The Chiari network is a lax tissue representing remnants of the valve and can simulate the intra-atrial thrombus [3]. The smaller coronary sinus orifice guarded by the Thebesian valve is situated between the orifice of the inferior vena cava and the atrioventricular opening (Figure 3). The sinus can be distended when it receives more than its normal venous return, as can happen with persistent left superior vena cava (SVC).

The triangle of Koch, defined by the tendon of Todaro (a fibrous extension from the Eustachian), the orifice of the coronary sinus and the septal leaflet of the tricuspid valve, is another important anatomical landmark as it is the location of the atrioventricular node. The boundaries of the triangle and its relation to other anatomic structures can be traced on MDCT [4].

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Figure 1. Crista terminalis. Axial CT depicts the pyramidal right atrial appendage with the large crista terminalis (black arrow). The prominent anterior pectinate muscle arising from the crista is known as “septum spurium” (white arrowhead). There is a large variability in the appearances of the crista and pectinate muscles and these should not be mistaken for intracavitary mass or thrombus.

Partitioning the atrial chambers, the true septum is limited to the valve of the oval fossa and its immediate muscular rim [5]. The fossa ovalis is an oval depression on the lower part of the atrial septum, above and to the left of the orifice of the inferior vena cava. A patent fossa ovalis is the result of an incomplete seal between the valve and the anterosuperior quadrant of the rim, and is present in approximately 25–30% of the population [6]. The superior rim of the fossa, referred to as the “septum secundum”, is an extensive infolding between the venous component of the RA and the right pulmonary veins (Figure 4).

The vestibule of the RA is the smooth-walled atrial myocardium that inserts into the leaflets of the tricuspid valve.

Right atrioventricular valve

The tricuspid valve is slightly more apical in location than the mitral, with a larger triangular orifice. It is composed of three leaflets (*i.e.* anterior, posterior and septal) attached to a fibrous annulus. The anterior leaflet is often the largest, with the septal leaflet being the smallest. The latter attaches to the membranous and muscular portions of the ventricular septum. The more apical hinge line of the tricuspid septal leaflet relative to the anterior mitral leaflet is a useful criterion to identify the morphological right ventricle. The leaflets usually attach via chordae tendineae to three papillary muscles that are an integral part of the right ventricular wall (Figure 5).

Right ventricle

The complex right ventricular geometry makes its study a challenging proposition. It is anterior and retro-sternal in position and is demarcated by the annulus of the tricuspid and pulmonary valves. The right ventricle (RV) is triangular from the side and crescentic in cross-section. The RV wall is thinner than that of the left ventricle (LV), measuring 3–4 mm, being thicker at the base than at the apex [7]. Its posterior wall is formed by the ventricular septum, which is concave towards the LV in both systole and diastole [8]. Compared with the LV, the RV has a larger volume but only approximately one-sixth of the LV mass [9].

Anatomically, it is best to divide the RV into three components: (i) the inlet, composed of the tricuspid valve, chordae tendineae and papillary muscles; (ii) the trabeculated apical myocardium; and (iii) the infundibulum, or conus, supporting the leaflets of the pulmonary valve [10] (Figure 6). A more simplistic way is to

Figure 2. Cavotricuspid isthmus. Coronal CT demonstrates right atrial isthmus (white double-headed arrow) between the inferior vena cava (IVC) and tricuspid annulus (white arrow). RA, right atrium; RV, right ventricle; RVOT, right ventricular outflow tract; SVC, superior vena cava.

Figure 3. Coronary sinus. (a) Sagittal CT shows the coronary sinus (CS) with the Thebesian valve (black arrow) at its orifice. The small atrial outpouching inferior to the Thebesian valve (white arrowhead) is called the subthebesian pouch. A large pouch may make it difficult to access the sinus. (b) CT volume-rendered image. The white block arrow points to great cardiac veins, the white arrow to posterior interventricular vein, the white arrowheads to posterior veins and the star marks coronary sinus entry into the right atrium.

view the RV as basal, mid and apical sections with anterior, lateral and inferior walls (Figure 7).

The trabeculae carneae are irregular muscular columns that project from the inner surface of the ventricle. With the exception of the conus arteriosus, they give rise to the characteristic coarse trabecular pattern. There are three muscular bands within the RV. One of the bands extends from the base of the anterior papillary muscle to the ventricular septum and may assist in preventing over-distension of the ventricle; this is therefore called the moderator band (Figure 8). The parietal and septal bands form a prominent muscular shelf, the supraventricular crest, which separates the sinus and conus regions. This crest courses between the pulmonary and the tricuspid

valves and assists in right ventricular free wall contraction towards the interventricular septum. It plays an important role in emptying the RV and closing the tricuspid valve [11]. The RV also contains multiple septal and free wall papillary muscles. The pulmonary artery arises from the conus and is positioned anterior and to the left of the aorta. The pulmonary valve sits on a free-standing muscular infundibulum and has a semilunar shape with three leaflets, namely right, left and anterior. Compared with the aortic valve, the pulmonary valve cusps are thinner, with no fibrous continuity with the corresponding tricuspid valve leaflet (Figure 9). Between the semilunar valves and the wall of the pulmonary artery are the sinuses of Valsalva and the sinotubular junction.

Figure 4. Interatrial septum. (a) Sagittal CT demonstrates marked bi-atrial dilatation with a small patent foramen ovale (PFO) (black arrow) in a patient with severe rheumatic mitral stenosis. There is a jet of contrast medium from the left atrium (LA) to the right atrium (RA) via the PFO. (b) Aneurysmal interatrial septum (arrow) on an axial CT. (c) Axial CT showing an ostium secundum type atrial septal defect (ASD) (arrow). Also note the gross right ventricular (RV) hypertrophy (white arrowheads), a reflection of severe pulmonary hypertension.

Figure 5. Axial CT in a patient with pulmonary hypertension. Fine chordae tendineae (black arrow) connect the prominent papillary muscles to tricuspid valve leaflets (white arrowheads). RA, right atrium; RV, right ventricle.

The right ventricular wall is mainly composed of circumferentially arranged superficial and longitudinally orientated deep muscle layers [12]. Right ventricular contraction is sequential and phasic. It begins at the inlet, progresses to the trabeculated myocardium and ends at the infundibulum (approximately 25–50 mm apart) [13]. Right ventricular systole involves tricuspid annular descent and free wall motion, primarily due to longitudinal contraction, which differs from the radial LV contraction [14]. The low resistance of the pulmonary vasculature makes it feasible for the compliant RV to pump the same stroke volume as the LV with much less effort.

The blood supply to the RV is mainly from the right coronary artery, with the acute marginal branches supplying the lateral wall and the posterior descending artery supplying the inferoseptal segments. The branches of the left anterior descending artery supply the anterior and anteroseptal regions. The conus artery supplies the infundibulum [13, 15] (Figure 10).

Figure 6. CT volume rendered image elegantly illustrates various components of the right ventricle (RV). White arrowheads point to the trabeculated apex and black arrows to the smooth infundibulum. Note the ridging of the pectinate muscles in the right atrium (RA). MPA, main pulmonary artery.

It is of fundamental importance to be able to distinguish the right heart chambers from the left, particularly in complex congenital heart disease. Table 1 is a list of the major morphological factors that help in differentiation and comparing the corresponding right and left chambers.

MDCT acquisition technique

ECG-gated cardiac CT acquisition parameters vary depending on scanner specifications, and are described elsewhere in this issue. Standard injection techniques

Figure 7. Short axis CT of the right ventricle (RV) in diastole. (a) Basal, (b) mid and (c) apical sections. Within the RV, black arrows point to the lateral wall, white to the inferior wall and red to the anterior wall, the white star to interventricular septum and the blue arrows in both ventricles to papillary muscles. LV, left ventricle.

Figure 8. Axial four-chamber view on CT depicts the moderator band (white arrowhead) extending across the right ventricle apex from the anterior papillary muscle to the interventricular septum, which contains the right bundle branch.

Figure 9. Coronal CT reformat reinforces the lack of fibrous continuity between the pulmonary valve (PV) and tricuspid valve (TV). The aortic sinus (white arrowhead) projects between these two valves.

used for dedicated coronary CT angiography are optimised for left heart chambers, resulting in varying levels of enhancement on the right. When it is desirable to examine the right heart, the injection protocol has to be modified so as to maintain adequate contrast on the right side of the heart during image acquisition. This can be achieved by adjusting the amount and flow rate of contrast medium to prolong the length of administration of the entire bolus [16]. However, monophasic injection of contrast medium alone at a uniform rate can result in streak artefacts in the SVC and also reduces the total amount of contrast media required for optimal enhancement [17]. On the downside, a pure saline chaser can decrease the attenuation in the RA and RV and also make it difficult to trace the right ventricular septal endocardial contour.

Clear delineation of the endocardial contour is essential for functional assessment. Multiphasic infusions of varying combinations of contrast material, saline contrast mixture and saline flush can be adopted to provide a more homogeneous enhancement profile over time (Figure 11).

A comparative study by Utsunomiya et al [18] examined varying modes of contrast material delivery using 16 MDCT with bolus-tracking timing. A biphasic infusion of contrast medium followed by a contrast-saline mixture was found to be optimal for right ventricular functional analysis. This was attributed to better delineation of the ventricular wall by the residual diluted contrast medium. A retrospective analysis by Kerl et al [19] of studies from groups of 25 subjects scanned with 3 different protocols (monophasic, 50–75 ml; biphasic,

Figure 10. Maximum intensity projection (MIP) image of right coronary artery (RCA). (a) Conus branch (CB); sinus branch (SB); acute margin (AM) supplying the anterior wall of right ventricle (RV). (b) Posterior descending artery (PDA). (c) A separate origin of CB (black arrow) from the aorta is seen in about 30–35% of patients.

Table 1. Distinctive anatomical features of right heart chambers

Right atrium (RA)
1. RA appendage is larger and more triangular with a broad junction with RA chamber
2. Prominent terminal crest between the ridged RA appendage and smooth venous component
Right ventricle (RV)
1. RV chamber including the apex is coarsely trabeculated and contains the moderator band
2. Presence of a well-developed infundibulum and multiple (three or more) papillary muscles along the septum and free wall
3. Presence of three atrioventricular valve leaflets with septal papillary attachments with lack of fibrous continuity between the tricuspid and pulmonary valves.

50–75 ml, followed by a 50 ml saline flush; or a split-bolus, 50–75 ml of contrast material, 50 ml of a 70:30 saline–contrast mix and a 30 ml saline flush) of 370 mg I ml⁻¹ contrast concentration infused at 5 ml s⁻¹ using test bolus timing also showed that split bolus infusion was optimal for delineation of right heart papillary muscles and the myocardium, the moderator band, and the tricuspid and pulmonary valves. Also, split bolus infusions result in better delineation of the intraventricular septum [20]. Lu et al [21] demonstrated that a triphasic protocol (60–65 ml of contrast media, 20 ml of 70:30 saline–contrast mixture and 40 ml of saline flush) resulted in better and prolonged right heart enhancement without beam-hardening artefacts. However, the pure saline flush may wash the contrast out of the pulmonary circulation, particularly when the acquisition time is prolonged or there is a slow right-to-left contrast material transit time [22]. Hence, these injection parameters have to be adjusted when it is necessary to additionally image the pulmonary arteries. Table 2 lists the salient points involved in obtaining optimal right heart enhancement.

MDCT: post-processing and image analysis

Even on a non-ECG gated thoracic CT, it is possible to obtain images encompassing four to five cardiac

cycles during a single acquisition. These are usually reconstructed at 2.0 mm thickness and 1.5–2.0 mm increments on a medium (soft-tissue) reconstruction kernel. Careful analysis of such images can provide useful information on cardiac morphology. ECG gating permits ventricular assessment without motion artefact. At the author's institution, ECG-gated cardiac CT is reconstructed at 0.75 × 0.5 mm increments for coronary analysis. The thinly reconstructed axial slices serve as the primary data set from which oblique cardiac planes are obtained. The horizontal four-chamber and short axis views are most useful for right heart analysis. The four-chamber view is aligned along the intrinsic axes of the heart and is therefore more anatomically precise than the standard MDCT multiplanar reformats [24]. The short-axis view is orthogonal to the four-chamber view and is orientated perpendicular to the ventricular septum (Figure 12). This is a crucial projection for functional analysis. A detailed paper by Lu et al [25] describes the various steps involved in creating these cardiac-specific views.

Standardised measurements of normal myocardial thickness and intracardiac dimensions have been described for echocardiography and MRI but have not yet been established for CT. A transverse right atrial diameter of more than 35 mm and a transverse right ventricular diameter of more than 45 mm are considered to be abnormal on MDCT [26]. The interatrial septum can

Figure 11. Axial CT images from different patients with varying levels of enhancement in the right heart. (a) Monophasic injection with resultant streak artefact due to beam hardening caused by dense non-diluted contrast in the right atrium (RA) (red arrow). (b) Biphasic injection optimised for left heart and coronary visualisation. Note the complete wash out of contrast medium from the right side. (c) Split bolus injection technique results in better right-sided opacification. LV, left ventricle; RV, right ventricle.

Table 2. Optimisation of right heart enhancement

1. Use high concentration contrast medium ($320\text{--}370\text{ mg I ml}^{-1}$) [23] to achieve target arterial enhancement
2. Use split-bolus infusion. Miss out the pure saline flush if simultaneous pulmonary opacification is desired
3. Maintain a rapid flow rate of 5 ml s^{-1} throughout the injection protocol to minimise the effect of venous return from the inferior vena cava
4. Match saline infusion rate and contrast material infusion rate to reduce dead space phenomenon^a

^aA small proportion of the injected contrast medium is retained in the "dead space" between the brachial vein and the superior vena cava for about 20–30s before being pushed out during the venous phase of perfusion. With split-bolus protocols, the second phase is also subject to "dead space" phenomenon if the injection rate is decreased between phases. Using identical injection rates with multiphasic protocol can reduce dead space phenomenon and result in more uniform enhancement.

measure up to 10 mm [27], whereas the interventricular septum should be no more than 13 mm in thickness [8]. A relatively stiff pericardium encloses the ventricles, which in turn are in anatomic continuity sharing common muscle fibres and the septal wall. Hence, functional changes in one ventricle can affect those of the other. The septum is a crucial component in this ventricular interdependence [28]. Systolic interdependence is mediated mainly through the interventricular septum, whereas diastolic interdependence relies on the pericardium. Normal curvature of the interventricular septum is convex towards the RV. Abnormal septal positions can range from flattening to complete deviation convex towards the LV [29] (Figure 13).

If patients undergo retrospective ECG-gated cardiac MDCT, the dynamic data set can be used to assess ventricular function. Measurements of right ventricular volume and function provide both diagnostic and prognostic information. The functional data set is axially reconstructed by using a segmental reconstruction algorithm at $1.5\text{--}2\text{ mm}$ thickness $\times 1\text{ mm}$ increment

intervals at every 10% of the RR interval on a 512×512 matrix on a cardiac field of view using a soft-tissue convolution kernel. This yields 10 cardiac phases (ranging from 0% to 90%). Visual analysis of these data on cine mode provides information regarding myocardial kinetics. The same data can also be used for RV volumetric analysis by reformatting the data in the short-axis orientation, tilting the cut plane parallel to the plane of the mitral valve and parallel to the LV vertical long axis (Figure 14). Although the analysis can be performed with much thicker contiguous slices of up to 8 mm, as in the case of cardiac MRI, the thinner slice thickness facilitates increased accuracy of right ventricle endocardial and epicardial delineation and also allows for precise recognition of the valvular borders of the irregularly surfaced RV. The downside of using thinner sections is the increase in post-processing time, particularly when using manual contour definition.

The negative inotropic and chronotropic effects of beta-blockers may interfere with the true RV volumes. There are various semi-automated software analysis

Figure 12. (a) Horizontal four-chamber view. Line drawn perpendicular to the plane of the ventricular septum gives rise to the short axis view (b). In this instance, basal chambers are demonstrated in end diastole. LV, left ventricle. RV; right ventricle.

Figure 13. Interventricular septum (star) on axial CT in three different patients. (a) Normal septum is convex towards the right ventricle (RV). (b) Flat septum in a patient with moderate pulmonary hypertension. (c) Septum deviation convex towards the left ventricle (LV) in a patient with severe right ventricular dysfunction.

programs that permit calculation of ventricular volumes and ejection fractions. The basic steps involved are described in Table 3.

Multiple studies have shown that ECG-gated MDCT provides reliable assessment of global RV function compared with other imaging techniques. A recent multimodality study by Sugeng et al [34] for assessing RV volumes and function concluded that MRI was the most accurate; MDCT consistently overestimated volumes and three-dimensional (3D) echo underestimated volumes. The lower temporal resolution (effective acquisition time window per cardiac cycle) of MDCT than MRI can lead to overestimation of the right ventricular end-systolic volume, and subsequently to slight underestimation of the stroke volume and ejection fraction (EF). The interobserver variability for calculation of RVEF with MDCT is similar to that obtained with MRI and is about 5% [33].

Each technique has its own limitations but the main concern for MDCT is the radiation burden, which is around 7–15 mSv for a retrospectively gated study compared with 5 mSv for radionuclide ventriculography. Obviously both echocardiography and MRI are free of radiation, and hence more suited for serial follow-up. The use of MDCT for primary assessment of right ventricular function should be reserved for those who cannot undergo either of these techniques.

Clinical implications

Right ventricular function is an important prognostic marker in a variety of cardiopulmonary conditions. The following is a brief summary of some of those diseases where evaluation of right ventricular function is of paramount importance.

Figure 14. Retrospective electrocardiogram-gated multidetector row CT. Short axis basal view in 70% of the RR interval (a) and 30% of the RR interval (b). On cine mode, the complete data set encompassing all the diastolic and systolic images from the base to the apex of the ventricle is used to assess wall motion, contraction and thickening. Manual tracing of the endocardial (red) and epicardial (green) borders and propagation of this contouring across the phases will result in volumetric analysis as explained in Table 3. LV, left ventricle; RV, right ventricle.

Table 3. Right ventricular volumetric analysis [30–33]

1. Identify end-diastolic and end-systolic frames (images showing the largest and smallest endocavitary diameters respectively). Analysis of electrocardiogram tracing can also help in confirming the phases
2. Reconstruct axial and short axis images of the right ventricular (RV) cavity during the selected systolic and diastolic phases
3. Segment RV endocardial border from the cardiac base to the apex. The volume to be covered is defined by the border of the valve planes (atrioventricular and pulmonary valves), endocardium and interventricular septum to the outflow tract. Papillary muscles, the moderator band and RV trabeculations should be included in the lumen
4. The end-diastolic and end-systolic volumes are obtained by automated summation of the volumes of all short axis slices (Simpson method). RV stroke volume (RV end-diastolic volume–RV end-systolic volume) and RV ejection fraction (RVEF) (RV stroke volume/RV end-diastolic volume $\times 100\%$) are also automatically calculated. Because of the greater end-diastolic volume of the RV, RVEF is lower than left ventricular ejection fraction

Acute pulmonary embolism

The clinical impact of the embolism depends on the clot burden and underlying cardiorespiratory status. Obstruction within the pulmonary vasculature leads to an increase in the RV afterload. The resultant RV dysfunction can result in leftward septal bowing with compression on the LV and decrease in cardiac output.

RV/LV ratios can be used as a risk stratification and prognostication tool in the context of pulmonary embolism [35]. The maximum distance between the inner surface of the free wall and the endocardial surface of the interventricular septum is measured either on the four-chamber or axial view (Figure 15). A potential pitfall is the variability in the RV size on the four-chamber view. However, the four-chamber view measurements have been shown to correlate better with echocardiography measurements than those made with axial views [36]. This difference in the measurements is more obvious in patients with pulmonary embolism. Dogan et al [37] showed that although there was no significant difference between the RV/LV diameter ratios measured on axial views in patients with and without pulmonary embolism, there was a significant

difference in the ratios obtained with the four-chamber view. The main advantage of the axial view is its ease of use. It allows for a simpler and faster evaluation. The diameter of each ventricle should be measured at its largest point, as the widest points may be found at different levels for each ventricle [38]. The RV/LV diameter ratios measured on non-gated *vs* gated MDCT also show a high level of correlation [39].

To date, the RV/LV diameter ratio is the only ventricular CT measurement directly associated with mortality after pulmonary embolism [40]. The normal RV/LV diameter ratio is 1 or less. A score of 1–1.5 is indicative of modest RV dysfunction, whereas a ratio greater than 1.5 indicates severe dysfunction [36]. For patients with a previous negative CT examination, measuring the interval increase in the ratio improves specificity since the patient may have pre-existing RV enlargement before pulmonary embolism [41]. RV end-systolic volumes are higher in patients with pulmonary embolism [37].

The extent of thromboembolic load [42], the ratio of the main pulmonary artery (MPA) to ascending aorta diameter [43], tricuspid regurgitation as evidenced by the reflux of contrast medium into the inferior vena cava (IVC) [44] and dilatation of systemic veins [45] are other

Figure 15. Measurement of right ventricle (RV) dimensions in (a) axial and (b) reconstructed four-chamber CT in a 27-year-old woman who presented with stroke. Right-sided chambers are dilated with a sinus venosus type atrial septal defect (black arrow). The maximum distance between the ventricular endocardium and interventricular septum measured perpendicular to the long axis gives the RV/left ventricle (LV) ratio (red lines on axial view and blue lines on reconstructed four-chamber view). The widest point is typically in basal third of ventricle but the maximum diameter within the RV and LV may not always be at the same level.

Figure 16. Axial CT series in a 45-year-old female patient presenting with acute chest pain, breathlessness and syncope. (a) and (b) show dilated main pulmonary (star) with bilateral filling defects in proximal pulmonary vasculature (white arrows) in keeping with acute pulmonary emboli. The right lower lobe is completely occluded (block arrow). (c) Right ventricle (RV) dilatation with increased RV/left ventricle ratio (blue lines). Also note the paucity of vessels, particularly in the right lower lobe that is proximally occluded. (d) Reflux of contrast medium in the inferior vena cava and hepatic veins secondary to tricuspid regurgitation (arrows) and secondary to high right ventricular filling pressures.

parameters that help in evaluating the magnitude of pulmonary embolism on MDCT (Figure 16).

Pulmonary hypertension

Pulmonary hypertension (PHT) is defined as a mean pulmonary arterial pressure (mPAP) of greater than 25 mmHg at rest or 30 mmHg with exercise. The recently updated Dana point classification [46] describes the multifactorial causes of PHT. RV function is an irrefutable factor undermining the prognosis in PHT.

RV hypertrophy and dilatation result from increased afterload, but there is variability in the RV compensatory response in different individuals [47]. In PHT, the development of RV dysfunction and failure portends a downhill course with poor prognosis. Following pulmonary endarterectomy for chronic thromboembolic disease, the most important predictor of death is the presence of post-operative residual PHT [48].

CT is an integral part of the diagnostic work-up in patients with suspected PHT. It has the dual role of comprehensive assessment of the pulmonary vasculature and lung parenchyma as well as evaluation of the right heart. A correlative study in 1984 by Kuriyama et al [49] showed that an MPA of 29 mm or larger on CT is suggestive of PHT with a sensitivity of 69–87% and a specificity of 89–100% (Figure 17). Subsequent studies have given conflicting results. A retrospective study in 1998 by Tan et al [50] demonstrated that an MPA diameter of 29 mm or more on CT had a sensitivity of 87% and a specificity of 89% for predicting PHT. However, no linear correlation has been found between pulmonary artery diameter and the severity of PHT. Also, pulmonary artery dilatation can occur in the absence of PHT in patients with pulmonary fibrosis and is therefore an unreliable sign of PHT in this cohort [51]. Devaraj et al [52] showed that a composite index of the ratio of the diameter of the MPA and ascending aorta combined with echocardiography-derived right ventri-

(a)

(b)

Figure 17. Axial CT shows (a) dilated main pulmonary artery (MPA; blue line) compared with the adjacent ascending aorta (A) and (b) right ventricular dilatation and hypertrophy (white arrowheads) in a 25-year-old female with severe idiopathic pulmonary arterial hypertension. RA, right atrium; RV, right ventricle.

cular systolic pressure was a more significant predictor of PHT than either measure alone.

More relevant than the MPA diameter is the pulmonary compliance. Pulmonary arterial (PA) distensibility is defined by the change in cross-sectional area between diastole and systole. The distensibility is lower in patients with PHT and is a more accurate non-invasive marker of chronic PHT [53]. The relative area of change in the right pulmonary artery (RPA) can be used as a predictor of mortality [54]. An elegant ECG-gated MDCT study by Revel et al [55] demonstrated that RPA distensibility correlated strongly with mean PA pressures. RPA distensibility can be assessed on the largest and smallest RPA cross-sectional areas during diastole and systole. The measurement is made midway between its origin and the first segmental branch and should be perpendicular to the long axis on orthogonal planes (Figure 18). A more complex method involves systematic measurements during 10 phases. The authors found that the best PA distensibility cut-off values for distinguishing between patients with and without PHT was 16% with the standard approach and 13% with the simplistic approach. The same study also demonstrated the feasibility of measuring RPA distensibility, right ventricular outflow tract (RVOT) systolic shortening and diastolic myocardial thickness on ECG-gated MDCT. These parameters correlate with mPAP, and hence can be potentially used for risk stratification. In spite of these impressive developments in non-invasive imaging, right heart catheterisation remains the gold standard for evaluation of right heart haemodynamics, more so as large prospective clinical trials using non-invasive imaging markers are still lacking.

Cor pulmonale

Cor pulmonale is defined as RV enlargement or hypertrophy secondary to pulmonary disease in the absence of left ventricular dysfunction [56].

Chronic obstructive pulmonary disease (COPD) is the commonest cause of cor pulmonale. Approximately 90% of patients with severe COPD will have PHT [57]. Hur et al [58] demonstrated that the mean RV wall mass measured by MDCT correlates well with the severity of COPD.

Congenital heart disease

MDCT can exquisitely demonstrate the morphological abnormalities of congenital heart disease (CHD), and therefore plays an important complementary role to other imaging modalities. The multiplanar capabilities of MDCT can be adapted to display a particular target, and thus it is extremely valuable for pre-operative planning. Also, RV function is one of the most important predictors of survival and post-operative outcome in patients with CHD. It is beyond the scope of this paper to discuss the various CHDs. In some cases, the RV is the dominant or systemic ventricle (Figure 19). These patients develop progressive right ventricular dysfunction. Conversely, in patients with isolated atrial septal defect (ASD), the RV may tolerate volume overload for a long time without a significant decrease in right ventricular systolic function [59]. Closure of such defects in the presence of established pulmonary artery pressure results in progressive remodelling of the RV and may have adverse consequences.

Generally, in addition to exercise capacity, RV size and function and development of tricuspid regurgitation help to determine appropriate surgical timing. With improving treatments, more patients with CHD reach adulthood. Their follow-up requires serial quantification of ventricular function. Although this is best performed by techniques such as MRI and echo as they do not involve ionising radiation, the presence of pacemakers or difficult acoustic windows may make MDCT a feasible alternative. Volume measurement by gated MDCT

(a)

(b)

(c)

(d)

Figure 18. Right pulmonary artery (RPA) distensibility. Axial (a) and coronal (b) views are used to align the dashed lines perpendicular to the RPA (red and green lines). (c) Sagittal reconstruction in systole and diastole of RPA is thus obtained after correction for its double obliquity. Manual tracing of the RPA contour outline is performed to calculate the area. RPA distensibility is calculated from the formula: $SA-DA/SA$. DA, diastolic area; SA, systolic area.

correlates well with cardiac catheterisation in children and can reduce the necessity for invasive procedures [60]. Comparable quantifications of RV volumes and EF between MRI and MDCT have also been published in patients with tetralogy of Fallot and transposition of the great vessels [61].

Valvular heart disease

RV dysfunction can occur with both right- and left-sided valvular disease. Tricuspid regurgitation (TR) is often functional and occurs because of annular dilatation in the setting of RV dysfunction. This is usually secondary to left heart disease or PHT. Dreyfus et al [62] demonstrated that solely treating the primary valve lesion, in this case the mitral, will not directly correct tricuspid annular dilatation or improve RV function, but an additional procedure in the form of tricuspid valve (TV) annuloplasty at the time of mitral valve repair can improve patient outcomes. There is a paucity of data to prove that TR correction in the setting of non-valvular PHT alters the natural course of RV dilation and development of cor pulmonale. In patients with mitral stenosis, significant PH may be the cause of mortality in 60–70% [63]. Surgical correction is proven to decrease PHT and is associated with excellent long-term survival and functional results [64]. In patients with non-ischaemic mitral regurgitation, pre-operative RV performance can define subgroups with different post-operative survivals [65]. Subnormal RVEF

at rest with or without LV dysfunction is associated with >50% mortality risk within 2 years in patients with mitral regurgitation [66].

Ebstein's malformation is a rare, complex malformation of the TV leaflets with right heart dysfunction. It accounts for less than 1% of all congenital heart defects. The septal and posterior leaflets are displaced apically, causing atrialisation of the inlet of the RV while the trabecular and outlet portions form the functional RV. The dilatation of the true tricuspid annulus causes tricuspid insufficiency. This anomaly may be associated with ASD, patent foramen ovale and conduction system abnormalities that increase the risk of sudden death. On MDCT, the salient features are a markedly enlarged RA with dilatation of the RVOT (Figure 20). The pulmonary trunk is usually normal or small. The clinical course is variable and management is complex, depending on the extent of disease. MDCT can be useful in anatomical delineation of the leaflet size and location, annulus and atrial size, and in assessing right ventricular function [67].

RV infarction

The RV is relatively resistant to irreversible ischaemic injury [68]. The estimated incidence of RV infarction accompanying inferior myocardial infarction varies from 30% to 50% [69]. Isolated right ventricular infarction can occur as a result of disease in acute marginal arteries or a

Figure 19. Axial CT in a patient with congenitally corrected transposition of the great arteries (TGA) (levo-TGA). The heavily trabeculated right ventricle (black arrows) is the systemic ventricle (SV). LA, left atrium.

Figure 20. Axial CT in a 25-year-old male patient with Ebstein's anomaly. There is apical displacement of the malformed tricuspid valve leaflets (white arrow). Right-sided cardiac chambers are markedly dilated with atrialised and hypertrophied right ventricle (RV). RA, right atrium.

Figure 21. Axial CT image in a 68-year-old male patient with a large right coronary artery infarction complicated by a large ventricular septal defect (white arrows) between the inferior wall of left ventricle and right ventricle.

non-dominant right coronary artery but has also been described with normal coronary arteries in the setting of PHT and right ventricular hypertrophy [70].

Concomitant RV involvement in acute myocardial infarction (AMI) is associated with major complications and in-hospital mortality [71] (Figure 21). An RVEF of less than 40% has been proven to be a significant independent predictor of mortality after AMI [72]. RV function is also a predictor of development of heart failure in patients with left ventricular dysfunction after AMI [73].

Features of RV dysfunction include chamber dilatation and wall motion abnormalities. However, regional wall motion abnormalities can be present without myocardial infarction, and hence have a low specificity.

Heart failure

Chronic left heart failure is the most common cause of RV dysfunction. RVEF has been proven to be an independent predictor of survival in moderate to advanced heart failure compared with cardiopulmonary exercise testing [74]. Most modern scanners can complete image acquisition in less than 15 s, making it feasible to obtain reasonable quality images even if the patient is very breathless.

RV assist device

Ventricular assist devices act as a bridge to heart transplants. An important consequence of left ventricular assist device (LVAD) placement is RV failure with a high mortality rate. Pre-operative evaluation of TR and RV

(a)

(b)

Figure 22. 45-year-old male patient with severe biventricular failure. The topogram (a) and axial CT image show the components of the biventricular ventricular assist device within the ventricle (red arrows) and the vascular conduits (red stars). Also note the left pleural (white star) and pericardial (black star) effusions.

Figure 23. CT findings of arrhythmogenic right ventricular cardiomyopathies in a 35-year-old man. (a) Axial images show marked right heart dilatation and a scalloped appearance to the right ventricle (RV) free wall (white arrowheads). The patient was also in congestive heart failure, as evidenced by bilateral pleural effusions (white star). (b) Magnified view of the inferior wall of the RV to show fatty infiltration (white arrowheads). (c) Sagittal view demonstrates enlargement of right ventricular outflow tract (black arrows). Also note the presence of pacemaker leads (red arrow). RA, right atrium.

geometry may identify the cohort of patients who could benefit from simultaneous biventricular assist devices [75, 76] (Figure 22). Right ventricular assist device can also be used to treat severe RV failure associated with massive pulmonary embolism.

Right ventricular cardiomyopathy

RV arrhythmias notably affect the younger population and are a well-documented cause of sudden cardiac death. Of the intrinsic right ventricular cardiomyopathies, arrhythmogenic right ventricular dysplasia (ARVD) has characteristic morphological attributes that can be visualised on non-invasive imaging. ARVD is a rare genetic cardiomyopathy characterised by a gradual loss of RV myocytes and progressive replacement of RV musculature by fibrofatty tissue. This process results in ventricular arrhythmia, RV failure and sudden death. RV dilatation, increased trabeculation, RV intramyocardial fat deposition mainly in the infundibulum, inferior and antero-apical walls focal wall thinning, scalloping along the RV free wall and RVOT enlargement are the salient structural abnormalities described on MDCT [77] (Figure 23). Left ventricular involvement in ARVD is well documented [78]. Fatty tissue in the LV and in the subendocardial aspect of the ventricular septum can be seen on MDCT [79]. Currently MRI is the most commonly used non-invasive imaging modality for the diagnosis of ARVD, but there are numerous case studies that have illustrated the potential use of MDCT given its improved temporal resolution and good fat discrimination. The presence of the latter finding on its own, however, has to be taken with caution as myocardial fat on MDCT is seen in conditions other than ARVD, such as ageing, prior myocardial infarction and chronic ischaemia.

Conclusion

RV function plays an integral role in the pathogenesis and outcome of many cardiovascular diseases. Imaging

the RV has long been a challenge owing to its complex geometry, but advances in echocardiography and MRI have improved our understanding of this pivotal structure. In recent years, there has been tremendous expansion in MDCT and its cardiac applications. As shown in this article, it is possible to make a comprehensive assessment of the right heart using CT. Further studies will hopefully continue to define and refine its role in this population. The main limitation of CT is the radiation constraint that is continually being addressed by the industry and clinical community.

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