UPDATE IN RADIOLOGY

Study of the right ventricle using magnetic resonance imaging

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Magnetic resonance imaging; Heart; Right ventricle; Right heart; Cardiac imaging

Abstract Magnetic resonance (MR) imaging has proven efficacy in the study of the heart. Its clinical applications are directed primarily at the study of the left ventricle, and the right ventricle is relegated to the background. This article reviews the anatomy and physiology of the right ventricle, as well as the manifestations of most common diseases affecting this chamber of the heart: infarction, cardiomyopathy, masses, and right heart failure. Knowing the distinctive features of the right ventricle with respect to the left and the particularities of the MR imaging protocol results in better technical performance in cases in which the reason for the examination or imaging findings point to the right ventricle. The importance of the right ventricle in the management of cardiopulmonary disease is growing and MR imaging can provide clinicians with the support they need.

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PALABRAS CLAVE
Resonancia magnética; Corazón; Ventrículo derecho; Corazón derecho; Imagen cardíaca

Estudio del ventrículo derecho mediante resonancia magnética

Resumen La resonancia magnética (RM) es una técnica de probada eficacia en el estudio del corazón. Sus aplicaciones clínicas se dirigen preferentemente al estudio del ventrículo izquierdo, quedando el ventrículo derecho relegado a un segundo plano. Este artículo ofrece una revisión de la anatomía y fisiología del ventrículo derecho, así como de las manifestaciones de la afección más frecuente en esta cámara cardíaca: infarto, miocardiopatías, masas y fallo cardíaco derecho. El conocimiento de los rasgos diferenciales del ventrículo derecho con respecto al izquierdo y de las particularidades del protocolo de estudio mediante RM, consigue un mayor rendimiento de la técnica en aquellos casos en que el motivo de petición o los hallazgos de imagen apuntan al ventrículo derecho. La RM reúne características para apoyar desde la imagen el protagonismo creciente que los clínicos están otorgando al ventrículo derecho en el manejo de las enfermedades cardiopulmonares.

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Traditionally, the right ventricle (RV) has received less attention than the left ventricle (LV), because of the little consideration given to its function. In addition, for a long time, chest radiography and angiography were the only available techniques for assessing the RV; however, these techniques have their limitations and angiography is associated with considerable risk.\(^1\) Echocardiography soon became a first-line technique because of its availability, safety, cost and diagnostic accuracy; nonetheless, this technique is of limited use for evaluating the RV because of the retrosternal location of this ventricle and the method used to calculate its volumes. Computed tomography (CT) is the non-invasive technique of choice for the study of pulmonary and coronary vascularization, but its use in the evaluation of the heart chambers remains limited, and its associated risks should not be overlooked (ionizing radiation, ionized contrast agents). Although the use of magnetic resonance (MR) is well established in other areas, we had to wait for the technological development of coils, sequences and processing software to confidently approach the study of the heart. The spatial and temporal resolution, multiplanar capability, tissue contrast, and safety make MRI a suitable non-invasive technique for examining the anatomy and function of the RV. Nonetheless, there are limitations inherent to the technique, such as long imaging times, the fact that it requires the collaboration of the patient, synchronization issues in cases of arrhythmia, and general contraindications, not to mention its cost and the scarce availability and experience in the use of this technique (Table 1).

### Characteristics and study of the right ventricle using magnetic resonance imaging

The RV is the most anteriorly situated cardiac chamber and makes little contribution to the cardiac silhouette on chest radiographs. It has a complex shape, showing no axis of symmetry,\(^2\) and is more difficult to model than the LV. From the axial view, the RV has a triangular shape and accommodates and wraps partially around the LV. In diastole, the RV receives the systemic venous blood and pumps it to the pulmonary circulation in systole, so RV and pulmonary circulation operate as a functional unit.\(^3\) It works as a volume pump, with its two primary functions being: to maintain a low systemic venous pressure and an adequate pulmonary perfusion. The architecture, pathophysiology, metabolism and coronary flow of both ventricles are different (Table 2), and these differences must be considered when performing and evaluating cardiac imaging studies.

### Table 1 Imaging techniques for the study of the RV.

<table>
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ARVD: arrhythmogenic right ventricular dysplasia; PHT: pulmonary hypertension; RV: right ventricle.

### Table 2 Specific differences of the RV compared with LV.

<table>
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<th>Anatomy and morphology</th>
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<td>Pulmonary resistance = 1/10 systemic</td>
<td>More compliant</td>
<td>Better adaptation to volume overload</td>
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RV: right ventricle; LV: left ventricle.
Magnetic resonance imaging technique

The RV can be assessed using a standard protocol: morphologic evaluation (black blood imaging: double inversion recovery or inversion recovery [IR], Half Fourier Acquisition Single Shot Turbo Spin Echo [HASTE], short time inversion recovery [STIR]), cine imaging (steady state free precession, SSFP), and myocardial enhancement (contrast enhanced IR sequences with suppression of myocardial signal).

The “basic” planes to study the RV using MR imaging are four-chamber (4C) and short axis (SA), which are routinely obtained in conventional cardiac MRI protocols. In case of clinical suspicion of RV involvement, it is recommended to have specific planes (Fig. 1): right two-chamber (2C), right three-chamber (3C or RV inflow-outflow view) and RV outflow tract (RVOT) views. Axial images (double IR and cine) with lower field of view provide higher spatial resolution of the free RV wall, which can be useful in arrhythmogenic right ventricular dysplasia (ARVD).

Depending on the findings or the clinical suspicion, additional techniques can be used in specific cases. Pulmonary MR angiography requires high temporal resolution to separate arterial and venous phases, and is useful for assessment of pulmonary arteries (pulmonary hypertension) and veins (abnormal venous return leading to shunt). Phase-contrast imaging can help to quantify valve disease and shunts. This requires an accurate technique, with the imaging section perfectly oriented perpendicular to the vessel, an appropriate selection of velocity encoding, and appropriate magnet isocenter location. Yet, there is a margin of error due to limitations inherent to the technique.4 Cardiac perfusion MR imaging requires high temporal resolution, compromising spatial resolution and anatomic coverage. Although this technique was developed for detecting myocardial ischemia, it is useful in the diagnosis of intracardiac shunts and cardiac masses due to the intracavitary contrast generated. FLIRT (Fixed Long Time Inversion Recovery) sequence obtained immediately after contrast administration may be useful in the detection of thrombi and masses.

Anatomy of the right ventricle

The RV is divided into three regions5,6: the inflow tract or inlet, the outflow tract or outlet, and the apical portion or body (Fig. 2). The inflow tract or inlet includes the tricuspid valve apparatus, which extends from the tricuspid annulus traversing the chordae tendineae, to the insertion of the papillary muscles. The outflow tract or outlet extends from the supraventricular crest to the pulmonary valve. It includes the infundibulum or conus arteriosus, a narrowing in the upper part of the RV, below the pulmonary valve, characterized by a smooth endocardial surface. The inflow and outflow tracts are arranged in a “V” shape whose apex corresponds to the trabeculated apical myocardium. The RV can also be divided into free or retrosternal wall (divided into anterior and lateral), inferior or diaphragmatic wall and interventricular septum.

A number of characteristic bands, trabeculae and muscles are present in the endocardial surface of the RV (Fig. 3)
that differentiate it from the LV.\textsuperscript{5,6} The tricuspid leaflets are inserted on the papillary muscles, which are numerous and small and tend to be attached to the septum, unlike the LV. The supraventricular crest (Wolff’s spur) is a muscular thickening that separates the pulmonary and the tricuspid valves (in the LV, the valves are in continuity). The moderator band contains fibers of the electrical conduction system, and connects the septum with the apical portion of the anterior wall, separating the outflow tract from the apical myocardium. It is consistently found in the LV 3C and 5A views. The septomarginal or septal trabecula is a muscular thickening in the interventricular septum in the shape of a “Y”, with one of its branches being the moderator band. This trabecula is visible on MR images only in cases of RV hypertrophy. The apical trabeculae are fleshy trabeculae that form a septum within the apex.

The normal RV diameter is smaller than that of the LV. The RV makes no contribution to the cardiac apex and has a thin wall (≤5 mm in the inferior aspect).\textsuperscript{7,8} The interventricular septum must be convex toward the RV throughout the cardiac cycle. Variations in RV morphology may cause diagnostic problems. The angulation of the long axes also influences the RV shape.\textsuperscript{9} The \textit{pectus excavatum} is a deformity of the free RV wall.

In a right-dominant system (80% of the population), the perfusion of the RV relies mostly on the right coronary artery,\textsuperscript{6} which supplies the lateral wall (acute marginal branches) and the inferior wall and septum (posterior descending artery). The anterior wall and septum are supplied by the left coronary artery (anterior descending artery), and the infundibulum is supplied by the conal branch. In a left-dominant system, the posterior descending artery arises

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**Figure 2** Regional anatomy of the right ventricle: thin MIP enhanced 3D MR-angiography of right cavities, with ventriculogram effect, demonstrates the inflow tract (IT), outflow tract (OT) and apical myocardium (AM) (RA: right atrium; PA: pulmonary artery; SVC: supraventricular crest; RV: right ventricle; TV: tricuspid valve).

**Figure 3** Characteristic structures of the right ventricle: supraventricular crest (SVC), septomarginal trabecula (SMT), moderator band (MB), anterior papillary muscle (APM) (RV: right ventricle; LV: left ventricle).
from the left coronary artery, and the RV is supplied equally by the left and the right coronary artery. Coronary supply to the RV is more favorable than to the LV because it occurs in both systole and diastole and because the RV has a more extensive collateral system.

**Physiology of the right ventricle**

The RV is connected to the LV, and both have to pump the same stroke volume. Ventricular interdependence refers to the mutual dependence of their functions, having an anatomical substrate: both ventricles share myocardial fibers (particularly in the interventricular septum) and the same pericardial space.

RV contraction starts in the inflow tract, progresses toward the apical myocardium, and finishes in the infundibulum in a peristalsis-like fashion. Contraction is complex and involves three mechanisms: inward movement of the free wall, traction at the points of attachment in the LV and movement of the tricuspid annulus toward the apex secondary to contraction of the longitudinal fibers. This is one of the main components of the RV ejection and can be quantitatively measured as tricuspid annular plane systolic excursion (TAPSE) whose normal value should be >2 cm.

MR imaging is considered the reference standard for the evaluation of the RV volume and systolic function. The limitations of echocardiography are due to the retrosternal location of the RV, the poor definition of the endocardial surface and the difficulty of applying volumetric models that allow the use of a geometric equation to calculate the RV volume.

MR imaging allows quantitative assessment of the RV using a series of consecutive sections of cine images that include the entire RV. This is usually performed in the 4C view, using the sequences also for the quantitative assessment of the LV. However, measurement is not without problems, including the difficult identification of the valve plane due to its poor visualization and its motion during heartbeat. To accurately determine the last image of the atrium and the first image of the RV, scout lines from cine sequences of the short axis in right 4C and 2C views can be obtained so the valve plane can be identified in systole and diastole. Another solution is to delineate the contours on 4C images, but this requires the acquisition of sequences that are not routinely used. Care must be taken when including the entire outflow tract.

Quantitative assessment of the RV allows the determination of the end-diastolic (EDV) and end-systolic volume (ESV)—with absolute and normalized values—stroke volume, cardiac output and ejection fraction (EF). The RV volume is 10–20% higher than the LV volume, so for a stroke volume equal to that of the LV, the RV should have lower EF. Increased EDV is associated with dilatation, and decreased EF with systolic dysfunction. Although the RV mass can be quantified, this is not routinely done because of the difficulty of drawing the epicardial and endocardial contours, as the wall is very thin. The RV myocardial mass is much lower than that of the LV and increased RV mass indicates hypertrophy.

RV diastolic function has not been as well studied as the LV function, and MR imaging can be used for its evaluation in a similar way to echocardiography, by analyzing the flow patterns through the tricuspid valve, pulmonary veins and inferior vena cava.

**Pathology of the right ventricle**

We will consider four groups: right heart failure, ischemia, myocardial diseases and masses. Congenital heart disease will not be included here.

**Right heart failure**

The most common cause of right ventricular failure is left ventricular failure. Primary RV failure may be secondary to direct injury (ischemia, myocardial infarction) or to pressure (increased afterload) or volume overload (increased preload). This leads to dysfunction, and the RV starts a number of adaptation mechanisms such as hypertrophy, dilatation, septal displacement and tricuspid insufficiency. When these mechanisms are exceeded and the RV is unable to maintain its function, RV failure occurs, resulting in peripheral venous congestion, edema, ascites, and ultimately cardiogenic shock. Because of the ventricular interdependence, right heart failure eventually leads to left failure, and vice versa. In cases of RV overload and cardiopathies with right heart failure, MR imaging is an excellent technique to evaluate the progression and the treatment response and to determine the outcome in a non-invasive manner.

Pressure overload or pulmonary hypertension (PHT) is characterized by elevated blood pressure of the pulmonary circulation either primary (idiopathic, or associated with scleroderma, HIV infection and other conditions), or secondary (chronic airflow obstruction, chronic pulmonary thromboembolism). PHT is present when the average pressure in the pulmonary artery (PAP) is higher than 25 mmHg. PAP is determined by right heart catheterization, but it can be estimated by Doppler echocardiographic assessment of tricuspid insufficiency. Although MR imaging cannot establish the diagnosis, it plays an important role in PHT as it provides structural and functional information of the right ventricle-pulmonary circulation unit. The availability of medical therapies that on occasions improve the poor prognosis associated with PHT has heightened the interest in finding markers for monitoring disease course.

Cardiac manifestations of PHT on MR imaging are a reflection of RV pressure overload, (Fig. 4) including RV hypertrophy, dilatation, and systolic dysfunction, as well as retrograde dilatation of the right atrium, inferior vena cava and hepatic veins. MR imaging also demonstrates abnormal septal motion, where the interventricular septum flattens and even becomes convex toward the LV during systole (also during diastole as the disease progresses), affecting the function of the LV. Different parameters such as mass, EDV, ESV, stroke volume and right ventricular EF have been evaluated, showing variable correlations with PAP. Although this correlation is insufficient to establish a diagnosis of PHT using MR imaging, it may be useful in monitoring treatment response. Foci of contrast enhancement can be seen at the right ventricular insertion points associated to fibrosis, probably due to shear stress.
MR angiography demonstrates dilatation of the pulmonary artery, reduction in peripheral vessel, focal ectasia and arterial tortuosity; however, CT is more efficient. Phase-contrast MRI of the pulmonary arterial flow provides hemodynamic data on mean and peak velocities, flow and stroke volume. In certain cases, MRI may help in the etiologic diagnosis of PHT, by identifying the clinical signs of chronic pulmonary thromboembolism (occlusion of vessels, intraluminal thrombi and well-defined filling defects), or help in the differential diagnosis with other causes of right heart failure.

Volume overload occurs in valve insufficiency (pulmonary and tricuspid), and in left-to-right shunts, such as interatrial communication (IAC) and anomalous pulmonary venous drainage (APVD). The consequences are similar to those found in pressure overload, but volume overload is better tolerated by the RV and dilatation predominates over hypertrophy. In addition to identify features of RV overload (dilatation, abnormal septal motion), MRI is particularly useful in the identification of underlying conditions that might have been overlooked, such as cardiac shunts (Fig. 5). Despite having technical limitations, MRI provides quantitative assessment of shunt lesions, with the use of either the volumetric method (difference between right and left ventricular stroke volumes, useful in the absence of valve insufficiency) or the Qp/Qs ratio by quantifying the pulmonary (Qp) and systemic (Qs) flow with phase-contrast sequences.

Carcinoid heart disease occurs in 20% of patients with carcinoid syndrome. It is a paraneoplastic effect caused by the release of vasoactive substances secreted by hepatic metastases that directly reach the right heart before being inactivated, resulting in valvular thickening and retraction. In most cases, there is severe tricuspid regurgitation. Pulmonary stenosis and insufficiency are also common. Severe volume overload occurs and, to a lesser extent, pressure overload, with right heart failure being the cause of death in one-third of patients. MRI is an excellent technique to depict the features of this condition (Fig. 6).

**Ischemia (infarction)**

The RV is relatively resistant to infarction due to its decreased strength of contraction and to a more favorable
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Figure 5  Right ventricular (RV) overload secondary to ostium secundum-type interatrial communication: steady state free precession (SSFP) cine MRI in the four-chamber (4C) view (left), perfusion 4C (center) and gradient echo 4C with saturation band over the left chamber (right). 40-year-old man with right heart dilatation of unknown etiology in echocardiography. The atrial septal defect, barely visible on conventional cine MRI (SSFP), is clearly identified (arrows) on myocardial perfusion MRI as a hypointense jet (due to the different signal between right and left chambers) and on the gradient echo image performed with saturation band over the left chambers (due to blood signal suppression). In addition, MRI shows signs of RV volume overload (dilatation with no hypertrophy), with a pulmonary flow/arterial flow ratio (Qp/Qs) of 1.6, indicative of left-to-right shunt (see also video 2 in supplementary material available online).

Cardiomyopathies

All cardiomyopathies, except ARVD, affect primarily and especially the LV; however, in a variable proportion of patients there is concomitant RV involvement. MRI plays an important role in the detection of RV involvement, which may not be suspected using other imaging techniques or diagnostic methods, having important prognostic and therapeutic implications.

ARVD, also known as arrhythmogenic RV cardiomyopathy, is a genetic cardiomyopathy characterized by fibrofatty replacement of the myocardium with loss of myocytes. It affects the RV, at the so-called “triangle of dysplasia” (apex, anterior infundibulum and inferior wall of the RV); however, in most severe cases, there is also LV involvement. Manifestations vary widely; initially the disease is asymptomatic, with risk of sudden death, and subsequently it causes arrhythmias and RV morphologic changes, with progression to biventricular heart failure in the late stage (Fig. 8). A particular emphasis has been placed on its diagnosis because this condition is associated with sudden death in apparently healthy young people, and because it is a potentially treatable condition.

Diagnosis of this condition is based on the presence of structural, functional and electrophysiological changes resulting from the histological changes. Although biopsy provides the definitive diagnosis, the criteria proposed in 1994 by the Task force of the Working Group Myocardial and Pericardial Disease of the European Society of Cardiology and of the Scientific Council on Cardiomyopathies of the International Society and Federation of Cardiology33 are commonly used in clinical practice. These criteria were revised in 2002 and 201012 and were grouped into the following five categories: structural abnormalities or dysfunction, wall tissue characterization, ECG changes, arrhythmias and family history. MRI is the technique of choice, but it can only identify abnormalities from the first category: regional or global contractility changes, RV dilatation and systolic dysfunction. A normal MRI cannot rule out ARVD, particularly in early stages.

Two variants of regional contractility changes can be considered, both more conspicuous in systole. Focal aneurysm (bulging) is a free wall deformity that can be difficult to differentiate from variations in the normal contraction pattern, especially those occurring adjacent to the insertion site of the moderator band in the free wall.10 This sign should therefore be interpreted cautiously. The “accordion sign” is the corrugated pattern of the free RV wall of the outflow tract, more frequently seen in carriers of the mutations associated with ARVD.34

Additionally, the following characteristic features have been identified1-39: fibrosis and/or fibrofatty wall oxygen supply than the LV.36 30–50% of infarctions in the LV inferior wall are associated with RV infarction,27 but isolated RV infarction is uncommon (only in cases of nondominant right coronary artery23). Prior to the widespread use of MRI, the diagnosis of RV infarction was achieved in the clinical setting of a patient with myocardial infarction of the LV inferior wall, using characteristic clinical and electrocardiographic findings, but some patients remained undiagnosed.

Early diagnosis in the acute phase is crucial28 because RV infarction is associated with higher in-hospital mortality than isolated LV infarction.29,29 Although most cases are clinically silent, RV infarction may cause arrhythmias and hemodynamic anomalies such as hypotension, requiring volume repletion. After recovery from the acute episode, long-term prognosis is favorable, since the RV easily recovers its function after infarction, maintaining its viability regardless of the state of the coronary artery that supplies it.29 MRI is the technique of choice for diagnosis of RV infarction,30 through the identification of delayed enhancement of the RV wall and segmental contraction abnormalities (Fig. 7).
infiltration, hypertrabeculation, wall hypertrophy and outflow tract dilatation. Although initially the identification of fibrofatty infiltration of the free RV wall played an important role in the diagnosis of ARVD, this finding is currently under question given its low sensitivity (due to motion artifacts and low spatial resolution) and specificity (it has been described in healthy individuals)\(^3\). The detection of fibrosis is based on myocardial enhancement, with a non-segmental patchy or diffuse pattern, representing a more specific sign, present in more than 50% of patients that meet the ARVD criteria.\(^3\)

The Brugada’s syndrome, or RVOT tachycardia, also involves RV arrhythmias. The diagnosis is based on ECG findings, but RV abnormalities have been described, and differentiation with ARVD may be difficult.\(^3\)

Hypertrophic cardiomyopathy (HCM) is a genetic cardiomyopathy characterized by sarcomere dysfunction that results in increased ventricular mass. MRI is used to estimate the myocardial thickness, quantify the LV mass, demonstrate intramyocardial contrast uptake and study LVOT obstruction.

Although the American Heart Association considers that HCM is a condition limited to the LV, evidence suggests that HCM may also involve the RV, although to a lesser extent, but this finding has been little studied.\(^37,38\) Up to 40% of patients with HCM may have RV wall thickening,\(^39\) but myocardial enhancement is uncommon.\(^39\) Generally, RV involvement correlates with LV involvement, but cases with predominant RV involvement have also been described.\(^38\) In some cases, HCM causes RVOT obstruction\(^40\) or predominantly involves the cardiac bands.

On the other hand, RV hypertrophy can also occur in athletes,\(^41,42\) where thickening of the moderator band is characteristic.

Noncompaction cardiomyopathy is a lack of compaction of the inner myocardial layer due to a failure during

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**Figure 6** Cardiac involvement in carcinoid syndrome: steady state in free precession (SSFP) cine MRI in two-chamber (upper left), four-chamber view (upper right), outflow tract of the right ventricle (lower left) and longitudinal plane through the pulmonary valve (lower row, center), and coronal True-FISP sequence with thoracic-abdominal coverage (lower right). 45-year-old women with carcinoid syndrome secondary to liver metastases from a carcinoid tumor of the ileum. MRI shows dilatation of right chambers (especially of the atrium) caused by RV overload, tricuspid insufficiency (arrow) and pulmonary valve stenosis (see also video 3 in supplementary material available online).
Figure 7 Right ventricle (RV) infarction: delayed myocardial enhancement sequences (inversion recovery sequence after contrast administration) in the 4-chamber view (upper left) and basal short-axis (SA) view (upper right), and steady state in free precession cine images in the SA view: in diastole (lower left) and systole (lower right). 65-year-old man with a history of inferior left ventricular (LV) infarction 15 years ago, who underwent an MRI examination to investigate the cause of RV dilatation observed on echocardiography. MRI shows non-viable myocardium after infarction of inferior LV wall, associated with infarction of the inferior and lateral RV wall, with dyskinesia (short arrows) and well-defined myocardial enhancement (long arrows). Quantitative assessment revealed biventricular systolic dysfunction (see also video 4 in supplementary material available online).

These cases are difficult to diagnose given the trabeculated nature of the RV. The MRI diagnosis is based on the noncompacted/compacted myocardium ratio. It can be associated with ventricular dysfunction and trabecular delayed enhancement.

In cardiac amyloidosis, there are amyloid deposits in the four cardiac chambers and valves that result in restrictive cardiomyopathy. MRI could help diagnose amyloidosis by demonstrating wall thickening and especially myocardial embryogenesis. This is a recently described condition, whose identification has been facilitated by the routine use of MRI. The clinical course and expression of this disease is variable, but there is some controversy on the diagnostic criteria and prognosis.

Noncompaction has been recognized as a cause of RV failure, arrhythmias and embolism. It primarily affects the LV, but biventricular involvement and even cases of isolated RV involvement have also been described. These cases are difficult to diagnose given the trabeculated nature of the RV. The MRI diagnosis is based on the noncompacted/compacted myocardium ratio. It can be associated with ventricular dysfunction and trabecular delayed enhancement.

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enhancement characterized by an intra- or subendocardial pattern. Adjustment of the inversion time of IR sequences is complicated. LV findings are more conspicuous, but RV involvement is frequent.

In idiopathic dilated cardiomyopathy, RV dilatation has been identified as a poor prognostic factor, irrespective of the EF of the LV. In addition, a decrease in the EF of the RV in the setting of biventricular dilatation with LV systolic dysfunction is suggestive of idiopathic dilated cardiomyopathy rather than ischemic cardiomyopathy. Cardiac involvement in patients with systemic sarcoidosis is uncommon. It is characterized by the presence of myocardial granulomas, which can be biventricular.

Neoplastic and non-neoplastic masses

MRI is the technique of choice in evaluating cardiac masses because of its superior tissue resolution and multiplanar capabilities. Most cardiac neoplasms are benign. Myxoma is the most common heart tumor, and although it usually arises from the atria, RV involvement occasionally occurs. The tumor is attached to the cardiac wall by a pedicle and usually has a heterogeneous appearance. Papillary fibroelastomas are the most common tumors of the cardiac valves. They appear as a small, vascularized structure that follows the valve movement. These characteristics make MR imaging difficult, even if the tumor has been readily identified by echocardiography (Fig. 10).

Malignant tumors are uncommon, but malignancy rates in right chambers are higher than in left ones. Therefore, right-sided tumors are a sign of poor prognosis. Findings suggestive of malignancy are an aggressive growth pattern that infiltrates adjacent structures, pleural or pericardial effusion, involvement of more than one chamber, an ill-defined appearance, and broad-based attachment or non-septal location (Fig. 11).

Most malignant tumors of the heart are secondary and metastatic spread can be by direct invasion, intravascular spread (through the inferior vena cava), or
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Figure 9  Noncompaction of myocardium with biventricular involvement: steady state in free precession (SSFP) cine MRI in 4-chamber (left) and short axis view at the middle ventricular (upper right) and apical (lower right) level. Biventricular hypertrabeculation with apical predominance. 24-Year-old man with a family history of noncompaction of myocardium and mild LV systolic dysfunction.

Figure 10  Papillary fibroelastoma arising from the tricuspid valve: steady state in free precession (SSFP) cine MRI in the right 2-chamber (2C) view: in diastole (upper left) and systole (upper right), perfusion study in right 2C view (lower left) and delayed myocardial enhancement (inversion recovery sequence after contrast administration) (lower right). Incidental finding on echocardiography. Cine MR images demonstrate with some difficulty a small ill-defined mass on the tricuspid valve that is alternately seen in the right atrium or right ventricle, depending on the cardiac phase (arrows). On the perfusion image is clearly seen as a filling defect (arrow). The lesion shows intense contrast enhancement (arrow) (see also video 6 in supplementary material available online).
Figure 11  Cardiac metastasis in hypernephroma: short time inversion recovery (STIR) 4-chamber (4C) view (upper left), steady state in free precession (SSFP) cine image right 2-chamber (2C) view (lower left), pre- (upper row center) and postcontrast (lower row center) T1-spin-echo 4C sequences, and post-contrast fixed long inversion recovery technique (FLIRT) in 4C (upper right) and 2CD (lower right). Large RV mass with septal and free wall infiltration with heterogeneous appearance and heterogeneous enhancement (see also video 7 in supplementary material available online).

Figure 12  Organized thrombus in the (RV): steady state in free precession (SSFP) cine image in 4-chamber view (left), fixed long inversion recovery technique (FLIRT) obtained immediately after contrast administration (center) and delayed myocardial enhancement (inversion recovery sequence after contrast administration) (right). 35-Year-old patient with Behçet syndrome and a history of RV thrombectomy. MR image shows a mass attached to the apical septum of the RV, with delayed peripheral enhancement. Postoperative changes in the RV apex.

hematologic or lymphatic spread. Cardiac lymphomas are usually large masses with a tendency to involve the right chambers and are a manifestation of disseminated primary lymphoma.

On MRI, intracardiac thrombi appear as masses partially or entirely attached to the endocardial surface. The detection of thrombi is important to avoid embolic events and because of the therapeutic implications, as it requires anticoagulation therapy. Early (FLIRT) and (postcontrast IR) delayed myocardial enhancement sequences are useful for diagnosis, but in some cases detection on the ventricular wall may be difficult. Differentiation from cardiac
neoplasms may be complicated, but neoplasms are characterized by contrast uptake. Nonetheless, rarely, large chronic thrombi may enhance and can be diagnostically challenging (Fig. 12). In the RV, the presence of thrombi is usually associated with systemic hypercoagulable states such as the antiphospholipid syndrome, thrombogenic vasculitis (Behçet), ulcerative colitis and neoplasms. It has been postulated that the presence of inflammation, granulation tissue or even endomyocardial fibrosis has a role in the development of intracardiac thrombi in some patients. In contrast with the LV, thrombus formation associated with areas of stasis is uncommon, given the low incidence of aneurysms after infarction.

Conclusion
Indications for the study of the RV using MRI can be grouped in four categories:

(1) Study of RV arrhythmias and ARVD screening (asymptomatic patient or screening of relatives). MRI alone cannot establish or rule out a diagnosis of ARVD, but it is part of the diagnostic criteria.

(2) Study of dilated RV. Echocardiography establishes the diagnosis and MRI is reserved for inconclusive cases. APVD, which is not usually assessed by echocardiography, is an indication for MRI. Some cases of IAC or RV infarction are identified as cause of RV dilatation on MRI.

(3) Study of the RV function. MRI is the modality of choice for quantification of ventricular volumes, and in many cardiopathies the RV function is an important prognostic factor.

(4) Study of RV masses or inconclusive echocardiographic images.

Finally, in other cases MRI can detect an unsuspected RV abnormality, in the setting of a cardiopathy primarily involving the LV.

MRI is shaping up as the reference standard technique for the study of the RV due to its unquestionable advantages and despite its limitations in terms of availability and long examination and study times. Echocardiography remains a first-line modality and right cardiac catheterization provides functional and hemodynamic information that in some cases is essential (diagnosis of PHT). In future years, it is likely that MRI will help us gain deeper insights into RV abnormalities, assuming a more important role in the diagnosis of these patients.

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Conflict of interest
The authors declare not having any conflict of interest.

Appendix A. Supplementary data
Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j. earrxeng.2012.07.004.

References

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