Journal of Cardiovascular Magnetic Resonance (2005) **7,** 649–655

ISSN: 1097-6647 print / 1532-429X online
DOI: 10.1081/JCMR-200065608

Taylor & Francis
Taylor & Francis Group

DELAYED CONTRAST ENHANCEMENT

Late enhancement: a new feature in MRI of arrhythmogenic right ventricular cardiomyopathy?

PETER HUNOLD, M.D.,[1,*] HEINRICH WIENEKE,[2] OLIVER BRUDER,[3] ULRICH KRUEGER,[4] THOMAS SCHLOSSER,[1] RAIMUND ERBEL,[2] and JÖRG BARKHAUSEN[1]

[1]*Department of Diagnostic and Interventional Radiology and Neuroradiology, University Hospital Essen, Essen, Germany*
[2]*Department of Cardiology, West German Heart Center, University Hospital Essen, Essen, Germany*
[3]*Department of Cardiology, Elisabeth Hospital, Essen, Germany*
[4]*Department of Cardiology and Angiology, Herzzentrum Duisburg, Duisburg, Germany*

Aim of the study was to evaluate whether late enhancement (LE) in contrast-enhanced MRI can be used to characterize fibrofatty myocardial replacement in patients with arrhythmogenic right ventricular cardiomyopathy/dysplasia (ARVC). Fifteen patients with suspected ARVC underwent CE-MRI using a 1.5 T scanner. Long and short axis SSFP cine images and T1-weighted fast spin echo images were collected in all patients. After injection of 0.2 mmol/kg Gd-DTPA (Magnevist, Schering, Berlin, Germany), inversion recovery gradient echo images were acquired in long and contiguous short axes to detect myocardial LE indicating areas of fibrous tissue within the myocardium. For definition of ARVC, the ESC Task force criteria were used. In 7 (47%) of 15 patients, ARVC was diagnosed based on the ESC criteria. In all of these 7 patients, MRI showed morphologic or functional criteria of ARVC according to the ESC. LE of the right ventricular myocardium was detected in 5 (71%) of the 7 ARVC patients, additional LE of the left ventricular myocardium in 2 of these patients. None of the 7 patients meeting the ARVC diagnostic criteria had fatty RV infiltration demonstrable by conventional T1-weighted imaging. Eight patients neither showed morphologic criteria of ARVC nor LE. In conclusion, late enhancement can be detected in the right and left ventricular myocardium in some ARVC patients. LE might represent intramyocardial areas of fibrous tissue.

Key Words: Arrhythmogenic right ventricular cardiomyopathy; Magnetic resonance imaging; Late enhancement; Myocardium; Cardiomyopathy; Contrast agents

1. Introduction

Arrhythmogenic right ventricular cardiomyopathy/dysplasia (ARVC) is frequently asymptomatic but may be clinically characterized by episodes of ventricular tachycardia. Histologically, total or partial replacement of the right ventricular myocardium by fibrous or fatty tissue is found. ARVC is a rare (prevalence 0.2–0.8%) but a clinically important disease (1, 2) which accounts for approximately 20% of sudden cardiac deaths in individuals under the age of 30 and is responsible for about 4% of deaths associated with physical activity in young athletes (2, 3). The replacement of myocardium by adipose and/or fibrous tissue leads to regional or global dysfunction of the right ventricle (RV). Severe dilatation of the RV, the reduction of right ventricular ejection fraction with no or only mild impairment of the left ventricle and localized RV aneurysms are major morphologic criteria for the diagnosis of ARVC as defined by the European Society of Cardiology Task force in 1994 (4) and the consensus report with the World Heart Federation (5). Echocardiographic examinations of the right ventricle are challenging, and abnormalities in ARVC, particularly localized aneurysms, can easily be overlooked (6–8). Magnetic resonance imaging (MRI) has emerged to become the first line imaging technique for ARVC detection (9, 10).

Cine MRI provides cross sectional images with high spatial and temporal resolution. These represent the basis for accurate and reproducible evaluation of wall motion abnormalities of the left and right ventricle. Recently developed steady state free precession (SSFP) sequences with ultra fast slice acquisition capabilities significantly improved the robustness and image quality as well as spatial resolution and image contrast (11). Therefore, MR imaging using these techniques must be considered the imaging modality of first choice for the detection of regional and global RV

Received 4 May 2004; accepted 16 April 2005.
*Address correspondence to Peter Hunold, M.D., University Hospital Essen, Department of Diagnostic and Interventional Radiology and Neuroradiology, Hufelandstrasse 55, Essen 45122, Germany; Fax: +49-201-723-1548; E-mail: peter.hunold@uni-essen.de

Table 1. Demographic data and ARVC criteria of all 15 patients

Pat. no.	Age/sex	Primary clinical presentation	Family history	ECG abnormalities, arrhythmias	Global or regional dysfunction
1	23/m	Palpitations	no	>1000 VES/24 h*	no
2	**31/m**	Syncope	no	>1000 VES/24 h*, late potentials*	RV aneurysm, RV dilatation†
3	52/f	Ventricular tachycardia	no	no	no
4	**28/m**	Ventricular fibrillation	Brother*	Sustained LBBB-type VT*	Severe RV dilatation†
5	**21/m**	Family history	Brother*	>1000 VES/24 h*	Severe RV dilatation†
6	29/m	Syncope	no	no	no
7	**29/m**	Syncope	Father†	yes	RV aneurysm, RV dilatation†
8	20/m	Familial history	Father*	no	no
9	**33/m**	Syncope	no	Sustained LBBB-type VT*, late potentials*	RV aneurysm, RV dilatation†
10	32/f	Ventricular tachycardia	no	no	no
11	19/f	Syncope	no	no	no
12	**25/m**	Ventricular tachycardia	Brother*	Nonsustained LBBB-type VT*	RV aneurysm, RV dilatation†
13	33/m	Syncope	no	>1000 VES/24 h*	no
14	**45/f**	Ventricular tachycardia	no	Sustained LBBB-type VT*, late potentials*	RV aneurysm, severe RV dilatation†
15	26/m	Family history	Mother*	no	no

Bold printed patients fulfill the criteria for ARVC.
*Minor criterion.
†Major criterion. [From Refs. (4, 5).]

dysfunction and structural alterations in patients with suspected ARVC (10, 12, 13).

Furthermore, due to the excellent soft tissue contrast, MRI can distinguish fatty tissue from normal myocardium. Several studies have shown that MRI directly visualizes the fatty replacement of myocardium using T1-weighted imaging (6, 12, 14, 15). However, fatty tissue replacement of the myocardium seems to be less arrhythmogenic and, therefore, clinically less important than fibrous replacement (16). Pathologically controlled studies have shown that Gd-based contrast material accumulates generally in tissues with increased water content (17, 18). Thus, late contrast enhancement (LE) occurs in myocardial areas of fibrosis and edema where the extracellular volume is enlarged (19).

Table 2. MRI findings of all 15 patients

Pat. no.	Age/sex	Right ventricular EDV/ESV/EF*	Localized RV aneurysms	Fatty replacement in T1w TSE	Late enhancement in turboFLASH
1	23/m	152/65/57	no	no	no
2	**31/m**	233/149/36	Inferior	no	RV
3	52/f	125/60/52	no	no	no
4	**28/m**	350/257/27	no	no	RV, LV
5	**21/m**	356/307/14	no	no	RV, LV
6	29/m	186/80/57	no	no	no
7	**29/m**	223/132/41	Inferior	no	no
8	20/m	195/110/44	no	no	no
9	**33/m**	299/204/32	Infundibulum	no	no
10	32/f	115/59/49	no	no	no
11	19/f	124/51/59	no	no	no
12	**25/m**	252/172/32	Infundibulum	no	RV
13	33/m	134/66/51	no	no	no
14	**45/f**	237/169/29	Inferior	no	RV
15	26/m	175/83/53	no	no	no

Bold printed patients fulfill the criteria for ARVC based on the information given in Table 1.
*EDV and ESV are given as mL, EF is given as %.

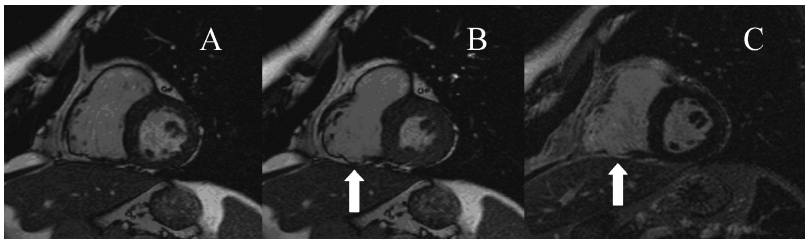

Figure 1. Diastolic (A) and systolic (B) short axis cine MR images show dyskinesia of the right ventricular free wall and the inferior wall of the right ventricle (arrows) in a 29-yr-old male patient (patient no. 7). The inversion-recovery turbo FLASH sequence shows late enhancement in the same wall segments.

This study was performed to evaluate whether late enhancement in contrast-enhanced MRI can be used to detect fibrous tissue within the right ventricular myocardium in patients with suspected arrhythmogenic right ventricular cardiomyopathy.

2. Methods

Within 11 months, 15 consecutive patients from the three affiliated cardiology institutions with clinical suspicion of ARVC were enrolled in this study. Following informed patients' consent, the study was performed in accordance with the regulations of the local institutional review board. Mean age of the patients was 30 years (range 19–52). All patients were referred to MRI because of clinically and electrocardiographically suspected ARVC as shown in Table 1. In all patients, coronary artery disease has been ruled out by coronary catheter angiography. None of them had previous ablational therapy.

2.1. *MR imaging*

Examinations were performed on a 1.5 T scanner (Magnetom Sonata, Siemens Medical Systems, Erlangen, Germany) equipped with high performance gradients. MRI scans of standard long-axis and contiguous short axis (no interslice gap) orientations were collected using different sequences: Cine imaging for the analysis of regional and global right and left ventricular function was performed with a conventional segmented steady-state free precession (TrueFISP: TR, 3 ms; TE, 1.5 ms; flip angle, 60°; views per segment, 15) sequence (11, 20). T1-weighted fast spin echo (TSE) sequences (TR, 700 ms; TE, 6.9 ms) without fat suppression were added in long and contiguous short axis views to assess the myocardium for fatty replacement (10, 14). To minimize partial volume effects, a slice thickness of 5 mm was used for all measurements.

Ten to 20 minutes following the injection of 0.2 mmol/kg Gd-DTPA (Magnevist, Schering AG, Berlin, Germany), scans were collected in the long and all contiguous short axis orientations using a breath-hold (end-inspiration)

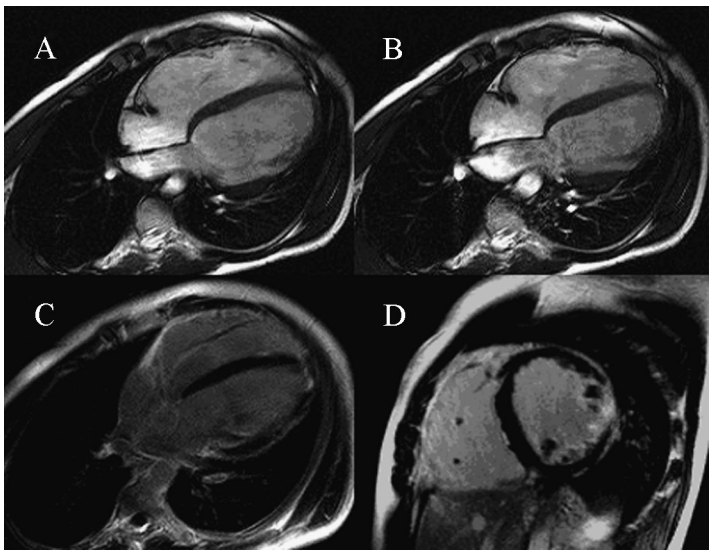

Figure 2. Diastolic (A) and systolic (B) cine MR images in the horizontal long axis orientation show severely enlarged, hypokinetic right ventricle in a 28-yr-old male patient (patient no. 4). The inversion-recovery turbo FLASH sequence shows contrast enhancement of the right (C, D) and the left ventricular wall (D).

ECG-triggered 2D inversion-recovery fast gradient echo sequence (TurboFLASH; TR, 8 ms; TE, 4 ms; flip angle, 25°; slice thickness, 5 mm) as described for the characterization of myocardial infarction (21–23). The inversion time (TI) was adjusted individually between 180 and 260 ms to null the signal intensity of normal RV myocardium. A rectangular field of view of 300 × 350 mm^2 and a matrix of 210 × 256 rendered an in-plane spatial resolution of 1.4 × 1.4 mm^2 for all sequences.

All MR examinations were interpreted in consensus by a radiologist and a cardiologist, both experienced in cardiac magnetic resonance imaging. Both readers were blinded to the medical history and the clinical findings of the patients. TrueFISP images were reviewed as cine-loops on a workstation, whereas hardcopies were used for the read-out of the TSE- and inversion recovery turbo FLASH images. Myocardial areas of high signal intensity in the contrast-enhanced turboFLASH images were classified as LE positive.

Measurements of RV volumes and ejection fraction were done using the Argus™ software (Siemens Medical Systems, Erlangen, Germany) by manual planimetry of the contiguous short axis slices and slice summation in the end-diastolic and end-systolic phase according to Pennell (24).

2.2. *ARVC diagnosis*

The final confirmation or exclusion of ARVC was based on the guidelines for the ''Diagnosis of arrhythmogenic right ventricular dysplasia/cardiomyopathy'' by the Task Force of the Working Group Myocardial and Pericardial Disease of the European Society of Cardiology (ESC) and of the Scientific Council on Cardiomyopathies of the International Society and Federation of Cardiology published in 1994 (4) and the modifications done in the consensus report with the Scientific Council on Cardiomyopathies of the World Health Federation published in 2000 (5). Demographic and ARVC criteria of the patients are given in Table 1. Endomyocardial biopsy was not performed in our collective.

Cine MR Imaging was considered the imaging modality of first choice to characterize ''global and/or regional dysfunction and structural alterations'' as minor and major criteria (10, 12, 13). Beyond cine imaging for the detection of regional or global dysfunction, other MRI features of abnormalities such as the proof of intramyocardial fat or late enhancement might not be used for ARVC diagnosis with respect to the given criteria (4, 5). All MRI findings of the patients are shown in Table 2.

3. Results

The main MR findings obtained in the 15 examined patients are summarized in Table 2. Mean total examination time was 56 ± 9 minutes. Seven (47%) of 15 patients showed akinetic or dyskinetic areas within the RV myocardium and dilatation of the right ventricle with reduced RV ejection fraction (Figs. 1 and 2). Both criteria were used as major diagnostic criteria for the diagnosis of ARVC. All seven patients had at least one additional major criterion or two additional minor criteria for the diagnosis of ARVC. Therefore, ARVC is highly probable in these patients according to the diagnosis guidelines (4, 5).

None of the seven patients with morphologic signs of ARVC exhibited signs of fatty replacement of the RV myocardium on the high-resolution T1-weighted turbo spin echo images. In five of the 7 patients with ARVC, however, late enhancement (LE) within the RV myocardium (Figs. 1 and 2) was detected corresponding to the areas of dyskinesia. The LE always presented transmural extent in areas of wall thinning. In 2 patients (no. 4/5) with right ventricular LE, additional transmural enhancement of the left ventricular lateral myocardium was seen (Fig. 2). These two patients were brothers and showed an almost identical pattern with severe RV dilation (RV end diastolic volume 350 mL/356 mL), reduced RV ejection fraction (27%/14%), diffuse LE of the RV myocardium and an area of LE in the mid-ventricular lateral wall of left ventricle. Both had no history of myocardial infarction or myocarditis.

Eight (53%) of 15 patients did not fulfill the diagnostic criteria of ARVC (4, 5). Therefore, the diagnosis of ARVC was deemed highly unlikely. In all of these patients, MRI failed to reveal morphologic or functional criteria of ARVC. Additionally, neither fatty replacement of the right ventricle nor LE was detected.

4. Discussion

Late enhancement in contrast-enhanced MRI appears in the right ventricular myocardium of some patients with ARVC. These regions might represent intramyocardial fibrous tissue. They were detected in 5 (71%) of the 7 patients with ARVC in our collective. Interestingly, fatty tissue was not identified by T1-weighted TSE MRI in any of these 7 patients.

4.1. *MRI criteria for ARVC*

In this study, morphologic criteria for ARVC were fulfilled in 7 (47%) of the 15 examined patients using MRI as the imaging technique of choice. Dilatation of the right ventricle, RV aneurysms, ectasia of the RV outflow tract, and enlargement of the right atrium are the most common MR criteria associated with the diagnosis of ARVC. Direct visualization of fatty replacement within the right ventricular myocardium is considered the most specific sign (1, 12, 14), but this MRI feature has not been implemented in the diagnostic criteria so far. Based on current literature, high signal intensity of fat and good contrast to normal myocardium combine to allow for accurate detection of fat within the right ventricular myocardium on T1-weighted spin-echo images (1, 6, 15). However, this technique has important limitations. Limited in-plane spatial resolution not exceeding 1.5 mm and image artifacts can render the

distinction of epi- or pericardial from intramyocardial fat most difficult (12). Correspondingly, in none of our 7 patients, we found those fatty infiltrates. Furthermore, in histological specimens, fatty infiltration occurs in more than 50% of normal hearts, especially in elderly patients (16). To date, only few efforts have been made to target fibrous tissue infiltrates within the myocardium with MR imaging.

4.2. *Pathologic findings in ARVC*

Arrhythmogenic right ventricular cardiomyopathy is characterized by fibrofatty replacement of the right ventricular myocardium. Histologically, the disease is characterized by a myocyte loss and fibrofatty substitution of the myocardium. Angelini et al. (25) examined specimens from endomyocardial biopsy and demonstrated that the percent area of myocytes decreased from 78% in control patients to 47% in ARVC whereas fat and fibrous tissue increased from less than 1% and 8% in the control group to 13% and 25%, respectively. A percentage of fat > 3% and of fibrous tissue > 40% with < 45% myocytes has been considered diagnostic of ARVC in a recent study (26). That demonstrates that the amount of fibrous tissue can be far higher than that of fat. Therefore, it seams reasonable to use MRI techniques that not only visualize fat but also detect fibrosis. Our data support that thesis since no fatty tissue could be visualized, but more than two thirds of the ARVC patients in our collective rendered late enhancement which is highly suggestive for fibrous tissue. However, it will not be possible to resolve whether this really is fibrous tissue without histopathology. Based on current literature, there are alternatives to explain the presence of LE as it has been shown to generally apply in edema, inflammation, and necrosis. A very recently published study by Tandri et al. reports LE in fibro-fatty areas of the right ventricular myocardium confirmed by endomyocardial biopsy (26). The authors found a very tight correlation between typical myocardial changes in histology and LE in MRI. Additionally, they found LE in 67% of patients with the final diagnosis of ARVC based on the ESC criteria, which very closely matches the percentage found in our study (71%).

Furthermore, Burke et al. suggest to distinguish two different types of right ventricular cardiomyopathy (16). According to them, typical ARVC is characterized by predominantly scarring and fibrous infiltration, whereas the other type is characterized solely by fat replacement of the myocardium. Fatty replacement of the myocardium seems to be less arrhythmogenic than fibrous replacement (16). Hence, the detection of fibrous infiltrates within the myocardium might be of importance regarding subsequent risk stratification.

4.3. *Late enhancement*

Using plain T1-weighted TSE sequences, fibro-fatty tissue can hardly be differentiated from normal myocardium (12). Recently, inversion-recovery gradient echo sequences have been introduced to visualize myocardial infarction and myocardial scars after administration of Gd-based contrast material (22, 23, 27). This technique, known as 'late enhancement', is characterized by excellent contrast between viable and non-viable myocardium, robustness and short acquisition times (22). The mechanism of enhancement is not completely understood; however, the enlargement of the interstitial space and different wash-in and wash-out kinetics of fibrotic areas within the myocardium compared to normal myocardium seem to be important factors (18, 19). The reliabilty of the late enhancement concept in MRI of ARVC might still benefit from techniques that facilitate the delineation between areas of late enhancement and surrounding tissue like epicardial fat or the blood pool. For those purposes, dark blood preparation and fat saturation might improve the technique in this very special collective (26).

4.4. *Left ventricular involvement*

LV involvement of arrhythmogenic right ventricular cardiomyopathy can only rarely be detected in-vivo. However, LV involvement has been detected in 40–100% (2, 28) at necropsy in patients with ARVC. To our knowledge, no systematic data are available on the frequency of LV involvement in MRI. Recently, McCrohon et al. reported a case of left ventricular involvement in ARVC seen in T1-weighted imaging without any evidence of macroscopic fibrosis on late enhancement imaging following contrast injection (29). A possible explanation of this finding, which is somewhat contradictory to our results, might be that the signal intensity of the myocardial infiltrations on different magnetic resonance images depends on the ratio of fatty and fibrous components. In our study, we found LE in the LV in 2 of the 6 ARVC patients. That is probably a remarkably higher prevalence than in the general ARVC population because these two patients were brothers; therefore, this small study population's results might be biased by including one familial form of ARVC.

4.5. *Limitations of the study*

ARVC diagnosis has been based on the ESC Task force criteria, one of the standard methods is histopathology of myocardial biopsy. Unfortunately, in our study, we did not have the opportunity to perform biopsies. So, our suggestions can not be proven on a cellular basis. However, the recently published study by Tandri et al. comparing MRI results with myocardial biopsy and electrophysiologic testing supports our suggestions and enhances the value of MRI in ARVC (26).

Although we included all consecutive patients referred from different cardiology departments with suspicion of ARVC in this study, the study population seems not to represent the typical ARVC patients. This might, in part, be a consequence of the small patient number. First, it is unusual to define more than half of the referred patients as having ARVC. Secondly, it is remarkable that none of our patients

had the typical finding of RVOT tachycardia. Thirdly, it remains unclear why none of our patients showed fatty myocardial infiltration in T1-weighted spin echo imaging which has been reported as the typical and pathognomonic feature in ARVC.

5. Conclusion

Late enhancement in contrast-enhanced MRI can be detected in the right and left ventricular myocardium in patients with suspected arrhythmogenic right ventricular cardiomyopathy suggesting areas of intramyocardial fibrous tissue. The addition of inversion recovery gradient echo sequences after contrast injection to the standard examination protocol might be advantageous in these patients. Clearly, the clinical impact of these findings in terms of diagnosis, treatment, and prognosis of the patients has not at all been clarified so far and can only be determined based on larger studies.

6. Abbreviations

ARVC	Arrythmogenic right ventricular cardiomyopathy/ dysplasia
LE	Late enhancement
RV	Right ventricle/right ventricular

References

1. Midiri M, Finazzo M, Brancato M, Hoffmann E, Indovina G, Maria MD, Lagalla R. Arrhythmogenic right ventricular dysplasia: MR features. Eur Radiol 1997; 7:307–312.
2. Gemayel C, Pelliccia A, Thompson PD. Arrhythmogenic right ventricular cardiomyopathy. J Am Coll Cardiol 2001; 38:1773–1781.
3. Corrado D, Basso C, Thiene G. Arrhythmogenic right ventricular cardiomyopathy: diagnosis, prognosis, and treatment. Heart 2000; 83:588–595.
4. McKenna WJ, Thiene G, Nava A, Fontaliran F, Blomstrom-Lundqvist C, Fontaine G, Camerini F. Diagnosis of arrhythmogenic right ventricular dysplasia/cardiomyopathy. 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 Cardiology. Br J Heart 1994; 71:215–218.
5. Corrado D, Fontaine G, Marcus FI, McKenna WJ, Nava A, Thiene G, Wichter T. Arrhythmogenic right ventricular dysplasia/cardiomyopathy: need for an international registry. Study Group on Arrhythmogenic Right Ventricular Dysplasia/Cardiomyopathy of the Working Groups on Myocardial and Pericardial Disease and Arrhythmias of the European Society of Cardiology and of the Scientific Council on Cardiomyopathies of the World Heart Federation. Circulation 2000; 101:E101–E106.
6. van der Wall EE, Kayser HW, Bootsma MM, de Roos A, Schalij MJ. Arrhythmogenic right ventricular dysplasia: MRI findings. Herz 2000; 25:356–364.
7. Lindstrom L, Wilkenshoff UM, Larsson H, Wranne B. Echocardiographic assessment of arrhythmogenic right ventricular cardiomyopathy. Heart 2001; 86:31–38.
8. Peters S, Brattstrom A, Gotting B, Trummel M. Value of intracardiac ultrasound in the diagnosis of arrhythmogenic right ventricular dysplasia-cardiomyopathy. Int J Cardiol 2002; 83:111–117.
9. Di Cesare E. MRI of the cardiomyopathies. Eur J Radiol 2001; 38:179–184.
10. Kayser HW, Van Der Wall EE, Sivananthan MU, Plein S, Bloomer TN, De Roos A. Diagnosis of arrhythmogenic right ventricular dysplasia: a review. Radiographics 2002; 22:639–648.
11. Barkhausen J, Ruehm SG, Goyen M, Buck T, Laub G, Debatin JF. MR evaluation of ventricular function: true fast imaging with steady-state precession versus fast low-angle shot cine MR imaging: feasibility study. Radiology 2001; 219:264–269.
12. Midiri M, Finazzo M. MR imaging of arrhythmogenic right ventricular dysplasia. Int J Card Imaging 2001; 17:297–304.
13. Schulz-Menger J, Friedrich MG. Magnetic resonance imaging in patients with cardiomyopathies: when and why. Herz 2000; 25:384–391.
14. Immer FF, Romanens M, Saner H. Images in cardiology. Visualising fatty deposits in familial arrhythmogenic right ventricular cardiomyopathy by magnetic resonance imaging. Heart 2000; 84:52.
15. Sommer T, Lewalter T, Bierhoff E, Pakos E, von Smekal A, Pauleit D, Hofer U, Luderitz B, Schild H. MRI diagnosis of right ventricular dysplasia. RoFo, Fortschr Geb Rontgenstrahlen Neuen Bildgeb Verfahr 1998; 169:609–615.
16. Burke AP, Farb A, Tashko G, Virmani R. Arrhythmogenic right ventricular cardiomyopathy and fatty replacement of the right ventricular myocardium: are they different diseases? Circulation 1998; 97:1571–1580.
17. Weinmann HJ, Brasch RC, Press WR, Wesbey GE. Characteristics of gadolinium-DTPA complex: a potential NMR contrast agent. AJR, Am J Roentgenol 1984; 142:619–624.
18. Adzamli IK, Jolesz FA, Bleier AR, Mulkern RV, Sandor T. The effect of gadolinium DTPA on tissue water compartments in slow- and fast-twitch rabbit muscles. Magn Reson Med 1989; 11:172–181.
19. Aso H, Takeda K, Ito T, Shiraishi T, Matsumura K, Nakagawa T. Assessment of myocardial fibrosis in cardiomyopathic hamsters with gadolinium-DTPA enhanced magnetic resonance imaging. Invest Radiol 1998; 33:22–32.
20. Carr JC, Simonetti O, Bundy J, Li D, Pereles S, Finn JP. Cine MR angiography of the heart with segmented true fast imaging with steady-state precession. Radiology 2001; 219:828–834.
21. Hunold P, Brandt-Mainz K, Freudenberg L, Vogt FM, Neumann T, Knipp S, Barkhausen J. Evaluation of myocardial viability with contrast-enhanced magnetic resonance imaging—comparison of the late enhancement technique with positronemission tomography [German]. RoFo, Fortschr Geb Rontgenstrahlen Neuen Bildgeb Verfahr 2002; 174:867–873.
22. Simonetti OP, Kim RJ, Fieno DS, Hillenbrand HB, Wu E, Bundy JM, Finn JP, Judd RM. An improved MR imaging technique for the visualization of myocardial infarction. Radiology 2001; 218: 215–223.
23. Kim RJ, Wu E, Rafael A, Chen EL, Parker MA, Simonetti O, Klocke FJ, Bonow RO, Judd RM. The use of contrast-enhanced magnetic resonance imaging to identify reversible myocardial dysfunction. N Engl J Med 2000; 343:1445–1453.
24. Pennell DJ. Ventricular volume and mass by CMR. J Cardiovasc Magn Reson 2002; 4:507–513.
25. Angelini A, Thiene G, Boffa GM, Calliari I, Daliento L, Valente M, Chioin R, Nava A, Volta SD, Calliaris I. Endomyocardial biopsy in right ventricular cardiomyopathy. Int J Cardiol 1993; 40:273–282.
26. Tandri H, Saranathan M, Rodriguez ER, Martinez C, Bomma C, Nasir K, Rosen B, Lima JA, Calkins H, Bluemke DA. Noninvasive detection

of myocardial fibrosis in arrhythmogenic right ventricular cardiomyopathy using delayed-enhancement magnetic resonance imaging. J Am Coll Cardiol 2005; 45:98–103.

27. Kim RJ, Fieno DS, Parrish TB, Harris K, Chen EL, Simonetti O, Bundy J, Finn JP, Klocke FJ, Judd RM. Relationship of MRI delayed contrast enhancement to irreversible injury, infarct age, and contractile function. Circulation 1999; 100:1992–2002.
28. Lobo FV, Silver MD, Butany J, Heggtveit HA. Left ventricular involvement in right ventricular dysplasia/cardiomyopathy. Can J Cardiol 1999; 15:1239–1247.
29. McCrohon JA, John AS, Lorenz CH, Davies SW, Pennell DJ. Images in cardiovascular medicine. Left ventricular involvement in arrhythmogenic right ventricular cardiomyopathy. Circulation 2002; 105:1394.