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REVIEW

The year 2018 in the *European Heart Journal—Cardiovascular Imaging*: Part II

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European Heart Journal - Cardiovascular Imaging was launched in 2012 as a multimodality cardiovascular imaging journal. It has gained an impressive impact factor during its first 5 years and is now established as one of the top cardiovascular journals and has become the most important cardiovascular imaging journal in Europe. The most important studies from 2018 will be highlighted in two reports. Part I of the review has focused on studies about myocardial function and risk prediction, myocardial ischaemia, and emerging techniques in cardiovascular imaging, while Part II will focus on cardiomyopathies, congenital heart diseases, valvular heart diseases, and heart failure.

Keywords echocardiography • CMR • CT • nuclear medicine

European Heart Journal - Cardiovascular Imaging has successfully consolidated as a multimodality journal during its first 5 years. It has now an important role as a significant resource for cardiologists, specialists in all imaging modalities, and other physicians working in the field of cardiovascular imaging. The tradition of highlighting the most important studies that were published in the last year[1,2] is continued.

In two articles, we will summarize the most important papers from the journal in 2018. Part I has just been published.[3] Part II will focus on cardiomyopathies, congenital heart diseases, valvular heart diseases, and heart failure (HF).

Recommendations and expert consensus documents from the European Association of Cardiovascular Imaging

One important assignment of *European Heart Journal - Cardiovascular Imaging* is to publish position papers, recommendations, and expert consensus papers from the European Association of Cardiovascular Imaging (EACVI). The journal published six recommendations and expert consensus papers in the field of multimodality imaging.[4–9] These papers are commented on in more detail elsewhere in the two documents.

Cardiomyopathies

Reant *et al.* performed a prospective study in hypertrophic cardiomyopathy (HCM) patients with New York Heart Association functional Class II. They used semi-supine bicycle and upright treadmill exercise echocardiography and compared the maximum provoked left ventricular outflow tract (LVOT) obstruction. Mean maximal peak exercise LVOT gradient with semi-supine bicycle was significantly lower than in treadmill exercise echocardiography. This pilot study suggested treadmill's greater value compared to semi-supine bicycle exercise echocardiography for determining maximum LVOT gradient in HCM.[10] Lu *et al.* examined whether LVOT obstruction influences the value of *E*/*e*′ in predicting outcomes in 640 HCM patients. Diastolic function was assessed by two-dimensional (2D) and Doppler echocardiography. A composite clinical outcome including new onset atrial fibrillation, sustained ventricular tachycardia/fibrillation, HF, transplantation, and death was examined over a mean follow-up period of 4.2 years. Higher *E*/*e*' was associated with worse event-free survival in non-obstructive group and total HCM cohort.

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During follow-up period, 95 patients underwent myectomy. Higher E/e' predicted worse clinical outcomes in non-obstructive HCM and in labile/obstructive after myectomy.[11]

In another study, Jain *et al.* examined the haemodynamic effect of normal respiration on LVOT obstruction in 20 patients with HCM with phasic respiratory variation of LVOT obstruction on initial transthoracic 2D echocardiogram (TTE) and Doppler examination. LVOT gradients were re-examined with simultaneous recording of a respirometer. Counterintuitive respiratory-related fluctuations in LVOT gradients were observed. LVOT gradients varied widely during the respiratory cycle; peak gradients were uniformly lowest during inspiration and highest during expiration. These findings challenge traditional haemodynamic teaching and demonstrate the contribution of left ventricle (LV) transmural pressure to LVOT obstruction in certain HCM patients.[12] Multiple studies demonstrated that imaging parameters could be reliably used in establishing early diagnosis and monitoring therapy in Fabry disease. Militaru *et al.* performed a review on multimodality imaging in Fabry cardiomyopathy. Given the rarity of this disorder, they concluded that awareness should be raised about the imaging 'red flags' (papillary muscle hypertrophy, reduced inferolateral basal longitudinal strain, reduced circumferential strain . . .), and likely, patients should be sent for evaluation in expert centres.[13] In a collaborative document between the Brazilian Cardiovascular Imaging Department and the EACVI, the most recent evidences about the non-invasive assessment of patients with Chagas disease were reviewed and summarized (*Figure 1*). The intent was to set up a framework for standardized cardiovascular imaging to assess cardiovascular morphologic and functional disturbances, as well as to guide the subsequent process of clinical decision-making.[14] Gotschy *et al.* compared the 2010 task force criteria measures of right ventricular outflow tract (RVOT) dilation with three alternative measures for improving the echocardiographic assessment of RVOT in patients with arrhythmogenic cardiomyopathy (AC). Cardiac magnetic resonance (CMR) and TTE were performed in 38 patients with a definite, borderline, or possible AC diagnosis and in 10 healthy controls. Among all RVOT diameters examined, the one defined by the parasternal long axis M-mode of the LV provided the best agreement between CMR and TTE and exhibited the best diagnostic accuracy for AC.[15] Van der Bijl *et al.* performed left ventricular 2D speckle-tracking echocardiography for detection of systolic dysfunction in genetic, dilated cardiomyopathies. They included 115 genotyped dilated cardiomyopathy (DCM) patients. Decreased LV global longitudinal strain could discriminate genotype positive phenotype negative individuals from normal controls, which may permit early institution of therapy for genetic DCM.[16] In a systematic literature review and meta-analysis of randomized controlled trials on non-invasive screening for coronary artery disease (CAD) in asymptomatic diabetic patients, Clerc *et al.* included 3299 patients. During 4.1 years of follow-up, 5.7% experienced any cardiac event. Compared with the standard care, non-invasive CAD screening reduced cardiac events by 27% in asymptomatic diabetic patients, largely through reductions in non-fatal MIs, and HF hospitalizations. The authors concluded that the results justify larger, appropriately powered trials to potentially revisit current recommendations.[17]

CMR is a useful tool to characterize cardiomyopathies and to provide insight into their underlying mechanisms. This was demonstrated by several papers published in the journal which evaluated different types of cardiomyopathies. Raman *et al.* evaluated the impact of fibrosis progression evaluated by late gadolinium enhancement (LGE) between 2 CMR studies performed at a median of 6 years in 72 HCM patients. They observed substantial fibrosis progression >4.7% myocardial mass in 26% of patients. LGE progression was higher in patients with impaired myocardial perfusion reserve and altered energetics evaluated by 31P spectroscopy (*Figure 2*). Substantial LGE progression was associated with LV thinning, increased cavity size and reduced systolic function, and conferred a five-fold increased risk of subsequent clinical events.[18] Grover *et al.* evaluated myocardial oxygenation changes by BOLD CMR imaging during adenosine stress in 20 MYBPC3 gene positive patients with HCM, 18 MYBPC3 mutation carriers without HCM 11 gene negative siblings, and 11 normal controls. They found reduced blunted myocardial oxygen response not only in patients with HCM but also in mutation carriers without HCM, with normal wall thickness and systolic and diastolic function. Thus, these two elegant studies point to the important role of myocardial ischaemia in HCM pathogenesis and fibrosis development and demonstrate that these findings precede the development of hypertrophy.[19] Furthermore, an important review by Quarta *et al.* summarized the indications of performing CMR in HCM patients, with particular importance to the clinical context in both adult and paediatric population, and highlighting implications for clinical research.[20] Phospholamban PLN p.Arg14del mutations lead to development of cardiomyopathy and risk of arrhythmia. Therefore, Te Rijdt *et al.* studied a population of 150 genetically homogeneous mutation carriers from the PHORECAST registry who had undergone CMR. They demonstrated that fibrosis by LGE is an early feature of this type of cardiomyopathy and was present not only in patients with established DCM, but also in 29% of mutation carries with preserved ejection fraction (EF). They also demonstrated that ECG alterations with negative T waves were frequently associated with LGE and that CMR-LGE was independently associated with occurrence of ventricular arrhythmia. Based on these findings, they conclude that CMR could be recommended in the diagnostic workout of both symptomatic and asymptomatic carriers of this DCM mutation.[21] Similarly, Marty *et al.* evaluated 85 patients with Becker muscular dystrophy (BMD). They demonstrated not only functional abnormalities and dyssynchrony but also significantly higher native T1 T2 and extracellular volume (ECV) even in subclinical phenotypes. This suggests that that native T1 and T2 mapping could provide biomarkers related to inflammation and fibrosis processes that stratify disease severity in BMD patients with a comparable sensitivity than quantitative contrast-enhanced CMR.[22]

Araujo *et al.* performed tissue characterization by T1 mapping and ECV in non-compaction cardiomyopathy. They demonstrated significantly higher T1 and ECV times suggesting expansion of extracellular matrix in this cardiomyopathy, even in myocardium without focal fibrosis (LGE). The authors also observed the association between ECV expansion and LV dysfunction, but not with the severity of non-compacted myocardium. This suggests that ECV imaging might be used for risk stratification in non-compaction cardiomyopathy. Differentiation between athlete's heart and cardiomyopathies remains challenging.[23] Therefore, Claessen *et al.* used exercise CMR for functional cardiac evaluation in 10 endurance athletes with mildly reduced left ventricular ejection fraction (LVEF) that were compared

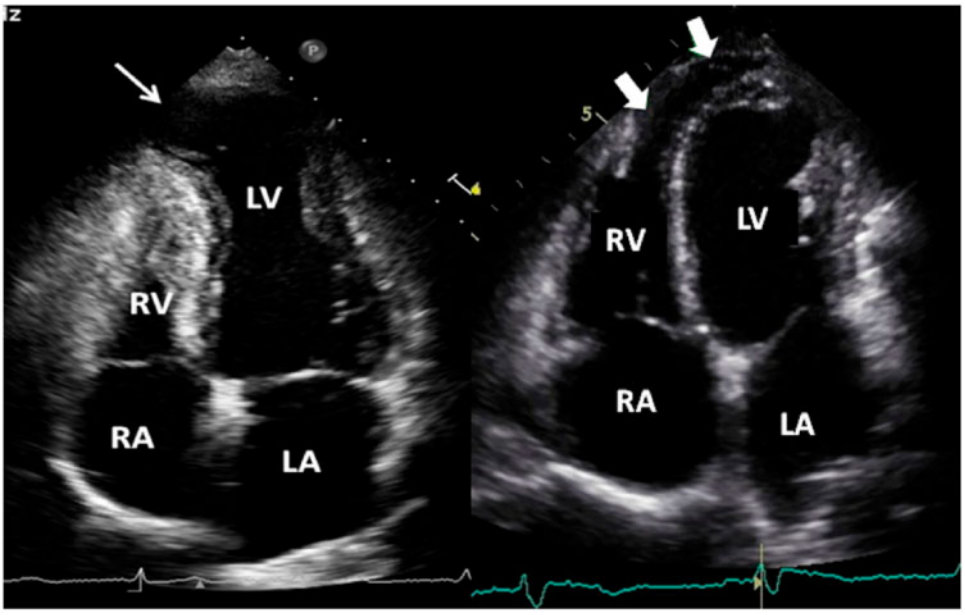

Figure 1 Apical four-chamber views showing left ventricular apical aneurysm (thin arrow, on the left), and right ventricular aneurysm (broad arrows, on the right) with a relatively normal LV in a patient with Chagas disease.[14]

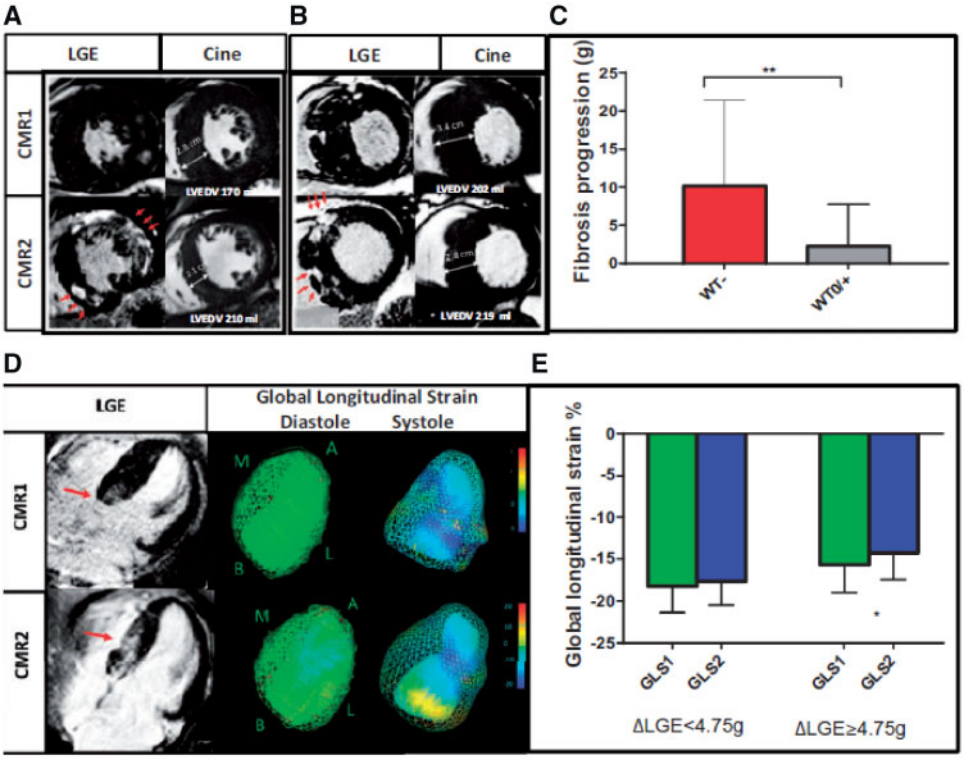

Figure 2 LGE/fibrosis progression (red arrow indicated LGE progression) results in a reduction of the wall thickness (WT-) (*A–C*) Increase in end-diastolic volume (*A, B*) and impairment in myocardial contractility (*D, E*) (WT0/- stable or increasing wall thickness; GLS, global longitudinal strain), $^{**}P < 0.01$, errors bars represent standard deviation, $^{*}P < 0.05$.[18]

to 9 patients with DCM and 5 athletes with presence of intramyocardial fibrosis. DCM patients and athletes with fibrosis had reduced adaptation of LVEF and contractility during exercise compared to healthy athletes. This suggests that functional cardiac evaluation during exercise could be a promising tool in differentiating healthy athletes with borderline LVEF from those with an underlying cardiomyopathy[24]

Congenital heart disease

Other studies illustrated the potential of CMR to evaluate congenital heart disease.

Gnanappa in 126 children and adults with chronic pulmonary regurgitation (PR) with repaired tetralogy of Fallot (rTOF) used CMR and cardiopulmonary exercise test (CPET) data in order to characterize the effects of varying degrees of right ventricle (RV) dilatation on LV volumes, function and stroke volume and on RV–LV interaction. They have also documented changes in these LV parameters and RV–LV interaction after pulmonary valve replacement (PVR). They found that patients with severe RV dilation had pulmonary regurgitant and forward flow and LV volumes, but lower RV ejection fraction than patients with less severe RV dilatation. Septal curvature and VO2 max were similar in both groups. After PVR, while there was significant reduction of significant reduction in RV volumes, stroke volume and pulmonary regurgitant flow, LV volumes, septal curvature, or VO2 max were unchanged. This may potentially explain preserved exercise capacity in rTOF with severe PR and RV dilatation and explain the lack of improvement in aerobic capacity after PVR.[25] Additionally, Samad *et al.* used machine learning to comprehensively investigate the predictive value of the baseline variables derived from CMR in rTOF providing models for the identification of patients at risk of deterioration in ventricular size and function at a median of 2.6 years. The algorithm identified baseline LV ejection fraction, LV circumferential strain, and PR as the primary parameters for predicting deterioration.[26] In patients with single ventricle repaired by Fontan, Kamphuis *et al.* evaluated intraventricular viscous energy loss. They found that Fontan patients had significant energy loss relative to kinetic energy compared to healthy controls, and highest energy loss is found in patients with discordant inflow-to-outflow.[27] Finally, Grotenhuis quantified imaging markers of myocardial fibrosis and assessed myocardial function in 30 long-term survivors after arterial switch operation for correction of transposition of great vessels relative to controls. They found that LVEF was normal although LV volumes were increased and LV strain was reduced. T1 times were increased, while no patient had LGE or perfusion defects. It is unknown whether these elevated imaging markers of diffuse myocardial fibrosis, along with an altered LV contraction pattern will progress into future clinically significant dysfunction and whether they are harbingers of adverse outcomes.[28]

In 2018, it has been published an important recommendation paper from the EACVI and chaired by Di Salvo *et al.*[6] This document is underscoring the diagnostic utility of a multimodality cardiovascular imaging. The multimodality approach is greater than the sum of individual tests. The aims of this document are to describe the role of each diagnostic modality in the care of adult with congenital heart disease patients and to provide guidelines for a multimodality approach. The authors tried to provide the most appropriate and cost-effective diagnostic pathway for each adult patient with congenital heart disease.

In addition, readers of the journal should be called out by a proof of concept paper. This paper shows how mixed reality holograms as surgical planning tool for congenital heart disease may have a high diagnostic value and contribute to understanding complex morphology. It is a new application for the images that could be generated.[29] Also, in 132 patients, computer-based tools could detect underlying diagnosis in complex congenital heart disease. Automated delineation of the ventricular area was feasible. These methods may in future allow for the longitudinal, objective, and automated assessment of ventricular function.[30]

Valvular heart disease

Among the great paper accepted for publication is 2018 in the *European Heart Journal - Cardiovascular Imaging*, there was a retrospective study conducted in asymptomatic patients with moderate mitro-aortic valve disease (a combination of moderate aortic stenosis and moderate aortic regurgitation) and an ejection fraction of 50% or more. These patients were followed up at Mayo Clinic. The authors were able to demonstrate that the presence of a relative wall thickness >0.42 or *E/e'* >14 can identify a high-risk patient subset in whom the risk for cardiovascular morbidities was persisting, even after aortic valve replacement (AVR).[31] These two simple parameters could be considered in order to avoid a too late reference for surgery of patients that should have a benign prognosis after AVR.

Echocardiography of course is the key in aortic stenosis (AS) but computed tomography (CT) is important too. Surgery is challenged by percutaneous approaches especially for the AS and not that much for aortic regurgitations. High aortic valve calcification (AVC) assessed with CT may be used to differentiate between severe and non-severe AS. Also, for transcatheter aortic valve implantation (TAVI), the evaluation of calcifications is required. One paper was looking at 93 patients that had low CT aortic valve calcification score (AVCS). These patients were compared to 470 patients with high AVCS.[32] There was a high device success and lower rates of paravalvular regurgitation. Balloon-expandable TAVI in patients with a mildly calcified aortic valve was not associated with increased risk of valve embolization or mortality. 1396 patients were included in the Bern TAVI Registry and prospectively followed up through echocardiographic and clinical evaluation.[33] Transcatheter heart valve thrombosis (THVT) was suspected in case of: (i) a mean transvalvular pressure gradient greater than 20 mmHg at TTE, or (ii) an increase of more than 50% of the mean transvalvular pressure gradient compared with previous measurements, or (iii) new symptoms or signs of HF with the presence of thrombus documented by transoesophageal echocardiography (TOE) or multi-slice CT. THVT occurred in 10 patients (0.71%) at variable time points after TAVI. Oral anticoagulant therapy (with vitamin K antagonists or non-vitamin K antagonists) was initiated in all but two patients and resulted in normalization of transvalvular pressure gradients and amelioration of clinical status within 1 month. At long-term follow-up (between 10 and 25 months

after valve thrombosis), echocardiographic findings were stable and no adverse events were reported.

Left bundle branch block (LBBB) is a frequent conduction abnormality after TAVI. An original study including 140 consecutive patients with a new onset of LBBB after TAVI was commented in the Journal.[34] Interestingly, classical dyssynchronous LBBB contractions were only observed in 2 patients (7%). In addition, patients with and without new onset LBBB experienced similar prognosis with regards to mortality.[35]

Bicuspid aortic valve (BAV) is a frequent condition (0.5–2% of the population), and it could be associated with aortic valve disease and/or aortic aneurism. A close correlation between genetic and biomolecular patterns of elastin and mechanical properties of the aorta has been reported. Aortic dimensions and elastic properties were determined by echocardiography, aortic stiffness (AS) by M-mode analysis, and longitudinal strain of the ascending aorta by speckle-tracking echocardiography. The presence of mutation was associated with increased amount of elastin soluble fragments, larger ascending aorta, and lower longitudinal strain.[36]

Primary mitral regurgitation (PMR) can be considered as a heterogeneous clinical disease.[37] The optimal timing of valve surgery for severe PMR remains unknown. It has been determined that using unbiased clustering analysis using dense phenotypic data (phenomapping), it could be identified phenotypically distinct PMR categories of patients. The risk or atrial arrhythmias could be estimated and that new methodological approach using a very large amount of imaging (mainly echocardiographic data) could be very relevant for best characterizing our patients and then provide a personalized management and the treatment.

Mitral valve (MV) abnormalities are recognized features of HCM, and there is preliminary evidence suggesting they are intrinsic phenotypic manifestations of sarcomere mutations, present in mutation carriers without LV hypertrophy (subclinical HCM). MV and papillary muscle echocardiographic parameters were measured in 192 genotyped individuals, including 50 overt HCM, 79 subclinical HCM, and 63 mutation-negative, healthy relatives as normal controls.[7] Sarcomere mutations are associated with primary abnormalities of the MV apparatus, specifically excess anterior leaflet length relative to LV cavity size and anterior displacement of the papillary muscles; both features predisposing to septal anterior motion. These abnormalities appeared to be early phenotypic consequences of sarcomere mutations, observed in mutation carriers with normal LV wall thickness.[38]

Improved MV leaflet coaptation with consecutive reduction of mitral regurgitation (MR) is a central goal of percutaneous mitral valve repair (PMVR) with the MitraClip system. As influences of PMVR on MV geometry have been suggested.[8] Geometry of the MV annulus was evaluated in 183 patients undergoing PMVR using echocardiography before and after the procedure and at follow-up. The reduction of MV antero-posterior diameter showed a significant inverse correlation with residual MR. Importantly, the reduction of MV antero-posterior diameter persisted at follow-up.[39] In addition, the effect of MitraClip implantation on LV remodelling has been studied. Serial echocardiography before, 1 and 6 months after MitraClip implantation was performed in 79 pts with severe MR. Patients were followed over a period of 32 ± 16 months with all-cause mortality as the primary endpoint. A sustained (6 month) reduction of MR ≤ 2 post-clip.

MitraClip implantation was observed in 83% of patients. The average decrease in LVEDV at 6 months after intervention was 13% ± 16%. Reverse remodelling at 6 months occurred in 40 patients (51%), and adverse remodelling occurred in 6 patients (8%).[40] The majority of patients undergoing MitraClip implantation for severe MR showed LV reverse remodelling. However, there was a small group in whom afterload mismatch resulted in sustained adverse remodelling with subsequent high mortality.

BAV is among the most common congenital cardiac abnormalities, and this anatomic variation is associated with greater risk of rapid leaflet degeneration and calcification, leading to stenosis of the aortic orifice. Although TAVI has emerged as a promising alternative to surgery in patients with symptomatic aortic stenosis and contraindications to surgery, or in select patients at high risk for surgery, bicuspid anatomy has been considered as an exclusion in randomized trials. In a recent study, Kawamori *et al.*, aimed of assess transcatheter heart valve (THV) geometry and valve function associated with implantation of the SAPIEN 3 valve in 280 consecutive patients (41 with BAV), as well as clinical outcomes, when compared with tricuspid aortic valve (TAV) disease. Compared to TAV, BAV patients had larger annuli, aortic size, and greater leaflet calcium volume, BAV patients had greater THV eccentricity index at all levels and lower THV expansion ratio at the mid-, sinus-, and outflow-level. Regarding valve function using post-procedural echocardiography, there were no differences in frequency of any grade of paravalvular aortic regurgitation and mean gradient between BAV and TAV. Besides, the rate of high mean gradient (≥20 mmHg) was not different between two groups. TAVI with SAPIEN 3 in BAV allows for feasible procedural results in spite of their THV deformity. However, evaluation of long-term clinical outcome is required, since an asymmetric expansion might lead to early degeneration of the leaflets.[41]

The independent prognostic value of a secondary tricuspid regurgitation was studied in 639 patients admitted for acute HF. Only 7% of patients with moderate or severe TR were free of other cardiac lesions. In adjusted models, moderate or severe TR was not associated with readmission for HF or mortality. Moderate or severe TR was associated with the risk of hospitalization but essentially when a pulmonary hypertension was associated with this secondary valvular regurgitation. The independent prognostic value of isolated secondary TR was not found in that observation study.[42]

Multivalvular disease remains a clinical challenge, even more when secondary to a cardiomyopathy. An extremely nice study has been conducted in 1021 consecutive patients with HF and reduced LVEF. These were optimally treated and followed during 5 years. They conclude that moderate bivalvular functional regurgitation conveys similar risk as isolated severe functional regurgitation, reflecting the deleterious impact of the global regurgitant load on the failing heart and the need of an integrated understanding for risk-assessment.[43]

The same authors did a nice study about 249 patients with a secondary MR and they looked the evolution of MR under guideline-directed therapy.[12] Progression of MR conveyed an increased risk of mortality at 61-month. It has been observed in 19% of the studied population. These patients with an increase in the degree of secondary MR during follow-up have a more than two-fold increased risk of

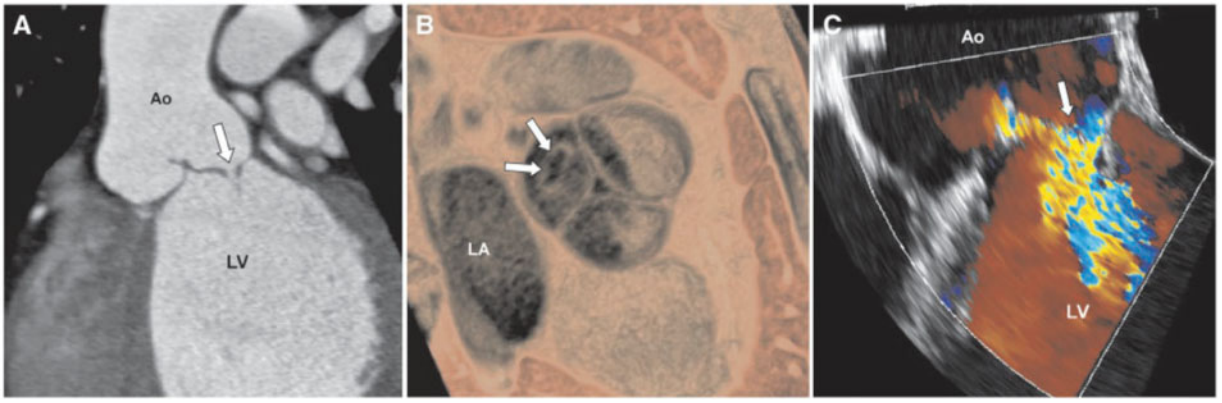

Figure 3 A 50-year-old man with infective endocarditis. (*A*) An oblique sagittal reconstructed image and (*B*) an in-plane volume-rendered image showing a perforated cusp (arrows). (*C*) With colour Doppler imaging, the perforated left coronary cusp shows regurgitant flow (arrow).[45]

death even after careful multivariable adjustment. Symptomatic status, left atrial size, tricuspid regurgitation, and neurohumoral pathways help to identify patients at risk for progressive secondary MR in an early disease process and open the possibility for closer follow-up and timely intervention.

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CT can be also of help for the evaluation of paravalvular leakage (PVL). Although small PVLs are common and usually benign, they have been associated with poorer survival. Forty-six patients who had been diagnosed with PVL and underwent cardiac CT were included in the study published by reference 44. On CT, the characteristics of PVLs including number, size, location, and shape are described. The sizes of PVL were smaller in patients who treated with device closure or observed without treatment compared to those in patients who underwent surgical correction. PVL sizes were larger in patients with higher regurgitant grades on echocardiography ($P < 0.05$). Cardiac CT can demonstrate the location and size of PVL. The size of PVLs are larger in patients who managed by surgical correction. PVL size measured on CT is correlated with the regurgitant grade on echocardiography. According to these preliminary results, cardiac CT can be used to detect and localize aortic and mitral PVLs. Cardiac CT was also used by the same group of authors (Koo *et al.*) to compare imaging findings of infective endocarditis between CT and TOE using surgical inspection as a reference standard. Forty-nine patients (69% men) who underwent pre-operative CT and TOE for infective endocarditis were included. The detection rates of infective endocarditis per patient with CT and TOE were 93.9% and 95.9%, respectively. In per-imaging analysis, the sensitivities of CT and TOE were not significantly different for both native and prosthetic valve infective endocarditis (*Figure 3*), indicating that cardiac CT could accurately demonstrate infective endocarditis in pre-operative patients with a similar diagnostic accuracy to TOE.[45]

The visualization of anatomic information using a familiar view for either a surgeon or an interventionist may aid in the planning of a treatment strategy. Kwizinsky *et al.* evaluated the prognostic value of asymmetric wall thickening using both echocardiography and CMR in 166 patients with aortic stenosis. Twenty-six percent of patients had asymmetric wall thickening. As opposed to patients with concentric hypertrophy with similar severity of valve stenosis, patients with asymmetrical hypertrophy had higher ECV and replacement fibrosis (LGE). Also asymmetrical hypertrophy was associated with worse outcomes independent of age, sex, left ventricular mass index, coronary artery, and aortic stenosis severity. This suggests that its presence may help identify patients likely to proceed quickly towards AVR.[46]

Finally, two reviews covered the role of CMR in PMR (Liu *et al.*) with specific emphasis on the possibility to better assess MR severity specifically when there is discrepancy between MR severity by echocardiography and clinical findings.[47] Another multimodality review described the anatomy of tricuspid valve (TV) and surrounding structures as revealed by CT, CMR, 2D/3D transthoracic echocardiography (TTE), and 2D/3D TOE, emphasizing strengths and weaknesses of each of these imaging tools.[48]

Heart failure

Sacre *et al.* examined mild cognitive impairment and its associations with subclinical cardiac dysfunction in patients with chronic heart disease yet to develop the clinical syndrome of chronic heart failure (CHF). Patients from the Nurse-led Intervention for Less Chronic Heart Failure Study ($n = 373$ with chronic heart disease other than CHF) were screened for mild cognitive impairment (Montreal cognitive assessment score <26) and underwent echocardiographic and

clinical profiling. Mild cognitive impairment was prevalent in patients with subclinical chronic heart disease at high risk of CHF and suggested a link between cardiac and cognitive functioning beyond shared risk factors.[49]

Morris *et al.* aimed to determine the lower limit of normality and the clinical relevance of LV early diastolic strain rate (LVSRe) for the detection of LV diastolic dysfunction (LVDD). They analysed 377 healthy subjects and 475 patients with risk for LVDD with preserved LVEF using 2D speckle-tracking echocardiography. They provided data regarding the normal range of LVSRe and highlighted the potential clinical relevance of using this new diastolic parameter in the detection of LVDD in patients with preserved LVEF.[50] Moneghetti *et al.* assessed the incremental value of advanced echocardiographic and cardiopulmonary exercise testing (CPX) parameters to validated risk scores in HF. The Meta-Analysis Global Group in Chronic Heart Failure (MAGGIC) and Metabolic Exercise Test Data Combined with Cardiac and Kidney Indexes (MECKI) scores were applied to 208 ambulatory patients with DCM who completed echocardiography and CPX. Patients were followed for the composite endpoint of death, heart transplant, left ventricular device implantation, and hospitalization for acute HF. Right atrial volume indexed was complementary to well-validated HF risk scores and highlighted the importance of exercise performance in DCM.[51]

Cvijic *et al.* investigated the relationship between time of onset of longitudinal shortening, regional myocardial work, and segmental LV wall thickness in patients treated with cardiac resynchronization therapy (CRT). They analysed 26 patients with sinus rhythm, non-ischaemic cardiomyopathy, and positive response to CRT (15% reduction in end-systolic volume). Longitudinal strain was obtained by 2D speckle-tracking echocardiography before and after [14.5 (7–29) months] CRT. Dyssynchronous myocardial shortening was related to thinning of early and thickening of late activated segments in HF with conduction delay. Correction of dyssynchrony lead to regression of inhomogeneity towards more evenly distributed wall thickness. Regional differences in myocardial work-load that were homogenized by successful CRT were considered as the underlying pathophysiological mechanism.[52] Peak cardiac power output-to-mass (CPOM) represents a measure of the rate at which cardiac work is delivered respect to the potential energy stored in left ventricular (LV) mass. Pugliese *et al.* studied the value of CPOM and CPET in risk stratification of patients with HF. They studied 159 patients with chronic HF (mean rest LVEF 30%) undergoing CPET and exercise stress echocardiography. Results showed that both peak CPOM and peak VO2 showed independent and incremental prognostic values in patients with chronic HF.[53]

Sympathetic nerve function plays a crucial role in CHF outcomes. In the clinical assessment of CHF condition, neuroimaging with ^{123}I-meta-iodobenzylguanidine (MIBG) is a unique diagnostic method to evaluate the integrity of cardiac sympathetic activity and innervation. Recently, a CHF mortality risk model using a cardiac ^{123}I-MIBG parameter together with clinical information was developed using a Japanese CHF database consisting of 1322 patients, which was shown effective for the prediction of 2- and 5-year cardiac mortality risks. More recently, Nakajima *et al.* published a study designed to validate the predictability of the 2-year mortality risk model, developed by combining cardiac ^{123}I-MIBG results with clinical information, using a cohort of CHF patients derived from multiple clinical centres in Japan. A cohort of 546 patients (follow-up time 30 ± 20 months) was enrolled. The prediction of the 2-year risk model was superior to that of individual variables such as left ventricular ejection fraction, BNP/N-terminal prohormone of brain natriuretic peptide (NT-proBNP), or MIBG results (such as heart/mediastinum ratio). It is particularly noted that, irrespective of LVEF and BNP/NT-proBNP levels, the risk model discriminated CHF patients at low to intermediate risks. The high-risk population was well discriminated from others, but the actual event rate observed was underestimated by this model. These results, if confirmed by other cohort of patients, could have an impact on clinical practice, for a better risk stratification.[54]

Conflict of interest: none declared.

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