Cardiac Magnetic Resonance Image: The costly gold standard

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The most frequently used techniques for assessment of the LV are echocardiography and electrocardiography. Echo is widely available, but image acquisition depends on the operator and the acoustic window (**Allison JD, Flickinger FW, Wright CJ, Falls DG III, Prisant LM, VonDohlen TW, Frank MJ. Measurement of left ventricular mass in hypertrophic cardiomyopathy using MRI: comparison with echocardiography. Magn Reson Imaging 1993;11:329–334**). Reproducibility is reasonable in normal LVs (**Gordon EP, Schnittger I, Fitzgerald PJ, Williams P, Popp RL. Reproducibility of left ventricular volumes by two-dimensional echocardiography. J Am Coll Cardiol 1983;2:506–513**.), but the quantification of volumes and LVMass relies on geometric assumptions that do not apply to ventricles undergoing asymmetric cardiac remodeling such as in cardiomyopathy(**Kronik G, Slany J, Mosslacher H. Comparative value of eight M-mode echocardiographic formulas for determining left ventricular stroke volume. Circulation 1979;60:1308–1316**.). CMR is independent of geometric assumptions (**Bellenger NG, Grothues F, Smith G, Pennell DJ. Quantification of right and left ventricular function by MRI. Herz 2000;25:392–399**.) and has been shown to be accurate(**Higgins CB. Which standard has the gold? J Am Coll Cardiol 1992;19:1608– 1609**.) and reproducible(. **Lorenz CH, Walker ES, Morgan VL, Klein SS, Graham TP Jr. Normal human right and left ventricular mass, systolic function and gender differences by cine magnetic resonance imaging. J Cardiovasc Magn Reson 1999;1:7–21**.).

Comparison of reproducibility of CMR and echocardiography

Interstudy reproducibility of CMR is excellent for measurements of LV volumes and mass in a large mixed study group and has superior interstudy reproducibility of CMR over 2-D echo in the same subjects and in any subgroups. Reproducibility is generally better in normal subjects than in patients with abnormal hearts, and this is the case for CMR and echo. Poorer image quality (e.g., patient's inability to keep up with breath-hold time, presence of arrhythmias) and, with CMR, slow-moving blood in patients with HF (making endocardial border detection difficult), are the most likely reasons for this finding.

CMR can precisely estimate a patient's LVMass and assess for other structural cardiac abnormalities. Myocardial tissue characterization by CMR has emerged to not only quantify LVMass, but also detect and quantify the extent and severity of focal or diffuse myocardial fibrosis, edema, inflammation, myocarditis, fatty replacement, myocardial disarray, and myocardial deposition of amyloid proteins (amyloidosis), glycolipids (Fabry disease), or iron (hemochromatosis) (Bacharova & Ugander, 2014). The amount of myocardial fibrosis measured by gadolinium enhanced cardiac MRI is an independent predictor of mortality in patients with moderate and severe **VAS**. Specifically, LGE on cardiac MRI of the mid-wall, the typical pattern of fibrosis in LVH, is a stronger predictor of mortality than infarct-pattern fibrosis from epicardial coronary disease and thus, may be an important intermediate outcome measure worth-while preventing (Dweck et al., 2011).

The drawbacks of MRI are

1. MRI scanners are very expensive; a single scanner can cost over a million pounds. This means that the number of scanners that a Primary Care Trust (PCT) can afford to fund is limited.

2. Long time consuming to acquire,

3. The combination of being put in an enclosed space and the loud noises that are made by the magnets can make some people feel claustrophobic while they are having a MRI scan.

4. MRI scanners can be affected by movement, making them unsuitable for investigating problems such as mouth tumors because coughing, or swallowing, can make the images that are produced less clear.

5. Bone and calcium do not show up on an MRI scan. This means that tissue calcification, cannot be detected using MRI scanning.

6. Lack of broadly applied criteria relating to LVMass. While

Echocardiogram performed in a 68-year-old man being evaluated for uncontrolled hypertension and symptoms of CHF. LVH is diagnosed by an elevated LVMI, which is calculated from the IVSd, PWTd, and LVIDd.

Cardiac MRI is the gold standard test for LVH, as it is even more accurate and reproducible than echocardiography (Bottini et al., 1995). It can precisely estimate a patient's LVMass and assess for other structural cardiac abnormalities. By calibration with CMR, a wide range of predictive values was found for the various ECG criteria for LVH with the most favorable results for the Romhilt-Estes score (Buchner et al., 2009). There are many ECG criteria for the diagnosis of LVH. There are, however limited data on the accuracy of these criteria in comparison with CMR. Krittayaphong et al studied a

total of 1,882 patients, 994 males and 888 females. Average age was 64.6 +/- 11.3 years. LVH was diagnosed by CMR in 23.3% in female and 25.4% in male. ECG criteria for the diagnosis of LVH had a relatively low sensitivity (0.25-0.61), and high specificity (0.75-0.95). Female had a lower sensitivity, higher specificity, higher PPV similar NPV and higher overall accuracy than male. Cornell product, Romhilt-Estes (at least 4 points) and Sokolow-Lyon were the ECG criteria with the best accuracy, sensitivity and specificity, respectively (**Krittayaphong R1, Nomsawadi V, Muenkaew M, Miniphan M, Yindeengam A, Udompunturak S.ccuracy of ECG criteria for the diagnosis of left ventricular hypertrophy: a comparison with magnetic resonance imaging.J Med Assoc Thai. 2013 Feb;96 Suppl 2:S124-32**.).

Echocardiography vs. CMRI

	2D echocardiography	CMRI
	Less expensive, superior versatility, acceptability, and availability	Expensive, smallest expensive and has inferior versatility, acceptability, and availability
LVMass accuracy	Smaller	More accurate and precise (Bottini et al., 1995)
Interstudy reproducibility	Inferior to MRI echocardiography for normal, dilated, and hypertrophic hearts	Superior to 2D echocardiography for normal, dilated, and hypertrophic hearts (Grothues et al., 2002)

To determine if there is a significant difference in the predictive abilities of LVH detected by ECG-LVH versus LVH ascertained by cardiac MRI-LVH in a model similar to the Framingham Heart Failure Risk Score (FHFRS) Oseni et al, included 4745 (mean age 61±10 years, 53.5% women, 61.7% non-whites) participants in the Multi-Ethnic Study of Atherosclerosis. ECG-LVH was defined using Cornell voltage product while MRI-LVH was derived from left ventricular mass. Cox proportional hazard regression was used to examine the association between ECG-LVH and MRI-LVH with incident heart failure (HF). Harrell's concordance C-index was used to estimate the predictive ability of the model when either ECG-LVH or MRI-LVH was included as one of its components. ECG-LVH was present in 291 (6.1%), while MRI-LVH was present in 499 (10.5%) of the participants. Both ECG-LVH and MRI-LVH were predictive of HF. The absolute risk of developing HF was 8.81% for MRI-LVH versus 2.26% for absence of MRI-LVH with a relative risk of 3.9. With ECG-LVH, the absolute risk of developing HF 6.87% compared with 2.69% for absence of ECG-LVH with a relative risk of 2.55. The ability of the model to predict HF was better with MRI-LVH than with ECG-LVH. The aythirs cinckuded that ECG-LVH and MRI-LVH are predictive of HF. Substituting MRI-LVH for ECG-LVH improves the predictive ability of a model similar to the FHFRS (Oseni et al., 2017). The results of this manuscript are not without limitations. Although there are several criteria for detection of LVH by ECG, the authors only used Cornell voltage product, one of the most commonly used ECG criteria in clinical practice (Truong et al., 2010), and confirmed the results using another couple of criteria. There have been some controversies regarding whether to include the papillary muscles in quantifying the myocardial mass when using cardiac MRI. However, excluding the

papillary muscles has been shown to have better reproducibility (Armstrong et al., 2012; Vogel-Claussen et al., 2006). Despite these limitations, this study is the first to compare the predictive ability of ECG-LVH with MRI-LVH (the gold standard for estimating LVMass), using a model similar to a validated predictive model, in an ethnically diverse population of middle age and old adults.

The Role of Cardiac MRI in the Diagnosis and Risk Stratification of HCM

HCM, is the most frequent genetic cardiomyopathy characterized by substantial heterogeneity. Although the majority of patients with HCM remain asymptomatic with near-normal longevity, a small, but important, subset remain at risk for a wide range of clinical outcomes including SCD. CMR, has emerged as an imaging modality particularly well suited to characterize the phenotypic expression of HCM. CMR helps in the diagnosis of HCM by identifying areas of hypertrophy not well visualized by echo, providing more accurate wall thickness measurements and differentiating HCM from other causes of LVH. CMR has led to the identification of novel subgroups of patients with HCM, including those with LV apical aneurysms (a subgroup at increased risk for ventricular arrhythmias and thromboembolic stroke), as well as abnormalities that contribute to LVOTO. Additionally, contrast-enhanced CMR with LGE has recognized patients with extensive LGE (≥15 % LV myocardium) as individuals who may be at increased risk of SCD, independent of other high-risk features, with implications on management strategies including consideration for primary prevention ICDT. These observations justify an expanded role of CMR in the routine clinical assessment of patients with HCM (**Rowin EJ, Maron MS. The Role of**

Cardiac MRI in the Diagnosis and Risk Stratification of Hypertrophic Cardiomyopathy. Arrhythm Electrophysiol Rev. 2016;5(3):197-202.)

Examples
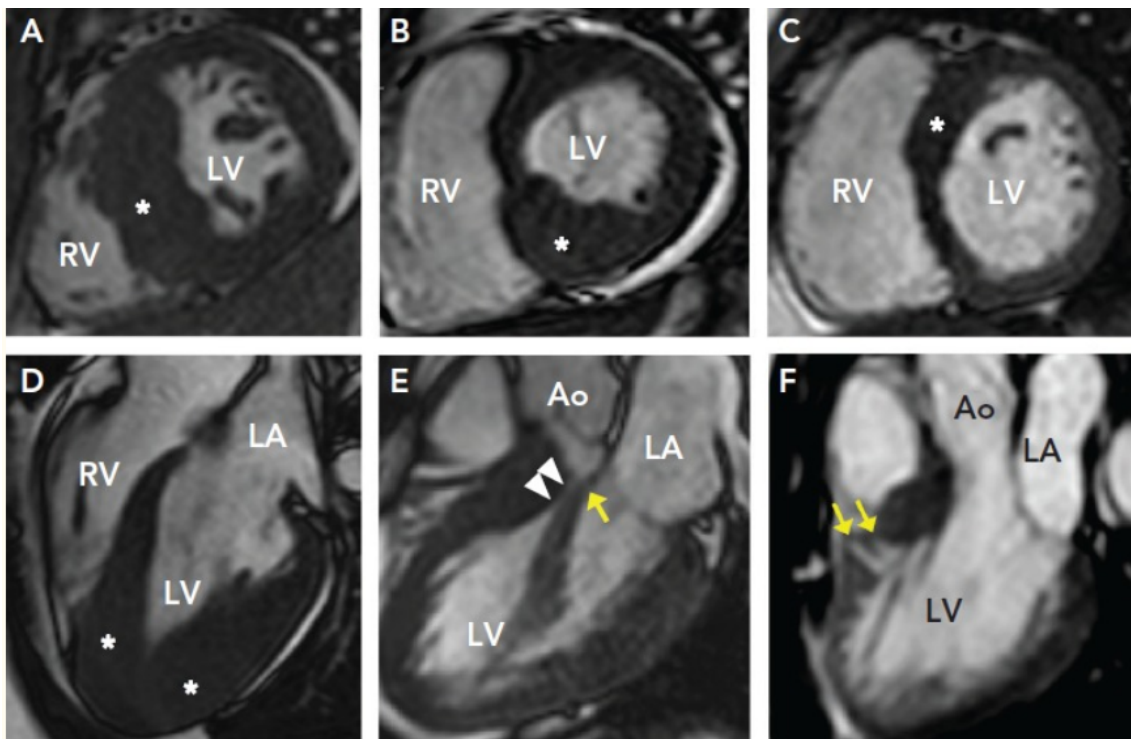
CMRIs in 6 patients carrying HCM with several phenotypic expression

A–C: Short-axis CMRIs demonstrating: (A) massive LVH (31 mm of wall thickness) confined to the IVS (asterisk), (B) massive LVH (wall thickness =30 mm) in the inferior septum and inferior wall (asterisk) and (C) mild asymmetric hypertrophy of the septum (asterisk; wall thickness =16 mm) in a patient with a disease-causing sarcomere mutation in the myosin-binding protein C gene. D: Four-chamber long-axis view demonstrating hypertrophy localized to the LV apex (asterisks). E: Three-chamber long-axis view demonstrating muscular midcavitary obstruction attributable to the insertion of anomalous anterolateral papillary muscle directly into anterior leaflet (arrow) contacting the midventricular septum in systole (arrowheads). F: A 24-year-old genotype-positive phenotype-negative man with two deep, narrow myocardial crypts (arrows) in the anterior septum, considered a morphological marker for affected status. Ao = aorta; CMR = cardiovascular magnetic resonance; HCM = hypertrophic cardiomyopathy; LA = left atrium; LV = left ventricle; RV = right ventricle.

Figure
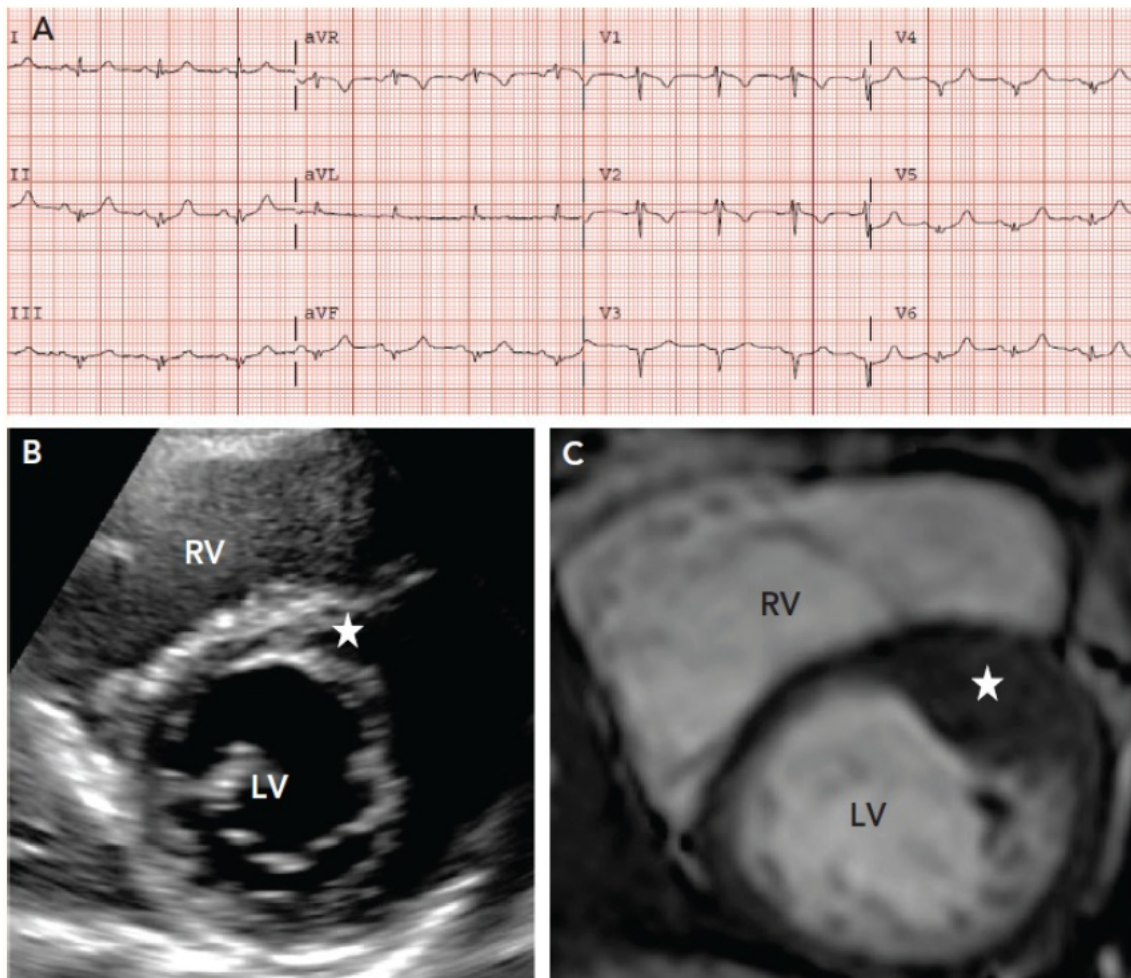
CMR for HCM Diagnosis

An asymptomatic 36-year-old woman with a family history of HCM. A: Twelve-lead ECG: incomplete RBBB and anterior and inferior Q waves. B: 2D echo demonstrated normal LV wall thickness. C: Given abnormal ECG, patient underwent CMR, which reveals an area of segmental hypertrophy in the anterolateral LV wall (asterisk) consistent with a diagnosis of HCM.

Figure
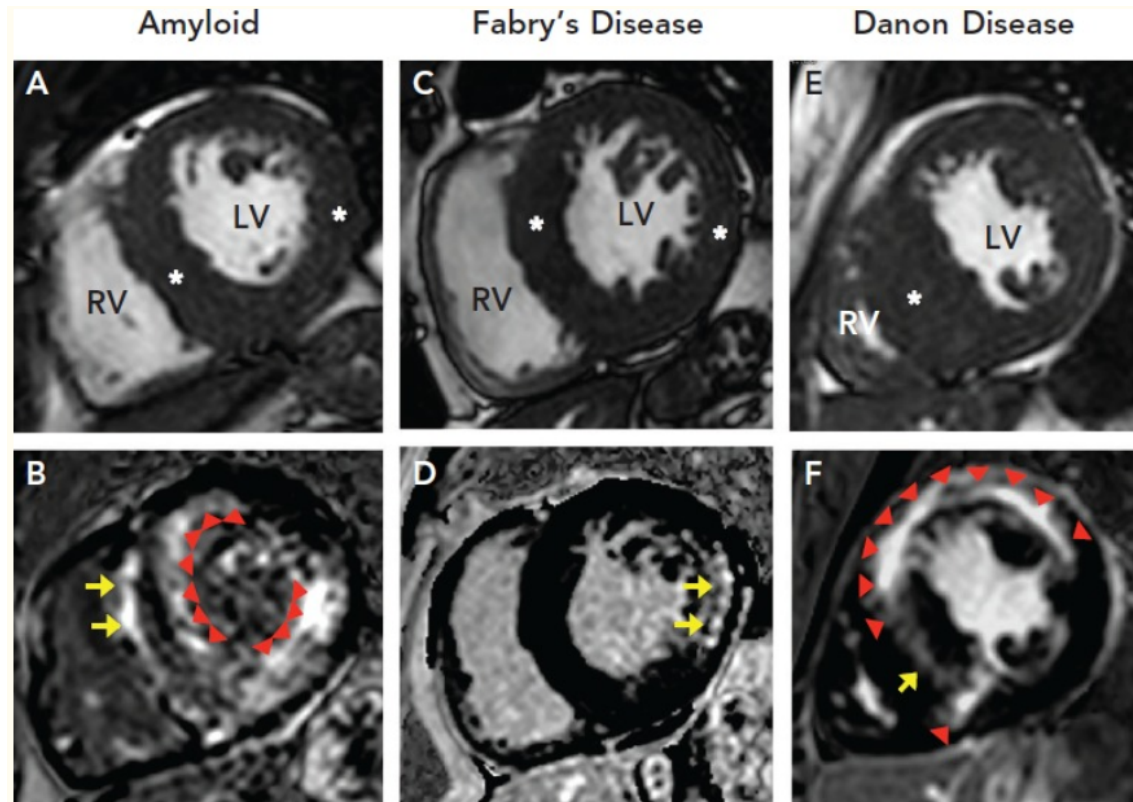
CMR for differentiation of etiology of LVH

I. **Cardiac Amyloidosis confirmed by cardiac biopsy:** A: Pre-contrast short-axis CMR image in a 64-year-old man with increased LV wall thickness in both septum and lateral wall (maximum wall thickness of 18 mm in septum and 14 mm in lateral wall). B: Post-contrast images in the same patient demonstrates early contrast washout with epicardial LGE in septum (arrows) and global subendocardial LGE (arrowheads) leading to concern for amyloidosis.

II. **Fabry's disease confirmed by genetic testing (pathogenic mutation in the galactosidase αgene).** C: Pre-contrast short-axis CMR image in a 44-year-old woman with increased LV wall thickness in both IVS and lateral wall (maximum wall thickness of 16 mm in septum and 13 mm in the lateral wall). D: Post-contrast images in the same patient demonstrate LGE confined to the basal inferolateral wall leading to concern for Fabry's disease.

III. **Danon Disease. Genetic testing was thereby sent and revealed a pathogenic mutation in the lysosomal-associated membrane protein 2 gene**. E: Pre-contrast short-axis CMR image in a 21-year-old man demonstrated massive LVH (wall thickness of 32 mm) confined to the IVS (asterisk). F: Post-contrast images in the same patient demonstrated transmural LGE throughout the

anterior and lateral walls with mid-myocardial LGE throughout the IVS in a pattern atypical for HCM.

Figure
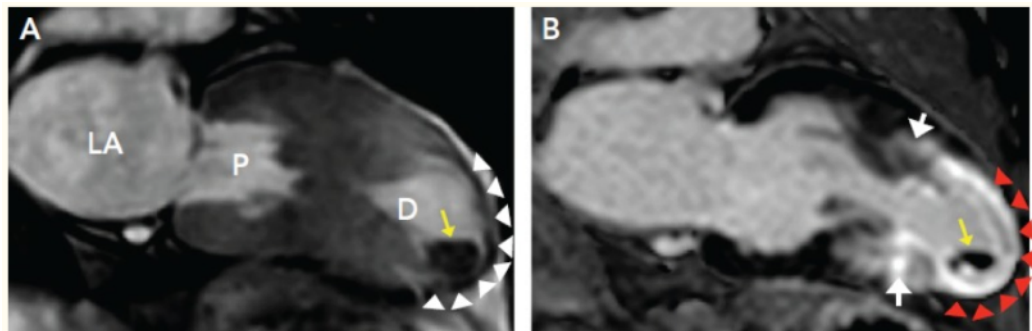
LV Scarring associated with apical aneurysm in a patient with HCM