# ORIGINAL PAPER

# Prevalence of cardiomyopathy in asymptomatic patients with left bundle branch block referred for cardiovascular magnetic resonance imaging

Masliza Mahmod · Theodoros D. Karamitsos · Joseph J. Suttie · Saul G. Myerson · Stefan Neubauer · Jane M. Francis

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Abstract The diagnostic evaluation of patients with isolated left bundle branch block (LBBB) is challenging due to limitations of several non-invasive tests. Our aim was to evaluate the diagnostic value of cardiovascular magnetic resonance (CMR) in asymptomatic patients with LBBB. Sixty-one asymptomatic patients with complete LBBB who were referred for CMR from January 2005 to November 2010 were identified. 29 patients (18 men) had normal echocardiograms (echo) whereas 25 (18 men) had abnormal findings on echo. Six had no echo and one had poor echo windows, and these patients were excluded from further analysis. Patients with cardiac symptoms or known coronary artery disease at the time of referral were also excluded. Of the 29 patients with normal echo, 9 (31%) were found to have pathological findings on CMR. The most common abnormalities were dilated cardiomyopathy-DCM (n = 6, 21%) followed by left ventricular hypertrophy (n = 2, 7%). Of the 25 patients who had an abnormal echo, CMR confirmed the diagnosis in 19 (76%) and provided clinically relevant additional information in 13 (52%) subjects. Of these 13 patients, 9 (69%) had characteristic patterns of myocardial late gadolinium

M. Mahmod · T. D. Karamitsos · J. J. Suttie · S. G. Myerson · S. Neubauer · J. M. Francis (⊠) Department of Cardiovascular Medicine, University of Oxford Centre for Clinical Magnetic Resonance Research (OCMR), John Radcliffe Hospital, Oxford OX3 9DU, UK e-mail: jane.francis@cardiov.ox.ac.uk enhancement (8 mid-wall and 1 patchy distribution consistent with DCM and cardiac sarcoid, respectively). CMR detects sub-clinical cardiomyopathy in a third of asymptomatic patients with LBBB despite normal echocardiograms. In those with abnormal echocardiograms, CMR provides additional clinically relevant information in over 50% of patients, including a high prevalence of mid-wall fibrosis in patients with impaired left ventricular function. These findings support the use of CMR as a valuable adjunct to conventional investigations in asymptomatic LBBB.

**Keywords** ECG conduction abnormalities · Late gadolinium enhancement · Dilated cardiomyopathy · Diagnosis · Echocardiography

## Introduction

The prevalence of left bundle branch block (LBBB) in the general population is low, ranging from 0.1 to 0.8% [1], but the majority of patients have underlying cardiac abnormalities which include coronary artery disease (CAD), hypertension and dilated cardiomy-opathy (DCM) [1–4]. Previous studies have demonstrated that asymptomatic patients with LBBB have worse long term cardiovascular outcomes when compared with their matched controls [5, 6]. Left bundle branch block can also occur however in apparently healthy individuals without overt heart

disease [5], and this poses a clinical dilemma for further investigation. LBBB can be an early marker of cardiomyopathy and may also have a causative role in the development of cardiac remodelling and hypertrophy [7]. Therefore it would be important to identify whether patients with asymptomatic LBBB have any features of underlying cardiac disease. The diagnostic evaluation of LBBB is challenging however, due to the limitations of standard non-invasive tests [8]. Two-dimensional echocardiography is the first line imaging modality and is good at identifying wall motion abnormalities, but it lacks the ability to provide tissue characterization and differentiate ischemic from non-ischemic cardiomyopathies. LBBB can also interfere with myocardial scintigraphy, giving a higher rate of false positive defects in the septum with overall lower specificity [9]. Multi-slice computed tomography is useful in detecting CAD but has limited value in the investigation of non-ischemic cardiomyopathy [10]. Nuclear and CT scans also utilize ionizing radiation, which is ideally avoided [11].

Cardiovascular magnetic resonance (CMR) is used increasingly in a wide range of cardiac diseases of both ischaemic and non-ischaemic origin [12–15]. Although patients with LBBB are sometimes referred for CMR, the clinical utility of CMR in asymptomatic patients with LBBB has not been studied. The present study aims to evaluate the diagnostic value of CMR in asymptomatic patients with LBBB who have no history of significant cardiac disease.

### Methods

Between January 2005 and November 2010, a total of 3596 patients underwent clinical CMR at our institution. As this was a retrospective analysis, ethical approval was waived by the Local Research Ethics Committee. From these 3596 scans we identified 117 patients with complete LBBB (QRS duration > 120 ms). All these patients were referred for further evaluation of LBBB. 56 patients had cardiac symptoms (angina, dyspnea, palpitations, pre-syncope or syncope) or known cardiac disease (CAD, valvular heart disease or cardiomyopathy) at the time of referral and were excluded from the study. The remaining 61 patients were asymptomatic and were included in the study. Of these, 29 had normal and 25 had abnormal echocardiograms. Six patients who did not have an echo and one who had poor echo windows (total 7 patients) at referral were excluded from further analysis. Criteria for an abnormal echo included reduced left ventricular (LV) ejection fraction (<54%), dilated cardiac chambers (LV end-diastolic diameter > 59 and 53 mm for men and women, respectively), presence of regional wall motion abnormality, LV hypertrophy (LV septal thickness > 13 mm), valvular heart disease and evidence of inducible ischemia on stress echo. The presence of LBBB was established by their treating physician prior to referral for CMR. The presence of abnormal septal motion typical of LBBB, if it was the sole finding, was not considered as an 'abnormal' echo. Echocardiographic reports were obtained from the referring physicians and from the hospital echo reports. Anthropometric data, pre-existing conditions, medications, smoking status and family history were also recorded.

CMR studies were performed on a 1.5 Tesla MR system (Siemens Sonata or Avanto, Erlangen, Germany) using steady-state, free precession breath-hold cines in long-axis planes and sequential 7 mm shortaxis slices from the atrioventricular ring to the apex. Late gadolinium enhancement (LGE) images were acquired 10 min after gadolinium-DTPA (0.15 mmol/ kg) in identical long- and short-axis planes using an inversion-recovery gradient echo sequence. Inversion times were adjusted to null normal myocardium. Myocardial stress perfusion using adenosine was performed at the request of the referring physician where clinically indicated, as previously described [16]. Ventricular volumes and function were measured using standard techniques as previously published (Argus Syngo MR software version B15, Siemens Healthcare, Erlangen, Germany) [17]. The following left ventricular parameters were obtained: end-diastolic volume, end-systolic volume, interventricular septal wall thickness, LV ejection fraction and myocardial mass (indexed to body surface area).

### Statistical analysis

Results from normally distributed continuous data are expressed as mean  $\pm$  standard deviation (SD) and non-normally distributed data are expressed as median (interquartile range-IQR). Categorical variables are presented as number and percentages. The independent *t* test or Mann–Whitney test was used to compare continuous variables between LBBB patients with

Table 1 Baseline         characteristics of the cohort		Normal echo $(n = 29)$	Abnormal echo $(n = 25)$	P value
	Age [years (mean, SD)]	$52.2 \pm 9.7$	57.5 ± 13.5	0.07
	Male gender, no (%)	18 (62)	18 (72)	0.39
	BMI [kg/m <sup>2</sup> (mean, SD)]	$27.7 \pm 4.9$	$28.1 \pm 5.7$	0.94
	SBP [mm Hg (mean, SD)]	$138.6\pm20.5$	$148.4 \pm 18.6$	0.25
	DBP [mm Hg (mean, SD)]	$80.0\pm9.3$	$82.2\pm6.9$	0.62
	Heart rate [bpm (mean, SD)]	$72.1 \pm 11.0$	$70.1 \pm 11.3$	0.54
	Medications			
	Aspirin, no (%)	4 (13)	3 (12)	-
	Beta-blockers, no (%)	4 (13)	1 (4)	-
	ACE/ARB, no (%)	4 (13)	5 (20)	-
	Diuretics, no (%)	1 (3)	1 (4)	-
	Calcium channel blocker, no (%)	1 (3)	-	-
	Nitrates, no (%)	1 (3)	-	-
	Statin, no (%)	5 (17)	3 (12)	-
	Past medical history			
	Hypertension, n (%)	9 (31)	8 (32)	-
	Hypercholesterolemia, n (%)	6 (20)	2 (8)	-
	Diabetes, n (%)	1 (3)	1 (4)	-
ACE Angiotensin-converting enzyme, ARB Angiotensin receptor blocker, BMI Body mass index, DBP Diastolic blood pressure, SBP Systolic blood pressure, SD Standard deviation, SPECT Single photon emission computed tomography	Family history of heart disease, n (%)	8 (27)	3 (12)	-
	Smoker/ex-smoker, n (%)	5 (17)	5 (20)	-
	Coronary angiogram			
	Unobstructive, n (%)	9 (31)	8 (32)	-
	Dobutamine stress echo	10 (34)	1 (3)	
	SPECT	2 (6)	_	

normal and abnormal CMR scans as appropriate. Chisquare test or Fisher's exact test was used to compare categorical variables between the two groups as appropriate. A value of P < 0.05 (2-sided) was considered significant. Statistical analysis was performed with SPSS version 19.0 (IBM SPSS Statistic 19).

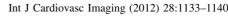
# Results

# **Baseline** characteristics

Baseline characteristics and LV measurements of the two group of patients (29 asymptomatic LBBB patients with normal echo and 25 asymptomatic LBBB patients with abnormal echos) are presented in Table 1. The mean age of the normal-echo group was  $52.2 \pm 9.7$  years, and 18 (62%) were men. None of the patients had a history of cardiac disease prior to referral for CMR. The most common associated medical conditions were hypertension (9 patients-31%) and hyperlipidemia (6 patients-20%). Eight patients (27%) had a family history of either CAD or cardiomyopathy. Nine patients (31%) had previous coronary angiography which was normal. Furthermore, 10 patients had undergone dobutamine stress echo (DSE) which was negative for ischaemia. Two patients of the normal echo group underwent myocardial perfusion SPECT, in which one of them was normal and the other one showed inducible ischaemia, with subsequent normal coronary angiogram.

For the abnormal echo group, the mean age was  $57.5 \pm 13.5$  years, and 18 (72%) were men. None of the patients had a history of cardiac disease prior to referral for CMR. The most common associated medical condition was hypertension (8 patients-

Table 2   Cardiovascular		Abnormal echo	Normal echo	P value
magnetic resonance findings		(n = 29)	(n = 25)	
	CMR LV dimensions and function			
	LVEDV [ml (mean, SD)]	$157.0 \pm 36.7$	$197.0 \pm 61.7$	0.007
	LVESV [ml (mean, SD)]	$60.9\pm24.6$	$101.4 \pm 57.8$	0.001
	LVEF [ml (mean, SD)]	$62.4\pm8.4$	$53.1\pm5.6$	0.006
	LV thickness [mm (median, IQR)]	7.9 (5.1)	10.8 (4.7)	0.082
	LVMI [g/m <sup>2</sup> (mean, SD)]	$65.4 \pm 14.5$	$84.1 \pm 18.1$	< 0.001
	CMR diagnoses, no (%)			
CAD Coronary artery dis- ease, CMR Cardiovascular magnetic resonance, IQR Interquartile range; LV Left ventricle/ventricular, LVEDV Left ventricular end-diastolic volume, LVEF Left ventricular ejection fraction, LVESV Left ventricular end-systolic volume, LVMI Left ventricular mass index, SD Standard deviation	Normal CMR	20 (69)	3 (12)	-
	Abnormal CMR			
	DCM	6 (21)	13 (52)	-
	LV hypertrophy	2 (7)	2 (8)	-
	Ebstein anomaly	1 (3)	0	-
	Athletic heart	0	1 (4)	-
	CAD	0	3 (12)	-
	LV Non compaction	0	1 (4)	-
	Sarcoid	0	1 (4)	-
	Pericardial abnormalities	0	1 (4)	-



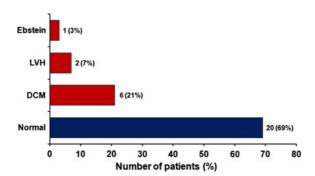


Fig. 1 CMR findings in asymptomatic patients with LBBB and normal echocardiogram

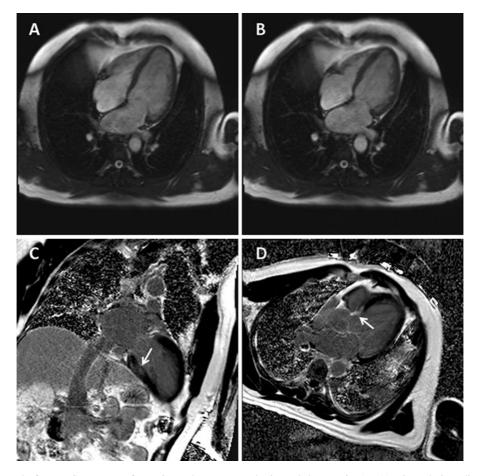
32%). Three (12%) patients had a family history of either CAD or cardiomyopathy. Eight (32%) patients had previous coronary angiography which was normal. One patient had undergone DSE which was negative for ischaemia, and none had SPECT.

CMR findings in asymptomatic LBBB patients with a *normal* echocardiogram

CMR findings of the 29 asymptomatic LBBB patients who had a normal echo are presented in Table 2. Of the 29 patients, 9 (31%) had abnormal CMR findings, summarized in Fig. 1 and Table 2. Dilated cardiomyopathy was the most common diagnosis, which was present in 6 patients (21%) of whom 1 had mid-wall fibrosis (Fig. 2). Left ventricular hypertrophy (defined as regional LV wall thickness  $\geq 13$  mm) was found in 2 patients (7%) and Ebstein anomaly in 1 patient (3%). In the remaining 20 patients (69%), no abnormalities on CMR were detected. Only three patients had adenosine stress perfusion CMR with no evidence of inducible ischaemia.

CMR findings in asymptomatic LBBB patients with *abnormal* echocardiograms

CMR findings of the 25 asymptomatic LBBB patients who had an abnormal echo are summarized in Table 3. CMR and echo agreed on the underlying diagnosis in 19 of those 25 patients (76%). In 13/25 patients (52%), CMR provided additional clinically relevant information over and above the echocardiographic findings which elucidated the cardiac diagnosis (Table 4). This included 8 patients in whom late gadolinium enhancement-CMR showed typical mid-wall fibrosis, establishing a diagnosis of DCM. In 6/25 patients (24%) CMR disagreed with the echocardiographic findings, including 3 cases in which CMR was normal where the echocardiogram had shown significant LV hypertrophy or abnormal LV function.



**Fig. 2** An example from a CMR scan of a patient who was diagnosed to have DCM with fibrosis. This 58-year-old hypertensive was asymptomatic and incidentally found to have LBBB. Echocardiogram was normal and coronary angiogram revealed non-obstructive CAD. A and B are cine images in the

 
 Table 3 Cardiovascular magnetic resonance findings of asymptomatic patients with left bundle branch block and abnormal echocardiogram

	<i>n</i> = 25
Agreement with diagnosis	19 (76%)
Additional clinically relevant information	13 (68%)
No additional clinically relevant information	6 (32%)
Disagreement with diagnosis, and new diagnosis by CMR	6 (24%)

## Discussion

An important finding of this study is that CMR is useful in the evaluation of asymptomatic patients with LBBB, in patients with both normal and abnormal echo

horizontal long axis (HLA) view during diastole (A) and systole (B) showing globally dilated left ventricle. C and D are late gadolinium-enhanced vertical long axis (VLA) and LV outflow tract (LVOT) views demonstrating mid-wall myocardial fibrosis (*arrows*)

studies. Our study demonstrated that CMR can identify abnormalities in one-third of asymptomatic patients with LBBB despite a *normal* echo. Additionally, in those who had *abnormal* echo, CMR provided new, clinically important information in half of the cases. The results of our study demonstrate the utility of a comprehensive CMR protocol, including cardiac morphology, structure and function, in evaluating the cause of LBBB in asymptomatic patients.

In the group of asymptomatic LBBB patients with *normal* echo, the commonest CMR finding was DCM. This is supported by Bayes-genis et al. who found a high prevalence of LBBB in their DCM patients [18]. The second commonest diagnosis, LV hypertrophy, is likely related to the high prevalence of hypertension in our cohort, which concurs with a

Findings agreed by both CMR and echo $(n = 13)$	Additional information by CMR	Final diagnoses	No (%)
Impaired LV function	Positive mid-wall LGE	DCM with fibrosis	8 (61)
	Patchy mid-wall LGE	Cardiac sarcoid	1 (8)
	Trabeculated LV	LV Non compaction	1 (8)
Dilated LV	Myocardial perfusion defect	CAD	1 (8)
RV wall abnormality	LV hypertrophy	Athletic heart	1 (8)
Pericardial effusion	Thickened pericardium and flattened septum on inspiration	Pericardial constriction	1 (8)

Table 4 Additional information by cardiovascular magnetic resonance in patients with left bundle branch block and *abnormal* echocardiogram

CAD Coronary artery disease, CMR Cardiovascular magnetic resonance, DCM Dilated cardiomyopathy, LGE Late gadolinium enhancement, LV Left ventricular, RV Right ventricular

previous study [19]. Moreover, congenital heart disease is a rare cause of LBBB [20, 21], which fits with the single case in our small series.

Of the asymptomatic LBBB patients who had *abnormal* echos, CMR not only confirmed the diagnosis of reduced cardiac function, but also provided additional clinically relevant information such as typical patterns of myocardial fibrosis or myocardial perfusion abnormality. The presence of myocardial mid-wall fibrosis has been associated with a poorer clinical outcome in DCM patients [22]. CMR also offered a different diagnosis in 24% of asymptomatic LBBB patients who had abnormal echos, half of which had a normal CMR scan. This is in keeping with previous studies showing CMR to be more accurate than 2-dimensional echocardiography for the assessment of ventricular volumes and function in both normal subjects and patients with LV hypertrophy or reduced cardiac function [23].

To our knowledge, LBBB has only been examined by three CMR studies published in the literature. However, all those studies only focused on CMR tagging, assessing a small number of asymptomatic LBBB patients in one study [24] and symptomatic LBBB patients in the other 2 studies [25, 26]. In contrast, the present study involves the largest group of patients with asymptomatic LBBB examined with a state of the art clinical CMR protocol and therefore this is the first study to demonstrate the diagnostic value of CMR in asymptomatic patients with LBBB.

### Study limitations

This study has several limitations. First, the number of patients is small, despite being derived from a large clinical referral database of more than 3,500 studies. The prevalence of totally asymptomatic patients with LBBB in the general population is probably small and, in our study, patients with previously known causes of LBBB (e.g. CAD) were excluded. It is, however, important to phenotypically characterise those patients and detect even subclinical structural heart disease. Second, this is a retrospective study and is therefore susceptible to referral bias. While this study reflects real-world clinical CMR practice, large population studies are needed to confirm our findings. Third, the information on echo was mainly based on reports from the referring physicians and was not reviewed by the investigators. Fourth, only a small number of patients had adenosine perfusion as it was not the primary reason for referral for CMR. Consequently, the prevalence of CAD may have been underestimated. Finally, the exact duration of LBBB is unknown, which may have influenced the CMR findings. The study only examined a single time point and no follow-up imaging was performed.

# Conclusions

CMR provides a useful adjunct to echocardiography in the diagnostic work-up of asymptomatic patients with LBBB. The main advantage of CMR is its ability to provide myocardial tissue characterisation with and without the use of gadolinium contrast. According to our study, CMR detects sub-clinical cardiomyopathy in a third of asymptomatic patients with LBBB despite normal echocardiograms. In those with abnormal echocardiograms, CMR provides additional clinically relevant information in half of the patients, including a high prevalence of mid-wall fibrosis in patients with impaired LV function. Larger scale studies are needed to confirm our observations and assess the prognostic value of CMR in this cohort of patients.

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#### Conflict of interest None.

#### References

- Francia P, Balla C, Paneni F, Volpe M (2007) Left bundlebranch block–pathophysiology, prognosis, and clinical management. Clin Cardiol 30(3):110–115. doi:10.1002/ clc.20034
- Baldasseroni S, Opasich C, Gorini M, Lucci D, Marchionni N, Marini M, Campana C, Perini G, Deorsola A, Masotti G, Tavazzi L, Maggioni AP (2002) Left bundle-branch block is associated with increased 1-year sudden and total mortality rate in 5517 outpatients with congestive heart failure: a report from the Italian network on congestive heart failure. Am Heart J 143(3):398–405
- Schneider JF, Thomas HE Jr, Kreger BE, McNamara PM, Kannel WB (1979) Newly acquired left bundle-branch block: the Framingham study. Ann Intern Med 90(3): 303–310
- Schneider JF, Thomas HE Jr, McNamara PM, Kannel WB (1985) Clinical-electrocardiographic correlates of newly acquired left bundle branch block: the Framingham Study. Am J Cardiol 55(11):1332–1338
- Fahy GJ, Pinski SL, Miller DP, McCabe N, Pye C, Walsh MJ, Robinson K (1996) Natural history of isolated bundle branch block. Am J Cardiol 77(14):1185–1190
- Miller WL, Ballman KV, Hodge DO, Rodeheffer RJ, Hammill SC (2005) Risk factor implications of incidentally discovered uncomplicated bundle branch block. Mayo Clin Proc 80(12):1585–1590
- Zannad F, Huvelle E, Dickstein K, van Veldhuisen DJ, Stellbrink C, Kober L, Cazeau S, Ritter P, Maggioni AP, Ferrari R, Lechat P (2007) Left bundle branch block as a risk factor for progression to heart failure. Eur J Heart Fail 9(1):7–14. doi:10.1016/j.ejheart.2006.04.011
- Bouzas-Mosquera A, Peteiro J, Alvarez-Garcia N, Broullon FJ, Garcia-Bueno L, Ferro L, Perez R, Bouzas B, Fabregas R, Castro-Beiras A (2009) Prognostic value of exercise echocardiography in patients with left bundle

branch block. JACC Cardiovasc Imaging 2(3):251–259. doi:10.1016/j.jcmg.2008.11.014

- Geleijnse ML, Vigna C, Kasprzak JD, Rambaldi R, Salvatori MP, Elhendy A, Cornel JH, Fioretti PM, Roelandt JR (2000) Usefulness and limitations of dobutamine-atropine stress echocardiography for the diagnosis of coronary artery disease in patients with left bundle branch block. A multicentre study. Eur Heart J 21(20):1666–1673. doi:10.1053/euhj.1999.2008
- Ghostine S, Caussin C, Daoud B, Habis M, Perrier E, Pesenti-Rossi D, Sigal-Cinqualbre A, Angel CY, Lancelin B, Capderou A, Paul JF (2006) Non-invasive detection of coronary artery disease in patients with left bundle branch block using 64-slice computed tomography. J Am Coll Cardiol 48(10):1929–1934. doi:10.1016/j.jacc.2006.04.103
- Einstein AJ (2009) Medical imaging: the radiation issue. Nat Rev Cardiol 6(6):436–438. doi:10.1038/nrcardio.2009.53
- Flett AS, Westwood MA, Davies LC, Mathur A, Moon JC (2009) The prognostic implications of cardiovascular magnetic resonance. Circ Cardiovasc Imaging 2(3): 243–250
- Karamitsos TD, Francis JM, Myerson S, Selvanayagam JB, Neubauer S (2009) The role of cardiovascular magnetic resonance imaging in heart failure. J Am Coll Cardiol 54(15):1407–1424
- Rudolph A, Abdel-Aty H, Bohl S, Boye P, Zagrosek A, Dietz R, Schulz-Menger J (2009) Noninvasive detection of fibrosis applying contrast-enhanced cardiac magnetic resonance in different forms of left ventricular hypertrophy relation to remodeling. J Am Coll Cardiol 53(3):284–291. doi:10.1016/j.jacc.2008.08.064
- Al-Mallah M, Kwong RY (2009) Clinical application of cardiac CMR. Rev Cardiovasc Med 10(3):134–141
- 16. Karamitsos TD, Arnold JR, Pegg TJ, Cheng AS, van Gaal WJ, Francis JM, Banning AP, Neubauer S, Selvanayagam JB (2009) Tolerance and safety of adenosine stress perfusion cardiovascular magnetic resonance imaging in patients with severe coronary artery disease. Int J Cardiovasc Imaging 25(3):277–283. doi:10.1007/s10554-008-9392-3
- Karamitsos TD, Hudsmith LE, Selvanayagam JB, Neubauer S, Francis JM (2007) Operator induced variability in left ventricular measurements with cardiovascular magnetic resonance is improved after training. J Cardiovasc Magn Reson 9(5):777–783. doi:10.1080/10976640701545073
- Bayes-Genis A, Lopez L, Vinolas X, Elosua R, Brossa V, Camprecios M, Mateo M, Cinca J, Bayes de Luna A (2003) Distinct left bundle branch block pattern in ischemic and non-ischemic dilated cardiomyopathy. Eur J Heart Fail 5(2):165–170
- Imanishi R, Seto S, Ichimaru S, Nakashima E, Yano K, Akahoshi M (2006) Prognostic significance of incident complete left bundle branch block observed over a 40-year period. Am J Cardiol 98(5):644–648. doi:10.1016/ j.amjcard.2006.03.044
- Boyle DM, Fenton SS (1966) Left bundle branch block. Ulster Med J 35(1):93–99
- Dizadji H, Tahmooressi P, Cernock WF (1974) Etiology of left bundle branch block. Hemodynamic and angiographic studies. J Electrocardiol 7(3):221–226
- 22. Assomull RG, Prasad SK, Lyne J, Smith G, Burman ED, Khan M, Sheppard MN, Poole-Wilson PA, Pennell DJ

(2006) Cardiovascular magnetic resonance, fibrosis, and prognosis in dilated cardiomyopathy. J Am Coll Cardiol 48(10):1977–1985. doi:10.1016/j.jacc.2006.07.049

- 23. Grothues F, Smith GC, Moon JC, Bellenger NG, Collins P, Klein HU, Pennell DJ (2002) Comparison of interstudy reproducibility of cardiovascular magnetic resonance with two-dimensional echocardiography in normal subjects and in patients with heart failure or left ventricular hypertrophy. Am J Cardiol 90(1):29–34
- 24. van der Land V, Germans T, van Dijk J, Zwanenburg JJ, Spreeuwenberg M, Marcus JT, Kamp O, Gotte MJ, van Rossum AC (2007) The effect of left bundle branch block on left ventricular remodeling, dyssynchrony and deformation of the mitral valve apparatus: an observational cardiovascular

magnetic resonance imaging study. Int J Cardiovasc Imaging 23(4):529–536. doi:10.1007/s10554-006-9187-3

- 25. Han Y, Chan J, Haber I, Peters DC, Zimetbaum PJ, Manning WJ, Yeon SB (2010) Circumferential myocardial strain in cardiomyopathy with and without left bundle branch block. J Cardiovasc Magn Reson 12:2. doi:10.1186/ 1532-429X-12-2
- 26. Rutz AK, Manka R, Kozerke S, Roas S, Boesiger P, Schwitter J (2009) Left ventricular dyssynchrony in patients with left bundle branch block and patients after myocardial infarction: integration of mechanics and viability by cardiac magnetic resonance. Eur Heart J 30(17):2117–2127. doi:10.1093/eurheartj/ehp212