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Case Report
LBBB

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4Dimensional XStrain Echocardiography assessment of Left Bundle Branch Cardiomyopathy in an elderly male: A case report

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Left bundle branch block (LBBB) is generally associated with a poorer prognosis in comparison to normal intraventricular conduction. LBBB may be the first manifestation of a more diffuse myocardial disease. The typical surface ECG feature of LBBB is a prolongation of QRS above 0.11s in combination with a delay of the intrinsic deflection in leads V5 and V6 of more than 60 ms and no septal q waves in leads I, V5, and V6 due to the abnormal septal activation from right to left. LBBB may induce abnormalities in left ventricular performance due to abnormal asynchronous contraction patterns. Asynchronous electrical activation of the ventricles causes regional differences in workload which may lead to asymmetric hypertrophy and left ventricular dilatation, especially due to increased wall mass in late-activated regions, which may aggravate preexisting left ventricular pumping performance or even induce it.

Keywords:

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Introduction

In the left bundle branch block (LBBB), the ventricles are activated sequentially with alterations in left ventricular mechanics, perfusion, and workload resulting in cardiac remodelling. Underlying molecular, cellular, and interstitial changes manifest clinically as changes in the size, mass, geometry, and function of the heart. Cardiac remodelling is associated with progressive ventricular dysfunction, arrhythmias, and impaired prognosis. Clinical and diagnostic notions about LBBB have evolved from a simple electrocardiographic alteration to a critically important finding affecting the diagnostic and clinical management of many patients. Advances in cardiac magnetic resonance imaging have significantly improved the assessment of patients with LBBB and provided additional insights into the pathophysiological mechanisms of left ventricular remodelling. We are presenting an interesting case report of LBBB cardiomyopathy in an octogenarian who was referred to us for a detailed colour echocardiographic assessment.

LBBB-ECG Criteria

Table 1: ECG criteria to define complete LBBB in adults

Electrocardiographic Criterion	AHA/ACCF/HRS [2]	Strauss et al [3]
QRS duration	>120 ms	>140 ms (men), >130 ms (women)
Left-sided leads (I, aVL, V5, V6)	Broad notched or slurred R waves	Broad notched or slurred R waves*
	Absent Q waves (with the possible exception of aVL)	
	Occasional RS pattern in V5 and V6	
Right-sided leads (V1, V2, V3)	Small initial r waves in V1-3	Broad notched or slurred mid-QRS*
		QS or rS in leads V1 and V2
R peak time	>60 ms in V5 and V6 but can be normal in V1-3	Not specifically mentioned
ST and T waves	Usually opposite in direction to QRS	Not specifically mentioned
	Positive concordance (uprightT wave with upright QRS)may be observed	

The American Heart Association (AHA), American College of Cardiology Foundation (ACCF), and Heart Rhythm Society (HRS) recommended an updated set of definitions for cardiac conduction disturbances in 2009 and 2018 [1, 2].

Strauss et al [3] then proposed more stringent criteria for LBBB to better predict cardiac resynchronization therapy (CRT) responders. This was motivated by the observation that patients with true LBBB—as opposed to those with conduction delay—were more likely to have more pronounced QRS durations with evidence of slurring and notching.

The table 1 compares and contrasts the electrocardiographic criteria defining LBBB.

ACCF indicates American College of Cardiology Foundation; AHA, American Heart Association; HRS, Heart Rhythm Society; and LBBB, left bundle branch block.

*≥2 leads (I, aVL, V_1, V_2, V_5, V_6).

Anatomy of the cardiac conduction system, left bundle branch (LBB), LBBB and the ECG criteria are exhibited in Figures 1-5.

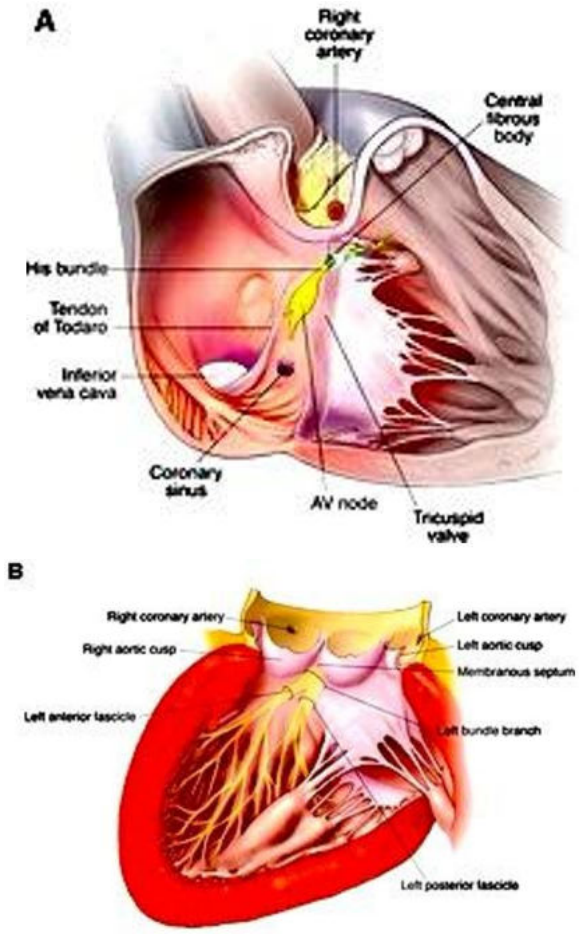


Figure 1: *Anatomy of the cardiac conduction system and its relation to surrounding structures .*

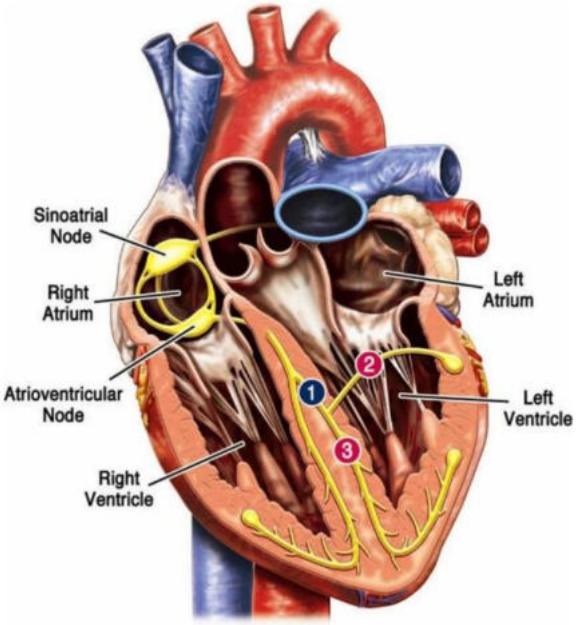


Figure 2: *Anatomy of LBB The left bundle branch comprises the main left bundle and distal anterior and posterior fascicles. LBBB from an incident disease requires a lesion just distal to the bundle of His (1) or extensive myocardial damage involving a large portion of the distal conduction system, including both the fascicles (2 and 3).*

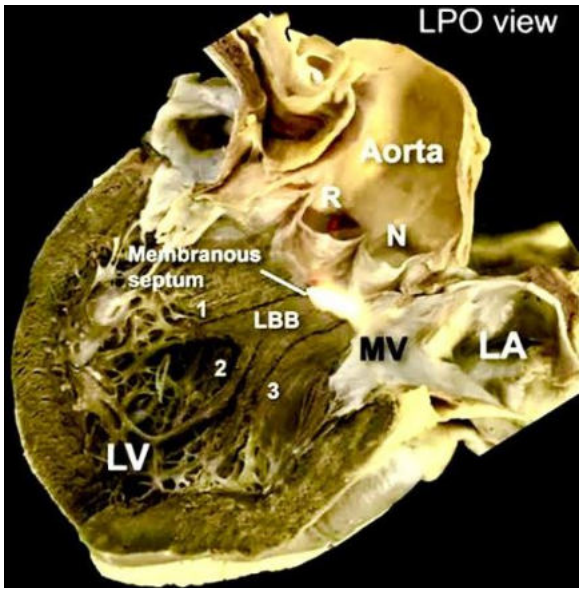


Figure 3: *4D tracking of the anatomy of the LBB The Left Posterior Oblique (LPO) view shows the trans-illuminated membranous septum located inferior to the interleaflet triangle between the right (R) and noncoronary (N) sinus of the aortic valve. Note that we have highlighted in dark color the limits of the endocardial position*

Of the left bundle branch (LBB) of His and its 3 fascicles, the left anterior fascicle (1), the left septal or middle fascicle (2), and the left posterior fascicle (3). LPO ¼ left posterior oblique.

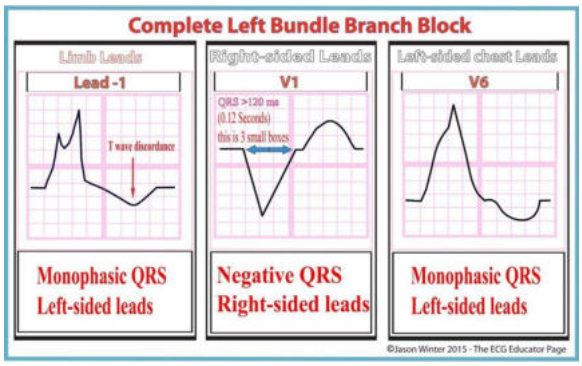


Figure 4: *Diagrammatic representation of the ECG criteria of LBBB.*

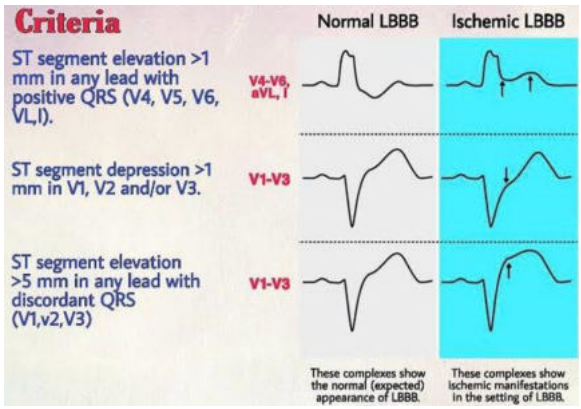


Figure 5: *Diagramatic representation of ECG manifestations of ischemia on LBBB.*

The prevalence of left bundle branch block (LBBB) in the general population ranges from approximately 0.1% to 1.0%, with the incidence increasing with age [4-6]. LBBB is strongly associated with structural heart and/or coronary artery disease [4, 6-8]. Patients with a newly recognized LBBB are at increased risk of cardiovascular events including heart failure, myocardial infarction, and sudden death [4, 7]. The evaluation of patients with incidental, newly recognized LBBB therefore, necessitates assessments for structural heart disease and coronary artery disease (CAD) in appropriate candidates [2]. Clinical and experimental data support that dyssynchronous left ventricular (LV) contraction (ie, early septal activation with delayed lateral wall contraction) itself may lead to a decline in LV systolic function [9, 10]. In patients with LBBB and a reduced LV ejection fraction (LVEF),

Cardiac resynchronization therapy (CRT) improves survival and reduces heart failure hospitalizations [11]. Among patients treated with CRT, reports describe "super-responders" whereby LVEF normalizes with the resolution of heart failure symptoms [12]. These observations have led to the notion that LBBB with resultant dyssynchrony may play a causative role in the development or progression of LV systolic dysfunction. This noteworthy syndrome is now commonly designated "dyssynchrony cardiomyopathy" or "LBBB-associated cardiomyopathy" [13, 14].

The relationship between left bundle branch block (LBBB) and dilated cardiomyopathy is well known. Isolated LBBB can also be seen in individuals with a structurally normal heart. Although LBBB confers increased mortality risk in elderly patients and those with underlying structural heart disease, it has minimal effects on younger healthy individuals [4, 15, 16]. However, chronic LBBB has been known to result in asynchronous LV contraction and subsequent impairment in LV function. Several studies have suggested a causative link between LBBB and chronic LV dilation, dysfunction, and heart failure [12, 17, 18].

The relationship between LBBB and LV dysfunction is complex and poorly understood. It may appear during the course of the disease indicating the severity and poor prognosis or it may play a causative role in the development of dyssynchronous contraction and worsening of LV function.

Case Report

An 82-year-old octogenarian gentleman afflicted with LBBB cardiomyopathy was referred to us for detailed color echocardiography. He was also suffering from multiple comorbidities: (i) coronary artery disease with percutaneous coronary intervention (PCI) and stenting performed to the proximal left anterior descending artery and right coronary artery in 2018 (ii) diabetes mellitus type 2 on oral hypoglycemic agents (iii) hypertension (iv) osteoarthritis of both knees.

On clinical examination, he was frail and weak. His height was 165 cm weight was 50 kg, pulse rate was 65/min, BP 120/80 in the right arm, in sitting position, SP02 97% at room air and respiratory rate 15/min.

There was no evidence of dyspnea at rest or tachypnea. Cardiovascular and systemic examinations were unremarkable.

Resting ECG revealed normal sinus rhythm with a rate of 78/min, regular and a LBBB pattern (QRS width 140 msec, QRS axis-60°) (Figure 6)

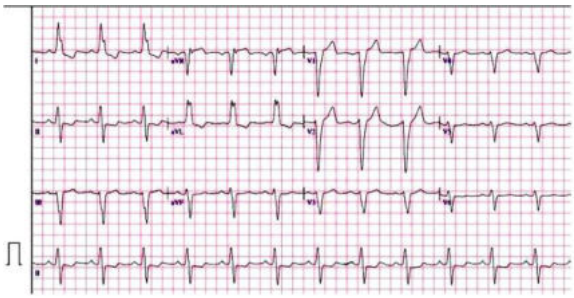


Figure 6: *Resting ECG consistent with LBBB.*

4Dimensional XStrain Echocardiography

All echocardiographic evaluations were performed by the author, using my Lab X7 4D XStrain echocardiography machine, Esaote, Italy. The images were acquired using a harmonic variable frequency (1-5 Mhz) electronic single-crystal array transducer while the subject was lying in the left lateral decubitus position.

Conventional M-mode, two-dimensional and pulse wave doppler (PWD) echocardiography was performed from parasternal long-axis, short axis and apical four-chamber views and the following parameters were derived: interventricular septum and LV posterior wall thickness in end-diastole and end-systole (IVS d and LVPW d, respectively), LV internal dimension at end-diastole and end-systole (LVID d and LVID s, respectively), LV end-diastolic and end-systolic volumes (LVEDV and LVESV respectively), ejection fraction (EF%), LV Mass in diastole (LV Mass d), Cardiac Output (CO) and Cardiac Index (CI).

4D XStrain speckle tracking echocardiography

From the apical position, 2Dimensional cine loops were acquired from two-chamber, three-chamber and four-chamber views. High-quality ECG signal was a must for proper gating and a minimum of three cardiac cycles were acquired for each cine loop. The study was performed with a frame rate between 40-75 fps and then stored digitally on a hard disk for offline analysis by software package XStrain™ advanced technology TOMTEC GMGH 3D/4D rendering Beutel™ computation capabilities [19].

The LV bull's eye depiction according to the 17-segment model was generated by XStrain 4D software, by integrating the results of each set of cine loops [20, 21]. The unique software provided segmental, regional and global peak systolic values of various LV strains. Moreover, XStrain-4D software created a 3D reconstruction for calculating LV volumes and EF [20], and XStrain 4D-EF by the "Beutel Mode" method (TOMTEC, Germany) [22].

After the extensive echocardiography assessment, the following data was obtained, as mentioned below:

Transthoracic Echocardiography			
M-Mode - at the level of the mitral valve			
DE Amp			
DE Amp	0.10 m/s		
EPSS	9.9 mm		
E-F Slope	150 ms		
M-Mode - at the LV level			
Left Ventricle			
IVSd	13.0 mm	IVSS	13.8 mm
LVPWd	8.8 mm	LVPWS	16.1 mm
LVIDS	34.0 mm	%LV FS	20%
EF	41%	LVEDV index	52.7 ml/m2
LVEDV	80.6 ml	LVESV index	31.1 ml/m2
LVESV	47.6 ml	SV	33.0 ml
CI	1.32 l/min/m2	CO	2.02 l/min
%PW	83%	% IVS	6%
LV Mass index	103 g/m2	LV Mass	158 g
LVIDd	42.5 mm	Relative Wall Thickness	0.41
2 Dimensional Echocardiography			
Auto EF - Biplane			
LVAd A4C	33.58 cm2	LVAd index A4C	21.9 cm2/m2
LVAS A4C	26.98 cm2	LVAd A2C	32.76 cm2
LVAd index A2C	15.0 cm2/m2	LVAS A2C	23.01 cm2
LVEDV (MOD A4C)	111.0 ml	LVESV (MOD A4C)	76.9 ml
LVEDV (MOD A2C)	113.3 ml	LVESV (MOD A2C)	69.5 ml
LVEDV (MOD BP)	115.1 ml	LVESV (MOD BP)	77.8 ml
LVEDV index (MOD A4C)	72.5 ml/m2	LVEDV index (MOD BP)	75.2 ml/m2
EF (MOD A4C)	31%	EF (MOD A2C)	39%
EF (MOD BP)	32%		

4Dimensional XStrain Echocardiography	
Volumetric data	
LVEDV	90.01 ml
LVESV	59.90 ml
EF	33.46%
CO	1.92/min
Sph i d	0.48
Sph i s	0.46

Dimensional XStrain Speckle Tracking Echocardiography	
Regional GLS	
Bas Ant	-18.54%
BasAntSep	-10.66%
Bas Sep	-7.02%
Bas Inf	-23.51%
Bas Post	-25.61%
Bas lat	-13.46%
Mid Ant	-10.57%
MidAntSep	-2.24%
Mid Sep	-5.54%
Mid Inf	-13.48%
Mid Post	-19.80%
Mid Lat	-9.63%
Apic Ant	-5.87%
Apic Sep	-7.80%
Apic Inf	-3.91%
Apic lat	-10.56%
Apex	-5.51%
Global GLS	
Global Strain (A2C)	-12.04%
Global Strain (A4C)	-9.95%
Global Strain (ALAX)	-12.79%
Global Strain	-11.59%

Highlighting features

The illuminating features of 4Dimensional XStrain echocardiography are outlined (Figures 7-12):

1. Jerky motion of ventricular septum with severe dyssynchronised oscillatory movement of the LV.

2. Small LV with 4D EDV and 4D ESV being 90.01 ml and 59.90 ml respectively

3. Severely reduced LVEF:

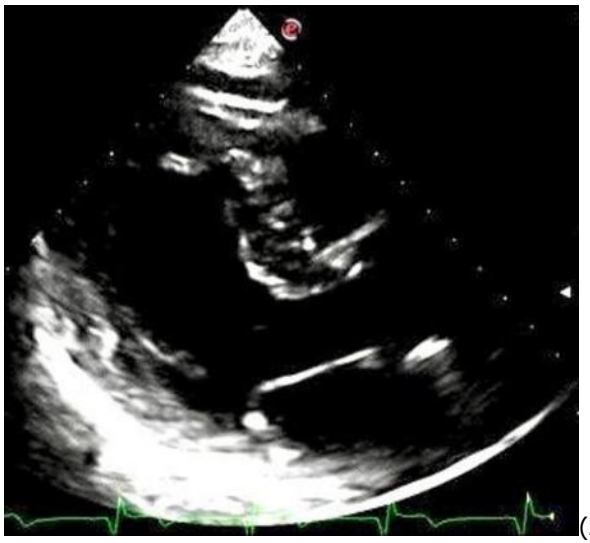
M-mode = 41%

2Dimensional Biplane Simpson's method=32%

4D = 33.46%

4. Severely impaired global longitudinal strain of LV.

Global Strain (A2C)	-12.04%
Global Strain (A4C)	-9.95%
Global Strain (ALAX)	-12.79%
Global Strain	-11.59%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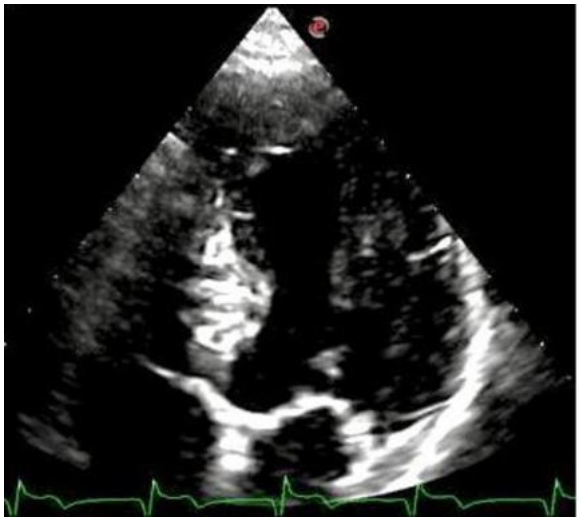
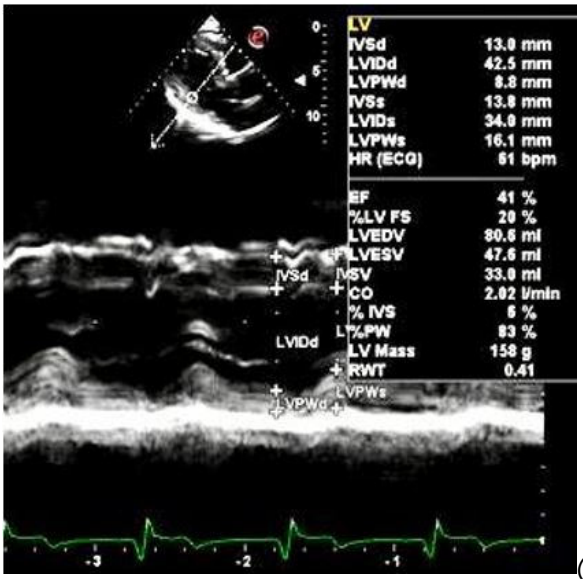
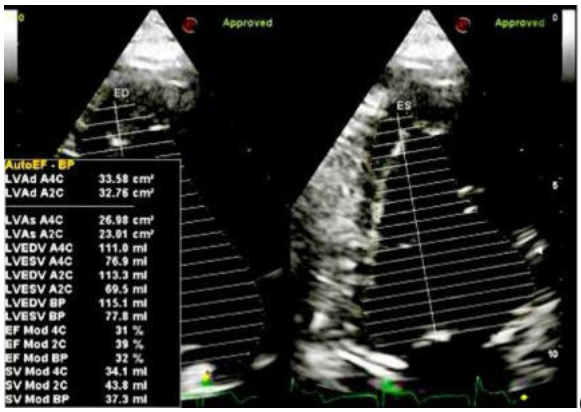

(B)

Figure 7: *Transthoracic Echocardiography (A) Lax view, (B) 4ch view.*

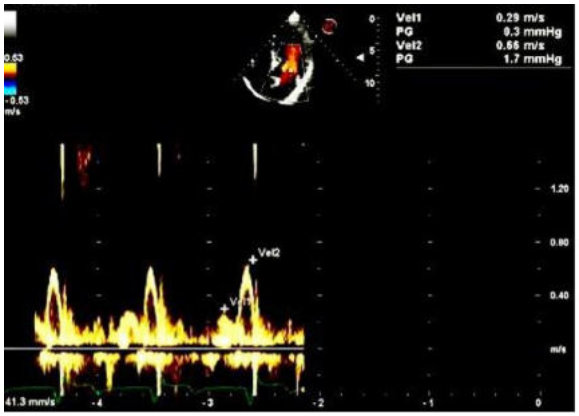


(A)



(B)

Figure 8: *Conventional M-mode and 2-Dimensional Transthoracic Echocardiography : (A) M-mode LV volumes and ejection fraction, (B) Biplane (Simpsons method) method for determining 2-Dimensional LV volumes and ejection fraction.*



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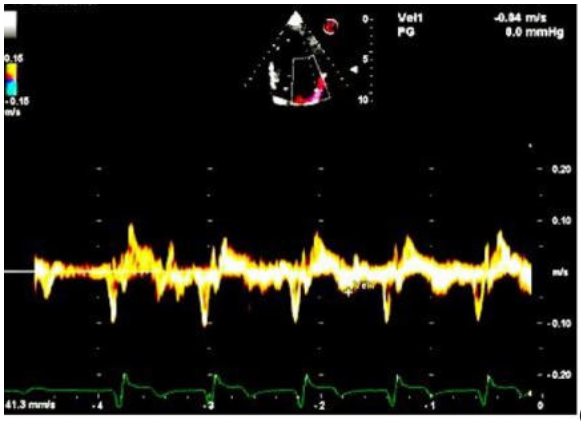


(B)

Figure 9: *(A) Pulse wave doppler across MV and (B) Tissue doppler imaging of lateral wall of LV.*

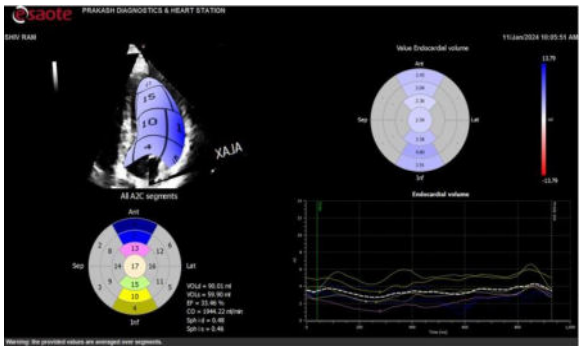
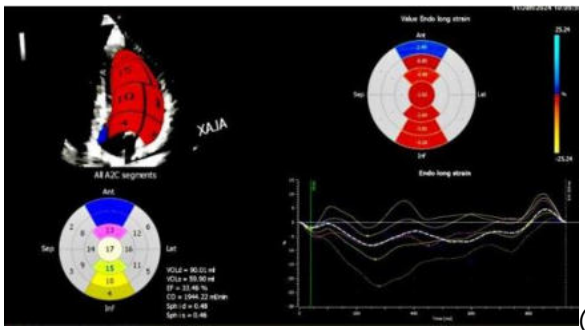


Figure 10: *LV endocardial volumes, 4D-EF%, cardiac output, and sphericity index.*



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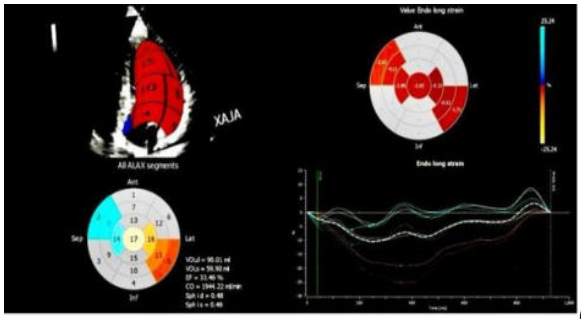

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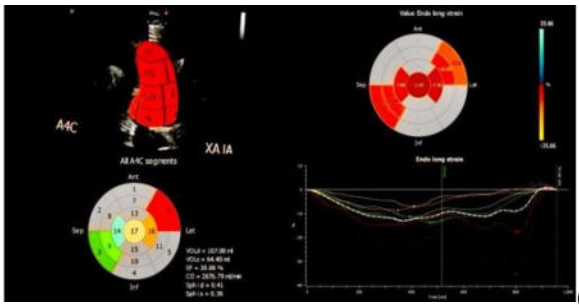
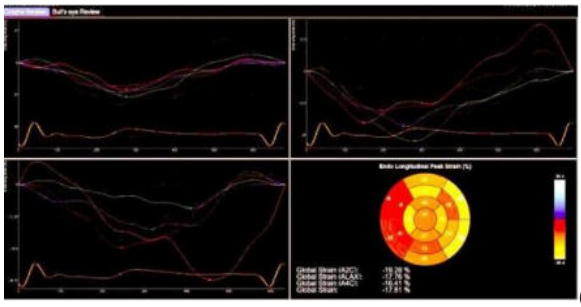


(C)



(D)

Figure 11: *Apical 2CH,apical LAX, apical 4CH regional strain plots and curves (A) Apical 2CH regional strain (B) Apical LAX regional strain (C) Apical 4CH regional strain (D) Combined graphs and bull's eye mapping of global strain values.*

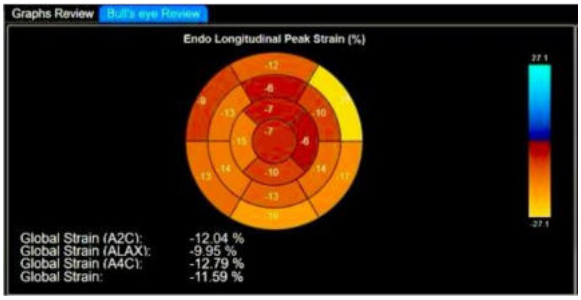


Figure 12: *Global Longitudinal peak strain (%) of LV.*

Discussion

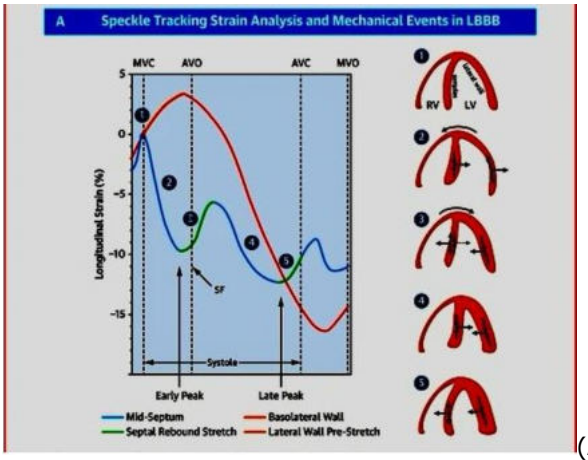
There is a 0.1% to 0.9% prevalence of LBBB in the general population, which increases with advanced age or with heart failure [23]. LBBB is an established cause of ventricular dyssynchrony due to delayed conduction within the ventricle and sometimes leads to hemodynamic deterioration. LBBB can be associated with the deterioration of LVEF because dyssynchronous ventricular activation leads to dyssynchronous contraction. Diastolic dysfunction can accompany this drop in LVEF because dyssynchrony leads to prolonged isovolumic contraction and relaxation times and thereby a shortened time for LV filling. Such deterioration in LV function, when accompanied by heart failure, provides a rationale for cardiac resynchronization therapy [24].

Speckle-tracking imaging is useful for detecting LBBB-related dyssynchrony, with a recent study showing that impaired GLS measured on two-dimensional speckle-tracking echocardiography has a significant and independent association with the occurrence of cardiovascular events in patients with LBBB. These results suggest that the measurement of GLS can provide better risk stratification than LVEF in patients with LBBB. Critical to recognizing LV dysfunction in LBBB are the phenomena of septal flash (early, rapid short septal inward motion) and apical rocking (a short-lived early systolic septal motion of the apex and a predominantly lateral motion during ejection) [25, 26]. Early systolic pressure generation in the septum prestretches the lateral wall, which is still relaxed. This augmented lateral preload leads to robust contraction and an outward motion of the septum in late systole.

Calle et al [27] assessed the value of echocardiographic strain analysis in patients with strict LBBB and the presence of septal flash. They observed an electromechanical continuum in patients with LBBB and septal flash, where specific strain patterns tightly correlated with the degree of LV remodelling and LV dysfunction, which supported the concept of LBBB-induced cardiomyopathy. The proposed strain-based classification might be valuable to assess the attributes of LBBB with the degree of adverse remodelling and LV dysfunction.

LBBB septal strain patterns. The mid-septal deformation curves were classified into 4 groups, based on the sequence and amplitude of septal shortening and stretching during LV systole (Figure 13 A). In stage LBBB-1, an early sigmoidal deflection (during QRS) is discerned, followed by late peak strain before aortic valve closure. The stage LBBB-2 and -3 septal strain patterns represent double-peaked or interrupted contraction patterns with 2 phases of systolic shortening, each one followed by re-lengthening. More specifically, in stage LBBB-2, an early small peak during QRS is followed by a larger dominant peak during ejection (before aortic valve closure). Conversely, in stage LBBB-3, a dominant early peak is followed by a smaller late peak. Stage LBBB-4 is characterized by early peak strain of the septum, followed by stretching during further systole without ejectional septal shortening.

Strain-based stages of LBBB-induced LV Remodeling. Based on the consistent gradient observed among echocardiographic parameters of cardiac remodelling in the LBBB-septal flash group, the different LBBB strain patterns here classified from LBBB-1 (minimal remodelling) to stage LBBB-4 (advanced remodelling) (Figure 13 B).



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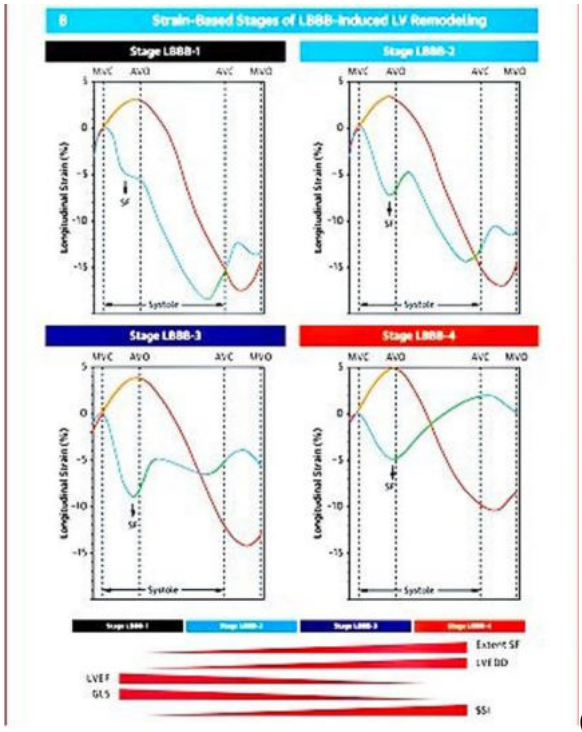

(B)

Figure 13: *Strain-Based Staging of Left Bundle Branch Block-Induced Left Ventricular Remodelling in Patients With Septal Flash (A) Speckle Tracking Strain Analysis and Mechanical Events in LBBB (B) Strain-Based Stages of LBBB-Induced LV Remodelling.*

Hwang et al [28] in their study of 269 patients (mean age of 69.5 $\pm$ 10.9 years) of LBBB on performing speckle tracking analysis detected significantly impaired global longitudinal strain (GLS). Additionally, in their study impaired GLS > -12.2% had a larger number of clinical events than those with preserved GLS.

Interestingly in our octogenarian patient, suffering from multiple comorbidities along with LBBB cardiomyopathy with severely reduced LV systolic function- (LVEF 32% by biplane simpson's method and 33.46% by 4D XStrain) there was striking impairment of GLS values on 4Dmienisonal XStrain speckle strain echocardiography.

Global Strain (2CH)	-12.04%
Global Strain (4CH)	-9.95%
Global Strain (3CH)	-12.79%
Global Strain	-11.59%

Therefore, the likelihood of an increased number of adverse clinical events is a strong possibility in our patients.

Conclusions

Patients with LBBB and severely reduced LVEF have remarkably poor long-term clinical outcomes that are significantly worse than those of patients with a similar LVEF but no conduction system disease. The high level of mortality and adverse events seen in this group may suggest a patient population that needs and would potentially benefit from resynchronization therapy.

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