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Assessment of Left Ventricular Asynchrony after Insertion of Dual Chamber Pacemaker by Using Two-Dimensional Speckle Tracking Echocardiography

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ABSTRACT

Background: Right ventricular pacing for the treatment of various conduction disorder, is associated with effects on left ventricular function leading to reduction in the left ventricular ejection fraction and causing pacing induced cardiomyopathy over time. **Objective:** To study the effects of dual chamber pacemaker on left ventricular function using 2D speckle tracking echocardiography. **Patients and Methods:** The present study was conducted on a cohort of 53 patients with conduction disturbances who underwent dual chamber pacemaker implantation at the Cardiology department of Tanta University Hospital. The study period spanned from December 1, 2021 to May 1,2022. Prior to pacemaker implantation, 2D Speckle tracking echocardiography was performed. Subsequent assessments were conducted at one week and three months intervals post implantation. **Results:** Our results demonstrated that at 3 months post pacemaker implantation (10) patients developed PIVD; 2 of them developed PICM. At one week follow up, GLS was significantly lower in patients who developed PIVD, compared to those who did not. Speckle tracking echocardiography provides angle-independent accurate measurements of LV strain. Global longitudinal strain at 1 week post pacemaker implantation can predict subsequent decline in LV contractility and ejection fraction, hence can be used to identify patients at risk to develop pacemaker induced ventricular dysfunction or cardiomyopathy. **Conclusion:** 2D-speckle tracking can be used as early predictor way for pacemaker induced ventricular dysfunction as well as pacemaker induced cardiomyopathy in patients who went dual chamber pacemaker.

Keywords: Left ventricular, electrocardiogram, conduction disorder, cardiomyopathy.

Introduction

Permanent cardiac pacing is the most efficient treatment for a variety of conduction disorders including high degree atrio-ventricular block and symptomatic sick sinus syndrome (Aste and Brignole, 2017).

One of the most common cardiac pacing mode is DDD, which is known as physiological pacing because it maintains atrioventricular synchrony but will affect ventricular synchrony (Reddy *et al.,* 2023).

In Dual chamber pacemaker there is isolated right ventricular (RV) pacing activates the interventricular septum before the left ventricular (LV) lateral wall, seen as a left bundle branch block pattern on the electrocardiogram (ECG) due to propagation of the electrical wave away from the sternum. This results in LV dyssynchrony and mismatched timing between chamber walls, with deleterious effects on LV function and adverse clinical outcomes, including heart failure, which could be assessed by a lot of modalities using Echocardiography (Song *et al.,* 2020).

In this study we will use two-dimensional (2D) speckle tracking strain to detect whether DDD pacing will affect LV function or not (Yaseen *et al.,* 2022).

Two-dimensional (2D) speckle tracking strain imaging allows angle-independent and multidirectional assessment of LV mechanics and function (Gunasekaran *et al.,* 2017).

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Using this technique, various important aspects can be assessed.LV mechanical dyssynchrony: calculated by measuring differences in the timing of peak systolic strain of various LV segments (Abawi *et al.,* 2022).

LV strain: representing LV systolic function; may detect more subtle regional changes as compared to conventional measurements of LV systolic function (Tops *et al.,* 2017).

The aim of this work is to assess if the use of 2D speckle tracking echocardiography would be an indicator of pacemaker induced ventricular dysfunction and pacemaker induced cardiomyopathy in patient who underwent dual chamber pacemaker.

Patients and Methods

The present study was conducted on a cohort of 53 patients with conduction disturbances who underwent dual chamber pacemaker implantation at the Cardiology department of Tanta University Hospital. The study period spanned from December 1, 2021 to May 1, 2022. Prior to pacemaker implantation, 2D Speckle tracking echocardiography was performed. Subsequent assessments were conducted at one week and three months intervals post implantation.

The patients were then divided into three groups according to the decline in ejection fraction (non - significant decline, pacemaker induced ventricular dysfunction (PIVD) and pacemaker induced cardiomyopathy (PICM). An informed written consent was obtained from all participants in the study after explanation of the benefits and possible risks of the study and how we will overcome these risks should they happened. There was a code number for each patient's files that included all investigations, so all data of the patients was strictly confidentially, and privacy was granted.

Inclusion criteria

All men and women above 18 years of age with conduction disturbances necessitating the implantation of dual chamber ventricular pacemaker.

Exclusion criteria

Structural heart abnormality including LV dilatation or LVEF<50%. Myocardial infarction or revascularization within prior 6 months. Significant valvular heart disease (starting from moderate in severity). Significant respiratory diseases. All other co morbidities that may itself cause LV remodeling including previous. Patient with chronic kidney eGFR<30 or patients on dialysis. Patient with thyrotoxicosis. Patient on current chemotherapy with cytotoxic drugs.

Type of interventions

All cases included in this study will be subjected to the following after obtaining an informed consent: Full history taking: Age and gender. Assessment of cardiovascular risk factors: Hypertension: patient was considered hypertensive if they were on antihypertensive drugs or their blood pressure was more than 140/90 (Jordan *et al.,* 2018). Diabetes. Smoking. History of concomitant disease (thyroid disease). Any medications the patient is taking for cardiac or non-cardiac purposes. Symptoms of cardiovascular disease were assessed, e.g. Dyspnea defined by American Thoracic society as a subjective experience of breathing discomfort that compromises qualitatively distinct sensation varying in intensity (Pesola and Ahsan, 2016). Dizziness. Presyncope. Syncope, which was defined as sudden transient complete loss of consciousness and postural tone resulting from global cerebral hypoperfusion with spontaneous complete recovery and without sequalae (Goldberger *et al.,* 2019). Oliguria: is defined as urine output less than 0.5 ml/kg/h lasting for at least 6 hours (Egal *et al.,* 2016). Altered consciousness. Complete clinical examination: Pulse rate, regularity, and blood pressure. Auscultation of heart and lungs. Palpation and inspection of chest wall and back. Laboratory investigations (complete blood picture {CBC}, random glucose level, renal function tests {urea/creatinine}, thyroid stimulation hormone {TSH}, prothrombin time {PT} and international normalized ratio {INR}. Electrocardiogram (ECG): 12 lead ECG recorded at paper speed of 25mm/s and a gain of 10 mm/mv. Echocardiography

An echocardiogram was done using vivid – E9 echocardiography (GE) medical system equipped with M5S probe (frequency 1,7-3.3) MHz. Examinations was performed in the left lateral decubitus position by an experienced operator. Apical (4,3, and 2 chamber views) and parasternal views were acquired at end expiration at frame rates 60-110. Three complete cardiac cycles were taken and stored in cine loop format. left ventricular end-systolic and end-diastolic diameters (LVESD and LVEDD)

measured according to Simpson model. the data was stored in digital format and transferred to Echo Pac for analysis by another experienced operator using Echo Pac 110.1.2. An echocardiogram was done for each patient at baseline, and at 1 week and 3 months post implantation.

Pacemaker induced ventricular cardiomyopathy (PICMP) was defined as a reduction in LVEF to <45% & pacemaker induced ventricular dysfunction was defined as a reduction in LVEF to<50%.

For the strain analysis, the endocardial border in the end systolic frame was manually traced from the apical four – chamber, two- chamber and long axis views with a region of interest drawn to include the myocardium. manual adjustment using a point and click approach was enabled to ensure that endocardial and epicardial borders were included. the software then finally generated time domain LV strain profiles for each of the six segments of each view, from which end-systolic strain was measured. Global longitudinal strain values of the 18 LV segments. Intervention: Patient education prior to pacemaker implantation: All patients were subjected to education about the nature of the bradycardia condition, all possible treatment options, the nature of the pacemaker system, the technique of implantation. Antiplatelet and anticoagulant drugs were stopped according to each individual condition.

For each patient, the dual chamber pacemaker was implanted by the following technique: (Barold *et al.,* 2010).

First an IV cannula was placed in the left arm in case venography needed tize the venous anatomy. Prophylactic antibiotics were given 1 h prior implantation. The local anesthetic was then given along the length of the intended pocket, according to the guidelines which suggest a maximum 3 mg/kg of 1% lignocaine. A left sided approach was done for most of patient as they were right-handed and right sided approach was done for patient with left-handed dominance. subclavian approach vein access was chosen. the access needle, attached to a 10-ml syringe containing a few millimeters of saline was introduced. the landmark for entry was junction between the medial and middle third of the clavicle where the tip of the needle bevel down and directed toward a point just above the sternal notch guided by fluoroscopy. at the point of meeting the clavicle, angle of entry with respect to the thorax would then be increased to pass beneath the clavicle. the needle was then advanced under negative pressure until blood is aspirated upon vein entry. A guidewire was then advanced under fluoroscopy to the inferior vena cava then we do another puncture medial or lateral to the first one according to anatomy then another guidewire was advanced under fluoroscopy to the inferior vena cava then we use the medial wire for right ventricular lead & lateral wire for right atrial lead. A 4-5 cm horizontal or oblique incision was then made, the horizontal incision is a cut made a proximality 1-2 cm below the junction of the middle and lateral thirds of the clavicle. this varied according to different patient circumstances. The subcutaneous pocket was then made in the pectoral region. Once the subcutaneous plane was reached, one or two fingers were advanced to separate the tissues apart medially and caudally. Along the medial guide wire, a peel-away sheath dilator combination (7 Fr) was advanced, and the wire withdrawn. The right ventricular lead was then advanced through the peel away into right atrium crossing the tricuspid valve to enter the RV. the direct crossing technique was first attempted with the stylet reshaped and advanced with some adjustments and rotation aiming to point the tip towards the tricuspid valve and into the RV. Once across, the stylet was gently withdrawn to allow the tip toward to fall towards the apex and obtain the best readings. If the high septum was desired position, then the lead would advance across the valve with the stylet advanced throughout the entire lead. If this technique failed, then we proceed to prolapsing the lead across the TV. Here the lead was advanced with the stylet withdrawn 5-10 cm into the RA aiming for the tip to catch on the annulus, where the lead was advanced to create a loop in the RA. the curved stylet then removed and the straight advanced prolapsing portion of the lead through the tricuspid valve then the tip jumping through the valve into RV. Anteroposterior and lateral position views were taken to confirm the presence of the lead in the RV and not the coronary sinus. The testing cable would then be connected to obtain pacing parameters with the black clip on the distal ring and the red on the proximal ring of the bipolar leads. after checking for the current of injury, sensing and impedance values were checked. pacing threshold testing was then done to ensure appropriate values. if the values were not satisfying values, then we would seek alternate lead position until reaching satisfying values and screws deployed to fix the leads. Peeling the peel away sheath was then done; sometimes even earlier in the procedure if there was significant bleeding. The next step we would suture the collar to the underlying muscle using non-absorbable sutures. Then we removed the stylet, which also served as stability testing for the lead. Along the lateral guide wire, a peel-away sheath dilator combination (7 Fr) was advanced, and the wire withdrawn. Then the right atrium lead was

then advanced through the peel away into inferior vena cava through the straight stylet. Then we exchange the straight stylet with J shaped stylet while pulling genteelly the RA lead till stylet reaching the tip of the lead giving J shaped to the end of the lead with slight manipulation was then applied to achieve the pendular movement of the lead, screws deployed to fix the leads. Peeling the peel away sheath was then done; sometimes even earlier in the procedure if there was significant bleeding. The next step we would suture the collar to the underlying muscle using non-absorbable sutures, then we removed the stylet gently under fluoroscopy, which also served as stability testing for the lead. after which each lead is connected to its specific socket at the pulse generator then using the screw to fix them inside the battery then placed in the pocket. Another suture was taken to secure the battery in the underlaying muscle then wound was closed. the subcutaneous tissue was closed by absorbable sutures in a simple interrupted suture manner, then the skin by non – absorbable sutures also by simple interrupted manner. The patient was then educated not to jerky movement with his left arm. the next day, an X -ray would be done to ensure lead position and programmed before discharge. Oral antibiotics was prescribed. and the patient discharged to return after 10 days for suture removal, wound checking, and programming.

Results

Table 1: Distribution of the studied cases according to demographic data (n = 53)

	No.	%
Gender		
Male	16	30.2
Female	37	69.8
Age (years)		
Min. – Max.	19.0 – 85.0	
Mean ± SD.	67.13 ± 11.95	
Median (IQR)	69.0 (65.0 – 73.0)	

Table 2: Comparison between the two studied groups according to gender & age:

	Normal (n = 41)		Decline (n = 12)		Test of sig.	p
	No.	%	No.	%		
Gender						
Male	10	24.4	6	50.0	χ^2=	^{FE}p=
Female	31	75.6	6	50.0	2.889	0.150
Age (years)						
Min. – Max.	19.0 – 85.0		45.0 – 75.0			
Mean ± SD.	66.61 ± 12.89		68.92 ± 8.16		t= 0.584	0.562
Median (IQR)	67.0 (62.0 – 75.0)		71.0 (68.0 – 73.0)			

IQR: **Inter quartile range** SD: **Standard deviation**
t: Student t-test **U: Mann Whitney test**
p:p value for comparing between the two studied groups

Table 3: Descriptive analysis of the studied cases according to clinical characteristics (n = 53)

	Min. – Max.	Mean ± SD.	Median (IQR)
Hb (g/dl)	10.0 – 15.0	12.11 ± 1.50	12.0 (11.0 – 13.0)
RBS	150.0 – 400.0	248.4 ± 72.81	230.0 (200.0 – 322.0)
WBCs	4000.0 – 10000.0	6388.1 ± 1599.4	6000 (5300 – 7699)
Urea	0.50 – 2.50	1.08 ± 0.44	1.0 (0.80 – 1.20)
INR	0.90 – 1.40	1.05 ± 0.10	1.0 (1.0 – 1.10)
eGFR	31.0 – 140.0	65.75 ± 25.33	59.0(41.0 – 71.50)

Table 4: Comparison between the two studied groups according to clinical characteristics:

Clinical characteristics	Normal (n = 41)	Decline (n = 12)	Test of Sig.	P
Hb (g/dl)				
Min. – Max.	10.0 – 15.0	10.0 – 15.0		
Mean ± SD.	12.11 ± 1.53	12.08 ± 1.44	t= 0.058	0.954
Median (IQR)	12.0 (11.0 – 13.0)	12.0 (11.0 – 13.0)		
RBS				
Min. – Max.	150.0 – 400.0	170.0 – 367.0		
Mean ± SD.	254.7 ± 77.74	226.9 ± 49.31	U= 216.0	0.523
Median (IQR)	233.0(198.0 – 322.0)	222.0(200.0 – 235.0)		
WBCs				
Min. – Max.	4000.0 – 10000.0	4000.0 – 9000.0		
Mean ± SD.	6413.7 ± 1554.6	6300.7 ± 1814.9	U= 239.0	0.882
Median (IQR)	6000 (5321 – 7699)	5835 (4722 – 7850)		
INR				
Min. – Max.	0.90 – 1.40	0.90 – 1.20		
Mean ± SD.	1.06 ± 0.10	1.01 ± 0.08	t= 1.455	0.152
Median (IQR)	1.0 (1.0 – 1.10)	1.0 (1.0 – 1.0)		
eGFR				
Min. – Max.	31.0 – 140.0	31.0 – 86.0		
Mean ± SD.	68.41 ± 26.67	55.67 ± 18.17	U= 191.50	0.246
Median (IQR)	60.0(51.0 – 90.0)	57.0(41.0 – 71.50)		

IQR: **Inter quartile range** SD: **Standard deviation**
t: Student t-test **U: Mann Whitney test**
p: p value for comparing between the two studied groups

Table 5: Comparison between the two studied groups according to different comorbidity:

Comorbidity	Normal (n = 41)		Decline (n = 12)		χ^2	p
	No.	%	No.	%		
HTN						
No	15	36.6	4	33.3	0.043	^{FE}p= 1.000
Yes	26	63.4	8	66.7		
DM						
No	27	65.9	7	58.3	0.228	^{FE}p= 0.736
Yes	14	34.1	5	41.7		
Thyroid						
No	28	92.7	11	91.7	0.014	^{FE}p= 1.000
Yes	3	7.3	1	8.3		
Smoker						
No	32	78.0	8	66.7		
Smoker	4	9.8	3	25.0	1.961	^{MC}p= 0.412
Ex-smoker	5	12.2	1	8.3		

IQR: **Inter quartile range** SD: **Standard deviation**
t: Student t-test **U: Mann Whitney test**
p: p value for comparing between the two studied groups

Table 6: Comparison between the two studied groups according to EF.

EF	Normal (n = 41)	Decline (n = 12)	t	p
Baseline				
Min. – Max.	54.0 – 76.0	51.0 – 66.0		
Mean ± SD.	60.20 ± 4.56	58.0 ± 4.13	1.495	0.141
Median (IQR)	60.0 (57.0 – 64.0)	57.50 (56.0 – 60.0)		
1 week				
Min. – Max.	52.0 – 73.0	50.0 – 63.0		
Mean ± SD.	58.49 ± 4.75	56.33 ± 4.05	1.425	0.160
Median (IQR)	58.0 (55.0 – 62.0)	55.50 (53.0 – 59.50)		
3 months				
Min. – Max.	52.0 – 72.0	40.0 – 55.0		
Mean ± SD.	63.63 ± 4.01	47.67 ± 3.98	12.191*	<0.001*
Median (IQR)	63.0 (60.0 – 66.0)	48.50 (45.0 – 50.0)		

IQR: **Inter quartile range** SD: **Standard deviation**
t: Student t-test **U: Mann Whitney test**
p: p value for comparing between the two studied groups

Table 7: Comparison between Baseline and 3 months in decline group according to EF (n= 12)

EF	Baseline	3 months	t	p
Min. – Max.	51.0 – 66.0	40.0 – 55.0		
Mean ± SD.	58.0 ± 4.13	47.67 ± 3.98	**5.942***	**<0.001***
Median (IQR)	57.50 (56.0 – 60.0)	48.50 (45.0 – 50.0)		

Table 8: Comparison between the three studied groups according to EF

EF	Normal (n = 41)	PIVD (n = 10)	PICM (n = 2)	F	p
Baseline					
Min. – Max.	54.0 – 76.0	53.0 – 66.0	51.0 – 56.0		
Mean ± SD.	60.20 ± 4.56	58.90 ± 3.75	53.50 ± 3.54	2.401	0.101
Median (IQR)	60.0(57.0 – 64.0)	59.0 (56.0 – 60.0)	53.50 (51.0 – 56.0)		
1 week					
Min. – Max.	52.0 – 73.0	52.0 – 63.0	50.0 – 55.0		
Mean ± SD.	58.49 ± 4.75	57.10 ± 3.84	52.50 ± 3.54	1.870	0.165
Median (IQR)	58.0(55.0 – 62.0)	57.50 (54.0 – 60.0)	52.50 (50.0 – 55.0)		
3 months					
Min. – Max.	52.0 – 72.0	45.0 – 55.0	40.0 – 43.0		
Mean ± SD.	63.63 ± 4.01	48.90 ± 2.96	41.50 ± 2.12	84.538*	<0.001*
Median (IQR)	63.0 (60.0 – 66.0)	49.0 (47.0 – 50.0)	41.50 (40.0 – 43.0)		
Sig. bet. Grps.	$p_1<0.001^*, p_2<0.001^*, p_3=0.040^*$				

Table 9: Comparison between the two studied groups according to ESD

ESD	Normal (n = 41)	Decline (n = 12)	t	p
Baseline				
Min. – Max.	22.0 – 35.0	26.0 – 39.0		
Mean ± SD.	31.0 ± 4.22	33.25 ± 3.65	1.671	0.101
Median (IQR)	32.0 (29.0 – 34.0)	33.50 (31.0 – 35.50)		
1 week				
Min. – Max.	24.0 – 36.0	27.0 – 41.0		
Mean ± SD.	32.95 ± 3.24	35.25 ± 4.0	1.822	0.088
Median (IQR)	34.0 (32.0 – 35.0)	36.50 (32.0 – 38.0)		
3 months				
Min. – Max.	25.0 – 39.0	30.0 – 44.0		
Mean ± SD.	30.39 ± 3.58	37.67 ± 3.85	6.087*	<0.001*
Median (IQR)	29.0 (28.0 – 33.0)	38.0 (35.0 – 40.0)		

Table 10: Comparison between Baseline and 3 months in decline group according to ESD (n= 12)

ESD	Baseline	3 months	t	p
Min. – Max.	26.0 – 39.0	30.0 – 44.0		
Mean ± SD.	33.25 ± 3.65	37.67 ± 3.85	5.942*	<0.001*
Median (IQR)	33.50 (31.0 – 35.50)	38.0 (35.0 – 40.0)		

Table 11: Comparison between the two studied groups according to EDD:

EDD	Normal (n = 41)	Decline (n = 12)	t	P
Baseline				
Min. – Max.	39.0 – 51.0	5.0 – 57.0		
Mean ± SD.	44.12 ± 3.21	44.92 ± 13.63	0.200	0.845
Median (IQR)	44.0 (42.0 – 46.0)	49.0 (42.50 – 52.0)		
1 week				
Min. – Max.	39.0 – 51.0	42.0 – 58.0		
Mean ± SD.	47.05 ± 2.96	49.67 ± 5.03	1.717	0.109
Median (IQR)	47.0 (45.0 – 50.0)	49.50 (45.0 – 54.0		
3 months				
Min. – Max.	5.10 – 57.0	45.0 – 59.0		
Mean ± SD.	45.10 ± 7.09	51.17 ± 4.91	2.766*	0.008*
Median (IQR)	46.0 (44.0 – 48.0)	51.0 (46.50 – 54.50)		

Table 12: Comparison between Baseline and 3 months in decline group according to EDD (n= 12)

EDD	Baseline	3 months	t	p
Min. – Max.	5.0 – 57.0	45.0 – 59.0		
Mean ± SD.	44.92 ± 13.63	51.17 ± 4.91	1.798	0.100
Median (IQR)	49.0 (42.50 – 52.0)	51.0 (46.50 – 54.50)		

Table 13: Comparison between the two studied groups according to strain

Strain	Normal (n = 41)	Decline (n = 12)	t	p
Baseline				
Min. – Max.	-22.0 – -18.0	-22.0 – -18.0		
Mean ± SD.	-19.98 ± 1.31	-19.25 ± 1.36	1.671	0.101
Median (IQR)	-20.0 (-21.0 – -19.0)	-19.0 (-14.0 – -11.0)		
1 week				
Min. – Max.	-22.0 – -18.0	-18.0 – -14.0		
Mean ± SD.	-20.39 ± 1.16	-15.58 ± 1.08	12.809*	<0.001*
Median (IQR)	-21.0 (-21.0 – -20.0)	-15.0 (-16.0 – -15.0)		
3 months				
Min. – Max.	-22.0 – -16.0	-15.0 – -10.0		
Mean ± SD.	-19.88 ± 1.14	-12.42 ± 1.83	13.366*	<0.001*
Median (IQR)	-20.0 (-21.0 – -19.0)	-12.5 (-14.0 – -11.0)		

Table 14: Comparison between the three studied groups according to strain

Strain	Normal (n = 41)	PIVD(n = 10)	PICM (n = 2)	F	p
Baseline					
Min. – Max.	-22.0 – -18.0	-22.0 – -18.0	-18.0 – -18.0		
Mean ± SD.	-19.98 ± 1.31	-19.50 ± 1.35	-18.0 ± 0.0	2.526	0.090
Median (IQR)	-20.0(-21.0 – -19.0)	-19.5(-20.0 – -18.0)	-18.0(-18.0 – -18.0)		
1 week					
Min. – Max.	-22.0 – -18.0	-18.0 – -15.0	-15.0 – -14.0		
Mean ± SD.	-20.39 ± 1.16	-15.80 ± 1.03	-14.50 ± 0.71	85.079*	<0.001*
Median (IQR)	-21.0(-21.0 – -20.0)	-15.5(-16.0 – -15.0)	-14.5(-15.0 – -14.0)		
3 months					
Min. – Max.	-22.0 – -16.0	-15.0 – -10.0	-13.0 – -10.0		
Mean ± SD.	-19.88 ± 1.14	-12.60 ± 1.84	-11.50 ± 2.12	148.59*	<0.001*
Median (IQR)	-20.0(-21.0 – -19.0)	-12.5(-14.0 – -11.0)	-11.5(-13.0 – -10.0)		
Sig. bet. Grps.	p_1<0.001*,p_2<0.001*,p_3=0.534				

Table 15: Comparison between Baseline and 3 months in decline group according to Strain (n= 12)

Strain	Baseline	3 months	t	p
Min. – Max.	-22.0 – -18.0	-15.0 – -10.0		
Mean ± SD.	-19.25 ± 1.36	-12.42 ± 1.83	19.835*	<0.001*
Median (IQR)	-19.0 (-14.0 – -11.0)	-12.5 (-14.0 – -11.0)		

In table (16): Univariate and multivariate Logistic regression analysis for the parameters affecting Decline to investigate the possible predictors for PIVD&PICM in the study population. In both univariate and multivariate we found that GLS is early predictor for PIVD &PICM.

Table 16: Univariate and multivariate Logistic regression analysis for the parameters affecting Decline:

	Univariate		#Multivariate	
	p	**OR (LL – UL 95%C.I)**	**p**	**OR (LL – UL 95%C.I)**
Gender (Female)	**0.097**	3.100(0.814 – 11.808)		
Age (years)	**0.555**	1.019(0.958 – 1.083)		
Hb (g/dl)	**0.953**	0.987(0.639 – 1.525)		
RBS	**0.248**	0.994(0.984 – 1.004)		
WBCS	**0.828**	1.0(1.0 – 1.0)		
Urea	**0.133**	2.817(0.731 – 10.859)		
INR	**0.167**	0.001(0.0 – 17.614)		
eGFR	**0.164**	0.978(0.948 – 1.009)		
HTN	**0.836**	1.154(0.297 – 4.487)		
DM	**0.634**	1.378(0.369 – 5.140)		
Thyroid	**0.907**	1.152(0.109 – 12.203)		
Cardiac	**0.230**	2.429(0.570 – 10.353)		
Renal	**0.882**	0.841(0.085 – 8.323)		
Smoker	**0.424**	1.778(0.434 – 7.280)		
EF 1 week	**0.882**	0.881(0.088 – 8.343		
ESD 1 week	**0.956**	1.665(0.434 – 5.015)		
EDD 1 week	**0.167**	1.160(1.373 – 2.799)		
Strain 1 week	**<0.001***	1.990(1.472-2.899)	0.778	0.804(0.144-3.770)

Discussion

Permanent cardiac pacemaker is the only effective therapy for patients with symptomatic sinus node dysfunction or AV nodal diseases (Aksu *et al.,* 2016). Despite its unquestioned clinical benefits, attention is being drawn to its negative effects accompanying long-term pacing of the right ventricle (Aksu *et al.,* 2016).

Chronic right ventricular pacing causes electrical and mechanical desynchrony which in turn leads to deleterious effects on cardiac function and heart failure, a phenomenon referred to as pacemaker induced cardiomyopathy (Motonaga and Dubin, 2017).

Many studies are trying to predict which patients are more likely to be affected by RV pacing in cases of single and dual chamber pacemaker implantation, but it remains a clinical challenge (Albatat *et al.,* 2020).

For this reason, global longitudinal strain (GLS) measured by 2D speckle tracking echocardiography is emerging as a potentially useful tool to identify subclinical LV dysfunction following pacing. GLS is being used to detect subclinical LV dysfunction in other conditions such as following chemotherapy (Smiseth *et al.,* 2016).

Our study aimed to analyse the effects of Dual chamber pacing on left ventricular function in patient who underwent dual chamber pacemaker implantation using 2D speckle tracking echocardiography. A comprehensive analysis of both LVEF and GLS was provided during short and long term follow up periods, together with combining pre-implantation data.

The study included 53 patients with conduction disturbances admitted for dual chamber pacemaker implantation with baseline ejection fraction above 50% measured by simpson method. Then 2D echocardiography with speckle tracking analysis was done at baseline before pacemaker implantation and at 1 week and 3 months intervals post implantation.

Regarding the demographics in our study

In our study the age of the studied population ranged from 19 to 85 years, with a mean age of the patients in non-decline group was 66.61 ± 12.89 years and in decline group was 68.92 ± 8.16. Most of the participants in the study were females 69.8% while the males were 30.2%.

In our study there was no significant difference regarding age and sex between the patients who developed pacemaker induced ventricular dysfunction and those didn't.

This was similar to Affan *et al.* (2017), where there was no significance between the decline and non-decline groups in baseline and in follow up.

Also, this was the same in Fozia *et al.* (2017), & Pirthiviraj *et al.* (2021) that both showed no significance between both studied groups as regarded age and sex in baseline and follow up.

While in Moustafa *et al.* (2021) there was significant difference as regarded age between the two groups with mean age in non-decline group was 67.20 ± 18.54 while mean age in decline group was 59.04 ± 14.18.

Regarding risk factors and comorbidities in the studied patients:

Regarding hypertension, in our study 66% of our patients were hypertensive with no significant difference between the pacemaker induced ventricular dysfunction group and the preserved function group.

This was the same as in Affan *et al.* (2017), Sarath *et al.* (2021), and in Goutam *et al.* (2023) too, that all showed no significant difference between the two studied groups.

As regard DM & other comorbidities there was not statistically difference between the pacemaker induced ventricular dysfunction group and the preserved function group in our study and all other studies.

The core of our study was assessment the left ventricular function following dule chamber pacemaker using 2D speckle tracking echocardiography.

There is reported evidence that patients with conduction disturbances treated by permanent pacemaker implantation through dual chamber pacing suffer from adverse LV remodelling and consequently in a drop in the LV function and ejection fraction during long term follow up; known as pacemaker induced ventricular dysfunction (Huang *et al.,* 2017).

Accurate quantification of LV function is crucial for risk evaluation and management of these patients. Serial assessment of LVEF was the widely used tool for measuring LV mechanical desynchrony and systolic function. However, it may be an insufficient tool in detecting early changes in cardiac structure and function. Recently, 2 D speckle tracking may emerge as a new parameter for reported studies, it is demonstrated to be more accurate, reproducible, and sensitive for early detection of myocardial dysfunction (Huang *et al.,* 2017).

2D speckle-tracking echocardiography is now validated as an effective method for assessment of myocardial strain. Many studies have demonstrated its benefit beyond traditional LVEF in assessment of various cardiac disease. It provides accurate and reproducible ejection fraction and myocardial strain, and function are altered by RV pacing, hence global longitudinal strain can be utilized to detect subclinical LV dysfunction, before changes in LV ejection fraction (Delgado *et al.,* 2009).

In this study we aimed to provide a comprehensive analysis of both LVEF and GLS, combining the baseline (pre-implant data), 1week follow up and 3 months.

There was statistically significant reduction in both LVEF and GLS at 3 months in the follow up post implantation in the patients who subsequently developed pacemaker induced cardiomyopathy (PICM) & PIVD. These finding were also noted by the logistic regression analysis which confirmed the significance of the baseline and 1-week GLS over ejection thus, GLS may be utilized as a clinically useful tool to predict those patients at higher risk for developing PIVD and who would benefit from heightened echocardiographic surveillance following pacemaker implantation.

As regard LVEF in both studied groups

In our current study, at 3 months follow up, 14 patients (28%) of the total number of patients. Had a significant decline in LVEF>50% percentage points, 4 of whom had a more severe decline in LVEF to <45% (PICM).

In our study, baseline EF & 1 week EF follow up post implantation showed no significant difference regarding the EF of both studied group with (p value 0.141& 0.160).

While at 3 months follow up there was significant differences between the group suffering from pacemaker induced cardiomyopathy and those not with (p values <0.05)

this come as the same of Affan *et al.* (2017) that showed: there was no significance between the 2 studied groups at the baseline with P value <0.05, while after 3 month follow up there was a significant difference regarding both studied groups.

Jung Yeon *et al.* (2021) showed that there was no significance between the 2 studied groups at the baseline with P value 0.92, while after 6 month follow up there was a significant difference regarding both studied groups P value <0.05.

Also, in Fozia *et al.* (2017) showed that there was no significance between two groups at baseline, but there was a significant difference after 1 month and 12 months

While in Prithiviraj *et al.* (2021) there was no significance at baseline and after 1 month between the two studied groups.

In Goutam *et al.* (2023) there is no significance between the two groups at baseline and after 1 month, while there was a significance between both groups after 12 months.

In Teima *et al.* (2020) there was no significance at EF at baseline and after 1 month but there was significance between the two groups after 3-month period.

In Jung *et al.* (2020) there was no significant difference at EF at baseline but there was significance between the two groups after 6-month period.

this wasn't the same as Moustafa *et al.* (2021) that showed: there was a significant difference between EF measured by 3D echo, at baseline,1 week and 3 months of the three-group normal group, PIVD group and PICM group.

Also in Sarath *et al.,* (2018) there was a significant difference between EF measured by 3D echo, at baseline,24h post implantation and 6 months of the three group normal group, PIVD group and PICM group.

Also in Sarath *et al.* (2018) there was no significance difference at baseline but there was a significant difference between EF measured by 3D echo, after 24h post implantation and 6 months of the three group normal group, PIVD group and PICM group.

As regard LVESD in both studied groups

In our study, baseline ESV and 1-week follow up post implantation, ESV showed no significance difference regarding both studied group with (p value 0.101 & 0.088) respectively.

while at 3 months follow up there was significant differences between the group suffering from pacemaker induced cardiomyopathy and those not with (p values <0.05).

This was as Teima *et al.* (2020) where there is no difference between ESV in the two studied groups at baseline and 1 month follow up but there was a significant difference between both groups after 3 months follow up.

But in Moustafa *et al.* (2021) showed that ESV results changes were significant between all studied groups in baseline,1 week and after 3 months follow up.

As regard LVEDD in both studied groups

In our study, baseline EDV and 1-week follow up post implantation we found that EDV showed no significance difference regarding both studied group with (p value 0.845 & 0.109) respectively.

while at 3 months follow up there was significant differences between the group suffering from pacemaker induced cardiomyopathy and those not with (p values <0.05)

This was as **Teima *et al.,* (2020)** [9] where there is no difference between EDV in the two studied groups at baseline and 1 month follow up but there was a significant difference between both groups after 3 months follow up.

But in **Moustafa *et al.,* (2021)** showed that EDV results changes were non-significant between all studied groups in baseline, 1 week and after 3 months follow up.

As regard global longitudinal strain (GLS)

In our study, baseline GLS showed no significance difference regarding both studied group with (p value 0.101).

while at 1 week & 3 months follow up there was significant differences between the group suffering from pacemaker induced cardiomyopathy and those not with (p values <0.05)

This was as Affan *et al.,* (2017) that showed significant decrease between two groups after 3 months follow up.

In Teima *et al.,* (2020), there was no significant change between the two groups at baseline but there was significant difference at 1month follow up and at 3 months.

This also was the same at Fozia *et al.* (2017); Prithiviraj *et al.* (2021) and Goutam *et al.* (2023)[22], that all showed there was no significance between the two groups at baseline but there was significant difference at 1month follow up and at 12 months.

In Sharath *et al.* (2018), there was no significance between the two groups at baseline but there was significant difference at 1day follow up and at 6 months.

While in Moustafa *et al.* (2021) showed that there was a significance between the two groups in the baseline and after 1 week follow up as well as after 6 months follow up.

Conclusion

2D-speckle tracking can be used as early predictor way for pacemaker induced ventricular dysfunction as well as pacemaker induced cardiomyopathy in patients who went dual chamber pacemaker.

References

Abawi, D., T. Rinaldi, A. Faragli, B. Pieske, D.A. Morris, S. Kelle, *et al.,* 2022. The non-invasive assessment of myocardial work by pressure-strain analysis: clinical applications. Heart Fail Rev., 27(4):1261–79.

Ahmed, F.Z., M. Motwani, C. Cunnington, C.S. Kwok, C. Fullwood, D. Oceandy, *et al.,* 2017. One-month global longitudinal strain identifies patients who will develop pacing-induced left ventricular dysfunction over time: the pacing and ventricular dysfunction (PAVD) study. PLoS One, 12(1):e0162072.

Aksu, T., E. Golcuk, K. Yalin, T.E. Guler, and I. Erden, 2016. Simplified cardioneuroablation in the treatment of reflex syncope, functional AV block, and sinus node dysfunction. Pacing Clin Electrophysiol., 39(1):42–53.

Albatat, M., J. Bergsland, H. Arevalo, H.H. Odland, P. Bose, P.S. Halvorsen, *et al.,* 2020. Technological and clinical challenges in lead placement for cardiac rhythm management devices. Ann Biomed Eng., 48:26–46.

Aste, M., and M. Brignole, 2017. Syncope and paroxysmal atrioventricular block. J. arrhythmia, 33(6):562–7.

Babu, N.M.S., S.C. Srinath, A. Lahiri, D. Chase, B. John, and J. Roshan, 2018. Three-dimensional echocardiography with left ventricular strain analyses helps earlier prediction of right ventricular pacing-induced cardiomyopathy. J. Saudi Hear Assoc., 30(2):102–7.

Barold, S.S., R.X. Stroobandt, and A.F. Sinnaeve, 2010. Cardiac pacemakers and resynchronization step by step: An illustrated guide. John Wiley & Sons.

Chin, J.Y., K. Kang, S.H. Park, Y.J. Choi, K.T. Jung, S. Lee, *et al.,* 2021. Pre-implant global longitudinal strain as an early sign of pacing-induced cardiomyopathy in patients with complete atrioventricular block. Echocardiography, 38(2):175–82.

Datta, G., D.G. Dastidar, and H. Chakraborty, 2023. Strain echocardiography in predicting LV dysfunction in RV apical pacing. Indian Heart J.

Dawood, M., E. Elsharkawy, M.A. Abdel-Hay, and M. Nawar, 2021. Predictors of pacing induced left ventricular dysfunction and cardiomyopathy assessed by three-dimensional echocardiography and speckle tracking strain. Egypt Hear J., 73:1–10.

Delgado, V., L.F. Tops, S.A. Trines, K. Zeppenfeld, N. Ajmone Marsan, M. Bertini, E.R. Holman, M.J. Schalij, and J.J. Bax Acute effects of right ventricular apical pacing on left ventricular synchrony and mechanics. Circulation: Arrhythmia and Electrophysiology, 1;2(2):135-45.

Egal, M., H.R.H. de Geus, J. van Bommel, and A.B.J. Groeneveld, 2016. Targeting oliguria reversal in perioperative restrictive fluid management does not influence the occurrence of renal dysfunction: a systematic review and meta-analysis. Eur. J. Anaesthesiol EJA., 33(6):425–35.

Goldberger, Z.D., B.J. Petek, M. Brignole, W.K. Shen, R.S. Sheldon, M. Solbiati, *et al.,* 2019. ACC/AHA/HRS versus ESC guidelines for the diagnosis and management of syncope: JACC guideline comparison. J. Am. Coll. Cardiol., 74(19):2410–23.

Gunasekaran, P., S. Panaich, A. Briasoulis, S. Cardozo, and L. Afonso, 2017. Incremental value of two dimensional speckle tracking echocardiography in the functional assessment and characterization of subclinical left ventricular dysfunction. Curr Cardiol Rev., 13(1):32–40.

Halliday, B.P., R. Senior, and D.J. Pennell, 2021. Assessing left ventricular systolic function: from ejection fraction to strain analysis. Eur. Heart J., 42(7):789–97.

Hasan, A.E., F.A.A. Rahman, and G.F. Haji, 2017. Short Term Evaluation of Left Ventricular

Function Following Dual Chamber Pacemaker a Speckle Tracking Study.

Huang, W., L. Su, S. Wu, L. Xu, F. Xiao, X. Zhou, *et al.,* 2017. Benefits of permanent His bundle pacing combined with atrioventricular node ablation in atrial fibrillation patients with heart failure with both preserved and reduced left ventricular ejection fraction. J. Am. Heart Assoc., 6(4):e005309.

Jordan, J., C. Kurschat, and H. Reuter, 2018. Arterial hypertension: diagnosis and treatment. Dtsch Arztebl Int., 115(33–34):557.

Motonaga, K.S., and A.M. Dubin, 2017. Hemodynamics of pacing and cardiac resynchronization therapy (CRT) for the failing left and right ventricle. Card Pacing Defibrillation Pediatr. Congenit. Hear Dis., 91–117.

Pesola, G.R., and H. Ahsan, 2016. Dyspnea as an independent predictor of mortality. Clin Respir J., 10(2):142–52.

Reddy, V.Y., P. Neuzil, D.F. Booth, R.E. Knops, R.N. Doshi, M. Rashtian, *et al.,* 2023. Dual-chamber leadless pacing: Atrioventricular synchrony in preclinical models of normal or blocked atrioventricular conduction. Hear Rhythm.

Smiseth, O.A., H. Torp, A. Opdahl, K.H. Haugaa, and S. Urheim, 2016. Myocardial strain imaging: how useful is it in clinical decision making? Eur. Heart J., 37(15):1196–207.

Song, M.K., N.Y. Kim, E.J. Bae, G.B. Kim, J.G. Kwak, W.H. Kim, *et al.,* 2020. Long-term follow-up of epicardial pacing and left ventricular dysfunction in children with congenital heart block. Ann Thorac Surg., 109(6):1913–20.

Teima, S.M., A.I. Bedier, A.H. Eladawy, A.R.A. Maaty, J. Singh, and E.M.E. Mahfouz, 2020. Assessment of left ventricular dyssynchrony after permanent cardiac pacing by using two dimensional speckle tracking echocardiography. World Heart J., 12(1):41–50.

Tops, L.F., V. Delgado, N.A. Marsan, and J.J. Bax, 2017. Myocardial strain to detect subtle left ventricular systolic dysfunction. Eur J Heart Fail., 19(3):307–13.

Yaseen, R.I., W.F. Abdelaziz, A.A.H. Katta, and A.E. Soliman, 2022. The effect of different pacing modes on left ventricular global and regional longitudinal systolic strain by two-dimensional speckle-tracking echocardiography. Menoufia Med. J., 35(1):1.