

## Original Article

# Evaluation of Dyssynchrony in Wolff-Parkinson-White Syndrome Patients before and after Radiofrequency Ablation

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## Highlights

- Radiofrequency ablation significantly improved QRS duration, PR interval, fractional shortening, and LVEF in WPW patients.
- Intraventricular dyssynchrony (SPWMD) was markedly reduced after ablation, while interventricular delay (IVMD) showed no significant change.
- Two-dimensional strain analysis demonstrated significant improvement of dyssynchrony indices in all echocardiographic views after ablation.
- The location of accessory pathways was not significantly associated with dyssynchrony, yet ablation successfully eliminated dyssynchrony in all patients.

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## ABSTRACT

**Objective:** To evaluate the impact of radiofrequency ablation (RFA) on dyssynchrony in children with Wolff-Parkinson-White (WPW) syndrome.

**Methods:** This pre-post interventional study was conducted at Rajaie Cardiovascular Institute in Tehran, Iran, and included patients with WPW syndrome who had accessory pathways and were candidates for RFA. Demographic and baseline data, such as age and sex, were recorded. Patients underwent ECG studies and RFA of their accessory pathways. Twelve-lead ECG was performed before and one day after RFA. Standard echocardiographic views were obtained using a Vivid S60 system with appropriate transducers in 1D (M-mode) and 2D modes, along with Doppler evaluation. Intraventricular and interventricular dyssynchrony indices were assessed, and 2D strain analysis was performed. Data were analyzed using IBM SPSS Statistics for Windows, version 26.0.

**Results:** This study included 54 patients with WPW syndrome, 17 of whom had ventricular dyssynchrony. Ablation significantly affected QRS duration, PR interval, fractional shortening, left ventricular ejection fraction, and septal-to-posterior wall motion delay but did not significantly alter interventricular mechanical delay. The most common accessory pathways were left lateral, right posteroseptal, and left posteroseptal. The location of the accessory pathway was not significantly associated with the occurrence of dyssynchrony.

**Conclusions:** Based on the findings of this study, RF ablation eliminated dyssynchrony in all WPW patients and improved functional and strain parameters in this population. These improvements enhance cardiac function and reduce associated risks, particularly in pediatric patients.

**Keywords:** Radiofrequency Ablation; Wolff-Parkinson-White; Dyssynchrony; Accessory Pathway

## Introduction

**A**ccessory pathways occur in 0.1-0.3% of the general population.<sup>1,2</sup> In Wolff-Parkinson-White (WPW) syndrome, ventricles are pre-excited both electrically and mechanically via an accessory pathway, resulting in eccentric ventricular activation and asynchronous depolarization. Left ventricular (LV) dyssynchrony, caused by delayed LV activation, has been previously studied in heart failure patients and is a known poor prognostic indicator, particularly in cases with widened QRS complexes.<sup>3</sup> This dyssynchrony impairs cardiac function by reducing ventricular filling time and promoting mitral regurgitation in heart failure patients. Evidence confirms that synchronous LV contraction correlates with improved ventricular function, symptom relief, and enhanced quality of life.<sup>4,5</sup>

Abnormal LV motion has also been documented in WPW syndrome,<sup>6,7</sup> with some studies implicating abnormal septal wall motion as a potential contributor to LV dyssynchrony and dysfunction.<sup>8,9</sup>

Although LV dyssynchrony in patients with pre-excitation has been documented in previous studies,<sup>10</sup> the relationship between accessory pathway location and its impact on LV dyssynchrony remains incompletely understood. Furthermore, the reversibility of LV dyssynchrony following ablation therapy requires additional investigation.

Echocardiographic assessment methods, including conventional echocardiography and speckle tracking imaging, have been employed to evaluate LV dyssynchrony in WPW patients with septal accessory pathways.<sup>11,12</sup>

Prior to ablation, the majority of patients with septal accessory pathways demonstrated significant LV dyssynchrony. As expected, catheter ablation resulted in a substantial reduction of LV dyssynchrony along with observable improvements in systolic function.<sup>11,12</sup> The mechanism likely involves early septal activation causing dyskinetic/dyssynchronous septal motion, analogous to the effects seen in right ventricular apical pacing-induced left bundle branch block.<sup>13-15</sup> This abnormal septal motion

may significantly impair cardiac function.

These findings underscore the clinical relevance of LV dyssynchrony in the development of LV dysfunction among patients with ventricular pre-excitation. Nonetheless, it should be noted that not all patients with right-sided accessory pathways and pre-excitation develop severe LV dysfunction.

The severity of LV dyssynchrony induced by accessory pathways may represent an independent risk factor in these patients. Current evidence indicates that septal accessory pathways at different locations may produce varying degrees of LV dyssynchrony.<sup>11, 13-15</sup> Notably, LV dysfunction can occur even in patients without WPW syndrome or accessory pathways.<sup>11,12</sup> Two-dimensional speckle-tracking echocardiography provides a quantitative assessment of LV dysfunction severity.<sup>16</sup>

While optimal management strategies remain debated, echocardiographic surveillance of cardiac dimensions and function is crucial. Catheter ablation appears most warranted for patients demonstrating LV remodeling, progressive LV dyssynchrony, or declining cardiac function during follow-up. Given the clinical significance of these findings for patient management and outcomes, we sought to systematically evaluate dyssynchrony patterns in WPW patients before and after ablation.

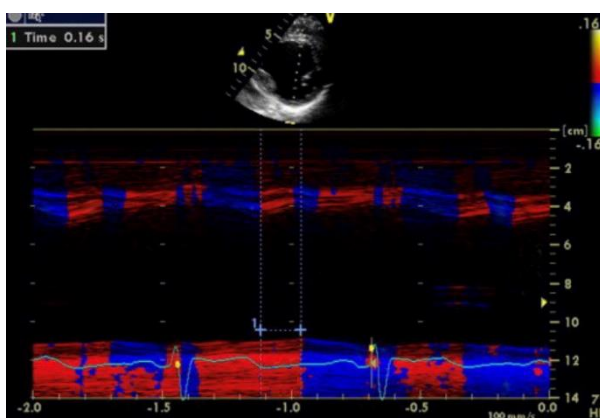
## Methods

This pre-post interventional study was conducted at Rajaie Cardiovascular Institute, affiliated with Iran University of Medical Sciences, on patients with WPW syndrome who had accessory pathways and were candidates for radiofrequency ablation (RFA). Informed consent was obtained from the patients' families. Demographic and baseline data, such as age and sex, were recorded. Antiarrhythmic drugs were discontinued three days before RFA. Patients underwent electrophysiological studies and RFA of their accessory pathways. The study was approved by the Ethics Committee of the Research Deputy of Iran University of Medical Sciences.

Patients with a confirmed diagnosis of WPW syndrome based on clinical symptoms and ECG findings were included. Patients with other cardiac diseases, concealed accessory pathways, and other arrhythmias accompanying WPW were excluded.

All patients underwent 12-lead ECG during sinus rhythm at complete rest before and 1 day after RFA. QRS and PR intervals were measured manually, with the longest QRS duration in any lead and the shortest PR interval recorded. Standard echocardiographic views were obtained using a Vivid S60 system with appropriate transducers, including 1D (M-mode) and 2D imaging with Doppler evaluation.

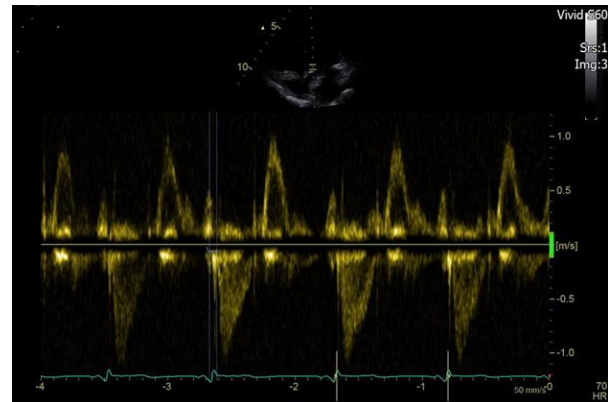
LV end-diastolic and end-systolic diameters were measured from M-mode parasternal long-axis views to calculate fractional shortening and left ventricular ejection fraction (LVEF). Intraventricular and interventricular dyssynchrony indices were assessed. For intraventricular dyssynchrony, septal-to-posterior wall motion delay (SPWMD) was measured using the color-coded method, with values  $>130$  ms considered indicative of LV dyssynchrony. SPWMD was determined by identifying maximal wall thickening as the marker of active contraction (Figure 1).



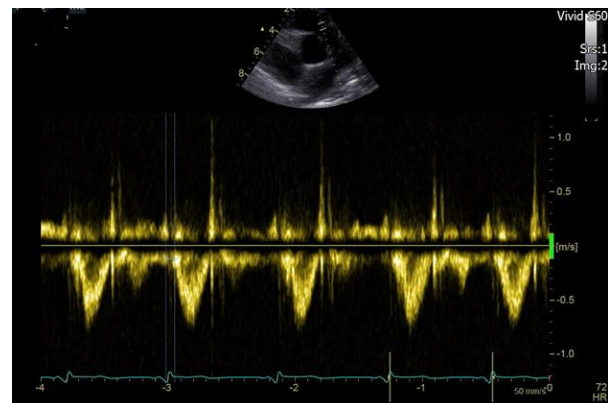
**Figure 1.** Measurement of septal-to-posterior wall motion delay (SPWMD) by echocardiography.

The time intervals between the electrical systole and the opening of the pulmonary and aortic valves were recorded. The interventricular mechanical delay (IVMD) was calculated as the difference between left and right ventricular pre-

ejection periods. IVMD values  $>40$  ms were classified as indicating left ventricular dyssynchrony, while values  $\leq 40$  ms were considered normal (Figures 2-3).



**Figure 2.** Measurement of interventricular mechanical delay (IVMD) by echocardiography, demonstrating left ventricular pre-ejection period assessment.



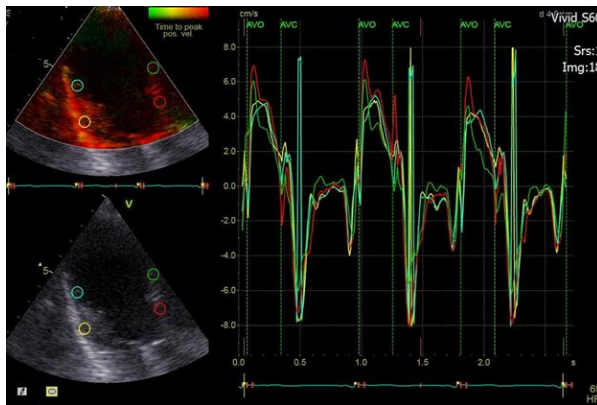
**Figure 3.** Measurement of interventricular mechanical delay (IVMD) by echocardiography, demonstrating right ventricular pre-ejection period assessment.

Two-dimensional strain analysis was performed using echocardiographic images with frame-by-frame tracking of acoustic tissue markers at acquisition rates of 60-120 frames per second. Segmental myocardial deformation was analyzed with negative strain values representing contraction and positive values indicating stretch. For each myocardial segment, the time interval from QRS onset to peak strain was measured. Left ventricular dyssynchrony severity was quantified by calculating the maximum temporal difference (Time 2D strain) between all paired segmental strain measurements (Figure 4).

Two-dimensional strain analysis was performed in the following views: (1) four-chamber view: inferoseptal vs. anterolateral walls; (2) three-chamber view: antero-septal vs. inferolateral walls; and (3) two-chamber view: anterior vs. inferior

walls. Measurements were obtained at basal, mid-ventricular, and apical levels in each view.

All analyses were performed both before and one day after RFA. For the assessment of interobserver variability, a blinded pediatric cardiologist independently recalculated all echocardiographic parameters without knowledge of temporal sequence (pre- vs. post-RFA).



**Figure 4.** Time 2D strain measurement on echocardiography.

## Statistical Analysis

All data were analyzed using IBM SPSS Statistics for Windows (version 26.0; IBM Corp., Armonk, NY). Continuous variables are presented

as mean  $\pm$  standard deviation for normally distributed data or median (range) for non-normally distributed data, with distribution normality assessed using the Kolmogorov-Smirnov test.

Between-group comparisons employed either the two-tailed Student t-test (normal distribution) or the Mann-Whitney U test (non-normal distribution). Categorical variables were analyzed using the Fisher exact test. For within-subject comparisons before versus after RFA, the paired t-test was utilized for normally distributed data or the Wilcoxon signed-rank test for non-normal distributions. The Kruskal-Wallis test served as the non-parametric alternative for multiple group comparisons. Statistical significance was defined as A P-value below 0.05 for all analyses.

## Results

The study cohort comprised 54 consecutive patients with WPW syndrome. Ventricular dyssynchrony was present in 17 patients (31.5%) and absent in 37 patients (68.5%). The population included 31 males (57.4%) with a mean age of  $10.83 \pm 3.57$  years. Complete demographic characteristics are presented in (Table 1).

**Table 1.** Demographic parameters of patients with and without dyssynchrony

Parameters	Without Dyssynchrony	With Dyssynchrony	P
Sex	Male (45.9%) 17 Female (35.3%) 6	Male (54.1%) 20 Female (64.7%) 11	0.462
Age	$3.46 \pm 10.94$	$3.90 \pm 10.58$	0.736
Weight	$19.82 \pm 43.89$	$14.14 \pm 35.17$	0.109
Positive medication history	(8.1%) 3	(52.9%) 9	0.000

Accessory pathway locations were distributed as follows: left lateral (31.5%), right posteroseptal (13%), left posteroseptal (7.4%), right ventricular free wall (7.4%), right midseptal (7.4%), and epicardial (5.6%). Right-sided pathways occurred in 24 patients (44.4%) compared to 30 patients (55.6%) with left-sided pathways. Accessory pathway location showed no significant correlation

with dyssynchrony occurrence ( $P=0.268$ ).

Ablation significantly improved several parameters: QRS duration, PR interval, fractional shortening, LVEF, and SPWMD. Complete quantitative results for these measures appear in (Table 2). Strain analysis was also performed, and the Time 2D strain analysis results are presented in (Table 3).



**Table 2.** Cardiac parameters before and after ablation in groups with and without dyssynchrony

Parameters		Without Dyssynchrony	With Dyssynchrony	P
QRS duration	Before ablation	138.91±23.18	152.94±24.43	0.048
	After ablation	73.51±8.88	76.47±7.01	0.233
	P-value	0.000	0.000	
PR interval	Before ablation	51.35±9.25	53.23±9.17	0.489
	After ablation	103.51±8.27	102.43±8.30	0.157
	P-value	0.000	0.000	
Fractional shortening	Before ablation	40.67±3.00	35.82±2.83	0.000
	After ablation	40.67±3.00	36.88±2.44	0.000
	P-value	-	0.015	
Ejection fraction	Before ablation	58.08±1.36	55.11±2.34	0.002
	After ablation	58.08±1.36	57.00±1.06	0.006
	P-value	-	0.001	
Interventricular mechanical delay	Before ablation	8.67±2.18	7.35±2.39	0.057
	After ablation	8.54±1.92	7.35±2.39	0.057
	P-value	0.324	-	
Septal-to-posterior wall motion delay	Before ablation	120.43±4.34	130.06±2.3	0.030
	After ablation	120.43±4.34	100.05±5.5	0.000
	P-value	-	0.001	

**Table 3.** Time 2D strain analysis in pre- and post-ablation states

Study Type		Before Ablation	After Ablation	P
Four-chamber	Base	23.52±13.66	12.94±4.69	0.001
	Middle	23.52±14.11	12.35±5.62	0.001
	Apex	24.11±14.16	12.35±4.37	0.001
Three-chamber	Base	30.00±16.20	13.52±4.92	0.000
	Middle	31.76±17.76	15.29±5.14	0.000
	Apex	25.29±13.28	11.17±3.32	0.000
Two-chamber	Base	17.05±12.12	11.76±3.92	0.024
	Middle	18.82±13.63	12.35±4.37	0.023
	Apex	18.23±14.24	11.17±3.32	0.035

The association between accessory pathway location and dyssynchrony patterns was analyzed. Among patients with dyssynchrony, left lateral pathways were most common (35.29%). All six cases (100%) with left lateral pathways showed dyssynchrony in the mid-segment of the three-chamber view, while four cases (66.7%) involved the base segment and three cases (50%) the apex segment of the same view. Two patients with epicardial pathways (11.76%) exhibited dyssynchrony in all segments (base, mid, and apex) of both the four- and two-chamber views. Among patients with left posterior pathways (n=2), both (100%) exhibited dyssynchrony in the base segment of the three-chamber view, with one patient (50%) showing additional involvement of the base and apex segments (four-chamber view)

and mid/apex segments (three-chamber view). Both patients with left posterolateral pathways (100%) demonstrated dyssynchrony in all segments (base, mid, apex) of the three-chamber view, while one (50%) had additional apex segment involvement in the four-chamber view. For right free wall pathways (n=2), all patients (100%) showed dyssynchrony in the apex segment (four-chamber view) and mid segment (two-chamber view), with one patient (50%) exhibiting additional dyssynchrony in the mid segment (four-chamber view) and base/apex segments (two-chamber view).

The analysis of rare accessory pathways revealed a single patient with combined anterior right/left pathway exhibited dyssynchrony in all segments (base, mid, and apex) of both four-

chamber and three-chamber views and two patients with right posteroseptal pathways showed identical dyssynchrony patterns - involving all segments (base, mid, and apex) of the three-chamber view plus base/mid segments of the four-chamber view.

## Discussion

In WPW syndrome, early electrical and mechanical activation occurs in myocardial segments adjacent to the ventricular insertion site of accessory pathways. The degree of ventricular pre-excitation depends on the relative timing between native conduction and eccentric activation through these pathways. More extensive pre-excitation can induce significant ventricular dyssynchrony, potentially compromising ventricular function.

Our study evaluated 54 pediatric WPW patients, including 17 (31.5%) with documented ventricular dyssynchrony. Catheter ablation successfully eliminated accessory pathways in all cases. The principal findings demonstrate complete restoration of ventricular synchrony post-ablation, accompanied by significant improvement in echocardiographic strain parameters. These results align with existing literature documenting normalization of ventricular depolarization patterns following successful ablation therapy.

Our analysis revealed no significant correlations between patient sex, age, or weight and study outcomes, confirming these variables did not confound the results. However, we identified a notable association between ventricular dyssynchrony and prior medication use: over 50% of dyssynchrony cases had received cardiac medications, compared to only 7% of non-dyssynchrony cases. This suggests patients developing dyssynchrony may have had more severe clinical presentations warranting earlier pharmacologic intervention.

The accessory pathways in our study population were predominantly left lateral (over two-thirds of cases), with equal representation of right and left posterolateral, right free wall, and right midseptal pathways. Left-sided pathways accounted for the majority of cases (55.6%). We

identified a distinct anatomical relationship between pathway location and dyssynchrony patterns, with left lateral pathways consistently producing dyssynchrony in the mid-septal region of the 3-chamber view. Notably, our analysis revealed no significant associations between accessory pathway location/sidedness and clinical symptoms or other demographic factors.

These findings differ from established literature. Udink et al.<sup>17</sup> demonstrated that right-sided accessory pathways with ventricular pre-excitation can induce dilated cardiomyopathy. Kwon et al.<sup>18</sup> reported significantly reduced LVEF in patients with septal pathways compared to those with right or left lateral pathways. Park et al.<sup>19</sup> further established in animal models and chronic patients that left lateral accessory pathways produce the most severe left ventricular dysfunction, with varying degrees of impairment based on precise anatomical location.

Both study groups demonstrated abnormally elevated QRS durations before ablation, with significantly higher values observed in the dyssynchronous group. Following ablation, QRS normalization occurred in both groups, eliminating the intergroup difference. The PR interval, initially shortened in both cohorts, increased post-ablation to normal limits with no residual difference between groups. These electrophysiological improvements align with existing literature: Tomaske et al.<sup>20</sup> documented QRS reduction from 129 ms to 90 ms post-ablation in WPW patients, while Zhang et al.<sup>21</sup> reported similar QRS normalization patterns.

LVEF, a critical functional and prognostic parameter, showed differential responses post-ablation. While no significant change occurred in the non-dyssynchronous group, the dyssynchronous group demonstrated a statistically significant 2% improvement ( $P < 0.05$ ).<sup>21</sup>

Consistent with our findings, multiple studies have documented EF improvement following ablation. Tomaske et al.<sup>20</sup> reported EF increase from 50% to 56%, while Kwon et al.<sup>18</sup> demonstrated a more substantial improvement from 42% to 67%. In our dyssynchronous group, IVMD showed no significant post-ablation change,

whereas SPWMD decreased significantly. As established markers of left ventricular dyssynchrony, the selective SPWMD improvement confirms the specific anti-dyssynchrony effects of ablation. This pattern mirrors previous reports: Tomaske et al.<sup>20</sup> observed significant SPWMD improvement without meaningful IVMD changes in WPW patients, and Kwon et al.<sup>18</sup> documented SPWMD reduction from 154 ms to 33 ms. Zhang et al.<sup>21</sup> similarly reported SPWMD normalization and complete resolution of left ventricular dyssynchrony post-ablation.

Two-dimensional strain analysis demonstrated significant post-ablation improvement in Time parameters across all echocardiographic views (four-, three, and two-chamber views) and ventricular segments (base, mid, apex) within the dyssynchronous group, confirming ablation's efficacy for correcting WPW-associated dyssynchrony. Notably, our study provides the first reported pre-/post-ablation strain parameter values in WPW patients, addressing a gap in existing literature.

The study had several limitations, including a moderate sample size (n=54) with diverse accessory pathway locations, which restricted pathway-specific analyses. More robust parameter assessment might be possible in larger cohorts or studies focusing on specific pathway types. Additionally, the limited follow-up duration (one day post-ablation) may not capture longer-term outcomes. Future investigations with extended follow-up periods could yield additional insights.

## Conclusion

The results of this study demonstrate that RFA effectively eliminates dyssynchrony in all patients with WPW syndrome while simultaneously improving both functional parameters and strain measurements. These electrophysiological corrections translate to enhanced cardiac function and reduced clinical risks, with particularly significant benefits observed in pediatric populations.

## Declarations: Ethical Approval

This study was approved by the Ethics Committee of Rajaie Cardiovascular Institute (Approval No: IR.IUMS.FMD.REC.1402.248).

## Funding

There is no funding for this study.

## Conflict of Interest

There is no conflict of interest in this study.

## Acknowledgment

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## References

1. Chung KY, Walsh TJ, Massie E. Wolff–Parkinson–White syndrome. *Am Heart J*. 1965;69:116-33.
2. Klein GJ, Yee R, Sharma AD. Longitudinal electrophysiologic assessment of asymptomatic patients with the Wolff-Parkinson-White electrocardiographic pattern. *N Engl J Med*. 1989;320(16):1229-33.
3. Shamim W, Francis DP, Yousufuddin M, Varney S, Pieopli MF, Anker SD, et al. Intraventricular conduction delay: a prognostic marker in chronic heart failure. *Int J Cardiol*. 1999;70(2):171-8.
4. Bristow MR, Saxon LA, Boehmer J, Krueger S, Kass DA, De Marco T, et al. Cardiac-resynchronization therapy with or without an implantable defibrillator in advanced chronic heart failure. *N Engl J Med*. 2004;350(21):2140-50.
5. Cleland JG, Daubert JC, Erdmann E, Freemantle N, Gras D, Kappenberger L, et al. The effect of cardiac resynchronization on morbidity and mortality in heart failure. *N Engl J Med*. 2005;352(15):1539-49.
6. Sasse L. Interventricular septal motion in Wolff–Parkinson–White syndrome. *Am J Cardiol*. 1977;39(3):469-70.
7. DeMaria AN, Vera Z, Neumann A, Mason DT. Alterations in ventricular contraction pattern in the

- Wolff–Parkinson–White syndrome. Detection by echocardiography. *Circulation*. 1976;53(2):249-57.
8. Emmel M, Balaji S, Sreeram N. Ventricular preexcitation associated with dilated cardiomyopathy: a causal relationship? *Cardiol Young*. 2004;14(6):594-9.
  9. Fazio G, Mongiovi M, Sutera L, Novo G, Novo S, Pipitone S. Segmental dyskinesia in Wolff–Parkinson–White syndrome: a possible cause of dilatative cardiomyopathy. *Int J Cardiol*. 2008;123(2):e31-4.
  10. De Boeck BW, Teske AJ, Leenders GE, Mohamed Hoesein FA, Loh P, van Driel VJ, et al. Detection and quantification by deformation imaging of the functional impact of septal compared to free wall preexcitation in the Wolff–Parkinson–White syndrome. *Am J Cardiol*. 2010;106(4):539-46.
  11. Kwon BS, Bae EJ, Kim GB, Noh CI, Choi JY, Yun YS. Septal dyskinesia and global left ventricular dysfunction in pediatric Wolff-Parkinson-White syndrome with septal accessory pathway. *J Cardiovasc Electrophysiol*. 2010;21(3):290-5. doi:10.1111/j.1540-8167.2009.01612.x.
  12. Tomaske M, Janousek J, Rázek V, Gebauer RA, Tomek V, Hindricks G, et al. Adverse effects of Wolff-Parkinson-White syndrome with right septal or posteroseptal accessory pathways on cardiac function. *Europace*. 2008;10(2):181-9. doi:10.1093/europace/eun005.
  13. Udink ten Cate FE, Kruessell MA, Wagner K, Trieschmann U, Emmel M, Brockmeier K, et al. Dilated cardiomyopathy in children with ventricular preexcitation: the location of the accessory pathway is predictive of this association. *J Electrocardiol*. 2010;43(2):146-54. doi:10.1016/j.jelectrocard.2009.09.007.
  14. Emmel M, Balaji S, Sreeram N. Ventricular preexcitation associated with dilated cardiomyopathy: a causal relationship? *Cardiol Young*. 2004;14(6):594-9.
  15. Fujii J, Watanabe H, Kato K, Yazaki Y, Sonobe T, Inoue M. M-mode and cross-sectional echocardiographic study of the left ventricular wall motions in complete left bundle-branch block. *Br Heart J*. 1979;42(3):255-62.
  16. Pavlopoulos H, Nihoyannopoulos P. Strain and strain rate deformation parameters: from tissue Doppler to 2D speckle tracking. *Int J Cardiovasc Imaging*. 2008;24(5):479-91. doi:10.1007/s10554-007-9286-9.
  17. Udink ten Cate FE, Kruessell MA, Wagner K, Trieschmann U, Emmel M, Brockmeier K, et al. Dilated cardiomyopathy in children with ventricular preexcitation: the location of the accessory pathway is predictive of this association. *J Electrocardiol*. 2010;43(2):146-54. doi:10.1016/j.jelectrocard.2009.09.007.
  18. Kwon BS, Bae EJ, Kim GB, Noh CI, Choi JY, Yun YS. Septal dyskinesia and global left ventricular dysfunction in pediatric Wolff-Parkinson-White syndrome with septal accessory pathway. *J Cardiovasc Electrophysiol*. 2010;21(3):290-5. doi:10.1111/j.1540-8167.2009.01612.x.
  19. Park HE, Chang SA, Kim JH, Oh IY, Choi EK, Oh S. Left ventricular dyssynchrony in pre-excitation syndrome: effect of accessory pathway location and reversibility after ablation therapy. *Heart Vessels*. 2013;28(2):199-207. doi:10.1007/s00380-012-0233-x.
  20. Tomaske M, Janousek J, Rázek V, Gebauer RA, Tomek V, Hindricks G, et al. Adverse effects of Wolff-Parkinson-White syndrome with right septal or posteroseptal accessory pathways on cardiac function. *Europace*. 2008;10(2):181-9. doi:10.1093/europace/eun005.
  21. Zhang Y, Xin M, Liu T, Song S, Wang W, Li J, et al. The effect of accessory pathway location on cardiac function in adult patients with Wolff-Parkinson-White syndrome. *Cardiol Res Pract*. 2021;2021:8841736. doi:10.1155/2021/8841736.