

Electrocardiographic Remodeling Before and After Interventional Closure of Secundum Atrial Septal Defects

Yulius Patimang¹, Andi Renata Bastario S.¹, Abdul Hakim Alkatiri¹, Andi Alief Utama Armyn¹, Irfan Idris², Muzakkir Amir¹, Dat T. Nguyen³, Andriany Qanitha^{1,2}

Abstract

Background: Secundum atrial septal defect (ASD) is a common congenital cardiac lesion that produces left-to-right shunting and chronic right-sided volume overload, leading to geometric and electrical remodeling. Electrocardiography (ECG) provides a simple, non-invasive method to detect ASD-related electrical abnormalities and to assess reverse remodeling following defect closure. This study aimed to evaluate the short-term (<24 hours) and long-term (>6 months) electrical changes after Secundum ASD closure.

Methods: We conducted a prospective, single-center, observational cohort study of Secundum ASD patients who underwent percutaneous or surgical closure at RSUP Dr. Wahidin Sudirohusodo. A total of 54 eligible subjects were included. ECG measurements were performed at admission (pre-closure), within 24 hours post-closure, and at more than 6 months post-closure. ECG parameters were statistically compared using a paired T-test or Wilcoxon signed-rank test.

Results: Of the 54 ASD patients, the majority were female (66.7%) and adults (68.5%), with 33 patients (61.1%) undergoing percutaneous closure. Significant reductions were observed across all ECG parameters within 24 hours post-closure, including P-wave amplitude (0.19 ± 0.04 to 0.11 ± 0.03 mV; $p < 0.001$), P-wave duration (97.78 ± 11.94 to 75.35 ± 13.36 ms; $p < 0.001$), PR interval (182.89 ± 26.47 to 156.83 ± 21.81 ms; $p < 0.001$), QRS duration (112.97 ± 14.84 to 88.31 ± 14.43 ms; $p < 0.001$), QRS axis (107.94 ± 23.00 to $95.25 \pm 24.62^\circ$; $p < 0.001$), QTc interval (403.84 ± 30.85 to 396.80 ± 33.76 ms; $p = 0.017$), and R-wave amplitude in V1 (0.74 ± 0.35 to 0.53 ± 0.24 mV; $p < 0.001$). From <24 hours to >6 months post-closure, most parameters continued to decline significantly, with the exception of P-wave amplitude, which remained stable ($p = 0.321$).

Conclusions: Interventional closure of Secundum ASD induces marked electrical reverse remodeling, evident both within hours of the procedure and during long-term follow-up. These findings underscore the value of ECG as an accessible and informative modality for tracking post-intervention cardiac recovery.

¹ Department of Cardiology and Vascular Medicine, Dr. Wahidin Sudirohusodo General Teaching Hospital, Faculty of Medicine, Universitas Hasanuddin, Makassar, Indonesia.

² Department of Physiology, Faculty of Medicine, Universitas Hasanuddin, Makassar, Indonesia.

³ Department of Cardiology, Erasmus Medical Center, Rotterdam, The Netherlands.

Correspondence:
Andriany Qanitha

Faculty of Medicine, Universitas Hasanuddin, Makassar, Indonesia.

Email: a.qanitha@unhas.ac.id

(Indonesian J Cardiol, 2025;46;150-157)

Keywords: Secundum atrial septal defect, electrocardiogram, reverse remodeling, congenital heart disease, percutaneous closure, surgical closure

Introduction

Atrial Septal Defect (ASD) is the second most common non-cyanotic congenital heart defect, with Secundum ASD accounting for over 80% of cases.¹ It causes a persistent left-to-right shunt, leading to chronic right-sided volume overload and progressive structural and electrical remodeling of the right atrium and ventricle.² Early surgical or device closure can reverse these changes by terminating the atrial shunt. However, many ASD patients remain asymptomatic until adulthood and are often diagnosed incidentally during routine cardiovascular assessment.³

Electrocardiography (ECG) is a convenient non-invasive tool for evaluating remodeling in ASD patients, as it is routinely performed before and after intervention. Characteristic ECG changes in ASD include Right Bundle Branch Block (RBBB), Right Ventricular Hypertrophy (RVH), Right Atrial Enlargement (RAE), Right Axis Deviation (RAD), the Crochetage sign, and, in more advanced cases, right ventricular strain patterns⁴, reflect right-sided overload. Comparing ECG parameters before and after closure can help assess the effect of intervention on remodeling, with serial improvements serving as quantitative markers of recovery in cardiac structure and conduction. Prior studies, including those by Bernardo et al., have shown that ECG parameters correlate with defect severity and can track reverse remodeling after percutaneous or surgical closure.⁵

Despite these findings, evidence supporting ECG as a tool to assess reverse electrical remodeling after ASD closure remains limited. To date, no data are available for the Indonesian ASD population. This study aims to evaluate ECG parameter changes in Indonesian patients with Secundum ASD who underwent interventional closure. The findings may support the development of an ECG-based framework for monitoring postprocedural electrical remodeling and improving long-term management after ASD closure.

Methods

This study employed a prospective, observational pre–post cohort study to evaluate changes in ECG parameters in patients with Secundum ASD following transcatheter or surgical closure. Data were obtained from patients who underwent Secundum ASD closure at the Integrated Heart Center, Dr. Wahidin Sudirohusodo General Hospital. ECG parameters were assessed within 24 hours and 6 months post-intervention. Patients with concomitant congenital heart disease, alternative causes of pulmonary hypertension, or significant pre-existing arrhythmias were excluded.

Statistical analyses were performed using paired t-tests for normally distributed variables and Wilcoxon signed-rank tests for non-normally distributed variables. The study protocol was approved by the Health Research Ethics Committee

Table 1. Baseline patient characteristics (categorical variables).

Variable	Total (n=54)	Percentage
Gender		
Male	18	33.3
Female	36	66.7
Age Group		
> 16 years old	38	70.3
≤ 16 years old	16	29.7
Intervention Method		
Percutaneous	33	61.1
Surgical	21	38.9
Pre-intervention Medication		
Furosemide	21	38.9
ACE-Inhibitor/ARB	5	9.2
Beta Blocker	11	20.3
MRA	14	25.9
PDE-5 Inhibitor	4	7.4
Prostacyclin Analogue	10	18.5
Digitalis	4	7.4
Pre-intervention Medication		
ASA	10	18.5
P2Y12 Inhibitor	23	42.6

Furosemide	12	22.2
ACE-Inhibitor	3	5.5
Beta Blocker	5	9.2
MRA	10	18.5
PDE-5 Inhibitor	3	5.5
Prostacyclin Analogue	8	14.8
Digitalis	2	3.7

ACE-I: Angiotensin-Converting Enzyme Inhibitor; ARB: Angiotensin II Receptor Blocker; MRA: Mineralocorticoid Receptor Antagonist; PDE-5: Phosphodiesterase-5; ASA: Acetylsalicylic Acid.

of the Faculty of Medicine, Universitas Hasanuddin, and Dr. Wahidin Sudirohusodo General Hospital (Approval No. 423/UN4.6.4.5.31/PP36/2020; No. UH21060397).

Results

A total of 54 patients with Secundum ASD who underwent interventional closure were included in the analysis; nine patients were lost to follow-up at the 6-month evaluation. Baseline characteristics of the study population are summarized in Tables 1 and 2.

In the comparison between pre-closure and the early post-closure period (<24 hours), all evaluated ECG parameters demonstrated significant reductions: P-wave amplitude (0.19 ± 0.04 vs. 0.11 ± 0.03 mV; $p < 0.001$), P-wave duration (97.78 ± 11.94 vs. 75.35 ± 13.36 ms; $p < 0.001$), PR interval (182.89 ± 26.47 vs. 156.83 ± 21.81 ms; $p < 0.001$), QRS duration (112.97 ± 14.84 vs. 88.31 ± 14.43 ms; $p < 0.001$), QRS axis (107.94 ± 23.00 vs. $95.25 \pm 24.62^\circ$; $p < 0.001$), QTc interval (403.84 ± 30.85 vs. 396.80 ± 33.76 ms; $p = 0.017$), and R-wave amplitude in V1 (0.74 ± 0.35 vs. 0.53 ± 0.24 mV; $p < 0.001$).

Table 2. Baseline characteristics of the study participants (numerical variables).

Variables	Min/Max (n = 54)	Mean \pm SD or Median (Q1 – Q3)
Age (years)	2/58	24.6 \pm 13.3
Body weight (kg)	11/80	45.1 \pm 16.1
Body height (cm)	83/179	148.9 \pm 20.2
Body mass index (kg/m ²)	11.57/35.09	19.6 \pm 4.5
Body surface area (m ²)	0.49/1.91	1.4 \pm 0.3
Defect size (mm)		
TTE	8/43	20.7 \pm 7.6
TEE	12/47	25.3 \pm 8.1
Flow ratio	1.55/6.90	3.4 \pm 1.5
Peripheral O ₂ Saturation (%)	92/100	97.2 \pm 1.9
mPAP (mmHg)	20/61	37.4 \pm 9.9
PVR (WU)	0.20/4.50	2.3 \pm 1.3
PVRI (WU/m ²)	0.12/3.39	1.7 \pm 0.8
Haematology		
Leukocyte (*10 ³ /μL)	2.90/17.20	8.8 \pm 2.7
Haemoglobin (gr/dL)	10.60/16/60	13.5 \pm 1.5
Platelet (*10 ³ /μL)	150/527	296.3 \pm 76.4
Renal functions		
Ureum (mg/dL)	10/57	20.9 \pm 7.7
Creatinine (mg/dL)	0.10/1/06	0.6 \pm 0.2
Liver function		
SGOT (U/L)	10/96	22.0 (17.8 – 29.3)

SGPT (U/L)	7/259	16.5 (12.0 – 25.3)
Coagulation		
PT (sec)	9.3/12.4	10.8 ± 0.7
INR	0.87/1.21	1.0 ± 0.07
APTT (sec)	22.3/36.3	28.2 ± 2.9
Electrolyte		
Natrium (mmol/L)	134/146	141.4 ± 2.6
Kalium (mmol/L)	3.1/4.7	4.0 ± 0.4
Chloride (mmol/L)	98/113	105.3 ± 3.1

Values are means ± SD or median (Q1-Q3). TTE: Trans-thoracal Echocardiography; TEE: Trans-esophageal Echocardiography; mPAP: mean Pulmonary Artery Pressure, PVR: Pulmonary Vascular Resistance; PVRI: Pulmonary Vascular Resistance Index; SGOT: Serum Glutamate Oxalat Transaminase; SGPT: Serum Glutamate Pyruvat Transaminase; PT: Prothrombin Time; INR: International Normalized Ratio; aPTT: activated Partial Tromboplastin Time, WU: Wood Units.

Table 3. Comparison of ECG parameters before and 24-hour after intervention.

ECG Parameters	Pre-intervention (n = 54)	<24 hour post intervention (n = 54)	ECG changes (Δ) mean difference (95% CI)	p-value
P wave amplitude (mv)	0.19 ± 0.04	0.12 ± 0.03	0.07 (0.06 – 0.08)	<0.001*
P wave duration (ms)	97.8 ± 11.9	75.4 ± 13.4	22.4 (19.6 – 25.3)	<0.001*
PR interval (ms)	182.9 ± 26.5	156.8 ± 21.8	26.1 (21.9 – 30.2)	<0.001*
QRS duration (ms)	113.0 ± 14.8	88.3 ± 14.4	24.7 (20.9 – 28.5)	<0.001*
QRS axis (°)	107.9 ± 23.0	95.3 ± 24.6	12.7 (10.1 – 15.2)	<0.001*
QTc interval (ms)	403.8 ± 30.9	396.8 ± 33.8	7.0 (0.5 – 13.6)	0.035*
R wave V1 amplitude (mv)	0.74 ± 0.35	0.53 ± 0.24	0.21 (0.14 – 0.28)	<0.001*

*p<0.05. Values are mean ± SD. Analyses were done using dependent t-test.

Hypothesis testing for pre- and post-intervention ECG differences is presented in Tables 3 and 4. When comparing the early (<24 hours) and late (>6 months) post-closure periods, these reductions were largely sustained across parameters. The only exception was P-wave amplitude (0.121 ± 0.03 vs. 0.119 ± 0.03 mV; p = 0.321), which showed no significant interval change.

We subsequently performed a subgroup analysis

to compare changes in ECG parameters before and after closure intervention, specifically P-wave duration, P-wave amplitude, and PR interval, between adult and pediatric patients, as illustrated in Figure 1. Statistically significant reductions were observed in all assessed ECG parameters in both adults and pediatric patients (p < 0.001 for all comparisons).

Table 4. Comparison of ECG parameters 24-hour vs. 6-month after intervention.

ECG Parameters	<24 hour post intervention (n = 45)	6-month post intervention (n = 45)	ECG changes (Δ) mean difference (95% CI)	p-value
P wave amplitude (mv)	0.12 ± 0.03	0.12 ± 0.03	0.001 (-0.005 – 0.007)	0.779
P wave duration (ms)	75.1 ± 13.8	69.5 ± 11.8	5.6 (3.4 – 7.8)	<0.001*
PR interval (ms)	155.5 ± 22.8	148.3 ± 19.3	7.2 (3.1 – 11.4)	<0.001*
QRS duration (ms)	89.7 ± 14.0	85.4 ± 14.2	4.4 (2.2 – 6.5)	<0.001*
QRS axis (°)	94.8 ± 23.6	81.3 ± 22.9	13.5 (8.4 – 18.6)	<0.001*
QTc interval (ms)	396.2 ± 33.7	384.4 ± 37.9	11.8 (0.6 – 23.0)	0.040*
RV1 amplitude (mv)	0.51 ± 0.25	0.33 ± 0.22	0.18 (0.13 – 0.24)	<0.001*

*p<0.05. Values are mean ± SD. Analyses were done using dependent t-test.

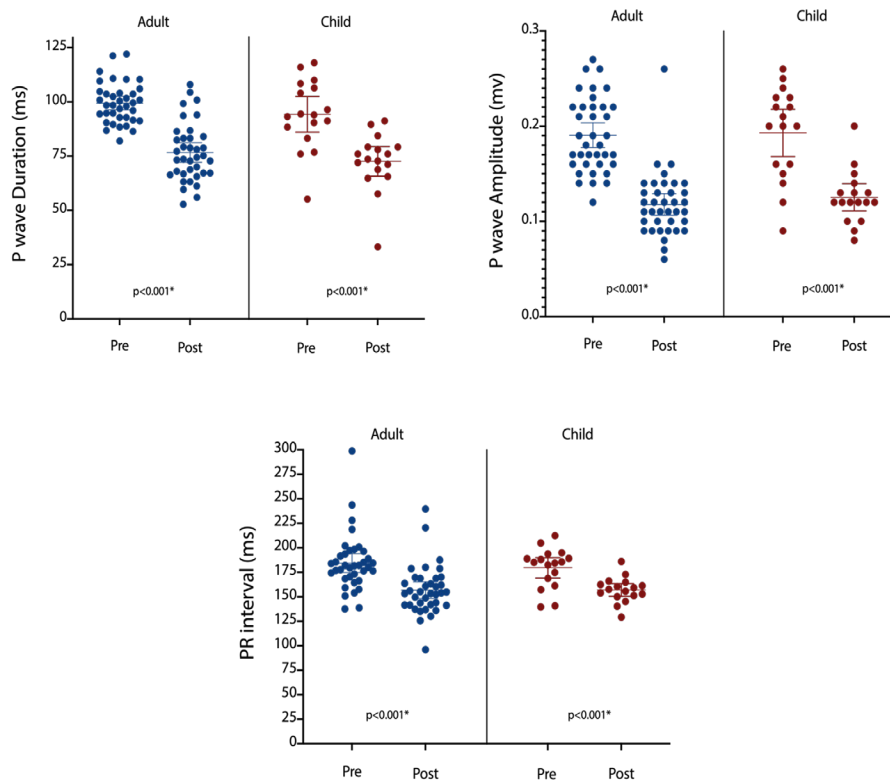


Figure 1. Subgroup analysis comparing ECG changes before and after ASD closure in adult vs. pediatric patients.

Discussion

This study demonstrates that closure of Secundum atrial septal defects induces rapid and sustained improvements in electrocardiographic parameters, reflecting the underlying structural and electrical remodeling of the right heart. Our findings highlight the dynamic interplay between anatomical reverse remodeling and electrophysiological normalization, emphasizing the utility of ECG not only as a diagnostic tool but also as a practical, non-invasive method to monitor post-procedural cardiac recovery. By evaluating both early (<24 hours) and late (>6 months) post-closure changes, this study provides novel insights into the timeline and magnitude of electrical remodeling, with important implications for patient management and long-term follow-up.

Electrocardiography is increasingly recognized not only as a diagnostic tool but also as a valuable modality for guiding and assessing outcomes in patients with Secundum ASD following intervention.⁶⁻⁸ Post-procedural ECG changes reflect the underlying anatomical and electrical remodeling that occurs after normalization of the right heart's volume load. This process, termed reverse remodeling, underpins the clinical benefits observed after ASD closure and begins almost immediately once the hemodynamic burden is relieved.^{6-9,10}

The link between structural and electrical remodeling is well established. Echocardiographic studies consistently demonstrate that ASD closure results in rapid and significant reductions in right atrial and right ventricular dimensions.^{9,11-12} For example, Bigdelu et al. (2023) reported marked decreases in right heart chamber size and pulmonary artery pressures within six months post-closure. As the right-sided chambers shrink, mechanical stretch on myocardial and conduction fibers is relieved, allowing normalization of conduction pathways, which is directly reflected on the ECG.¹³ The reductions in QRS duration^{6,8,13}, leftward shift of the QRS axis^{7,14}, and decreased R-wave amplitude in V1^{6,7} observed in our cohort correspond anatomically to the decreased right ventricular size and normalization of interventricular septal motion seen on echocardiography.

Advanced imaging modalities, such as cardiac magnetic resonance (CMR), have further corroborated this relationship. Mansour et al. (2022) demonstrated significant reductions in right atrial and ventricular volumes three months after ASD closure, which correlated closely with electrical remodeling observed on ECG.⁶ Our observation of a significant reduction in P-wave amplitude and duration within 24 hours post-closure reflects the rapid unloading and decrease in right atrial size. While P-wave

duration continued to improve at the >6-month follow-up, amplitude did not demonstrate further significant change, suggesting that the primary geometric remodeling occurs almost immediately. These findings are consistent with prior reports by Kamphuis et al. (2019).⁷ Recent studies have also highlighted the clinical relevance of P-wave dispersion, the difference between the maximum and minimum P-wave duration, as a marker of intra-atrial conduction heterogeneity.^{6,7,14} Evidence indicates that P-wave dispersion decreases significantly following ASD closure, potentially predicting a lower long-term risk of atrial arrhythmias.^{6,15-16}

The observed reduction in PR interval aligns with previous studies and reflects improved atrio-ventricular conduction as the right atrial geometry normalizes.^{6,14} Likewise, the progressive shortening of QRS duration at both early and late follow-up points indicates resolution of right ventricular conduction delay, often manifesting as a right bundle branch block pattern pre-closure.¹⁴ These electrical improvements are directly attributable to the reduction in right ventricular size and pressure, resulting in more synchronous and efficient ventricular depolarization.^{7,16}

The observed leftward shift of the QRS axis and the reduction in R-wave amplitude in lead V1 represent classic electrocardiographic markers of regressing right ventricular hypertrophy.^{7,16} These electrical changes closely reflect structural reverse remodeling of the right ventricle, as documented in echocardiographic and CMR follow-up studies after ASD closure.^{6,12} For instance, Saedi et al. (2022) reported significant improvements in right ventricular function using advanced echocardiographic parameters, including strain and strain rate, shortly after device closure, providing a functional correlate for the electrical changes observed. Resolution of these ECG abnormalities serves as a robust indicator of procedural success and is associated with favorable long-term outcomes.¹⁷

Our results underscore the utility of ECG as a straightforward, readily available, and informative method for tracking beneficial cardiac remodeling after ASD closure. Future prospective, multicenter investigations combining ECG and echocardiographic evaluations are needed to better define the relationship between structural and electrical reverse remodeling and to enhance post-procedural patient care.

This study has several limitations. As a single-center study, it is inherently susceptible to selection bias. A major limitation is the lack of concurrent long-

term echocardiographic data to directly correlate with the observed ECG changes; such correlations would have strengthened the link between electrical and structural reverse remodeling and represent an important avenue for future research. Additionally, more advanced ECG parameters, including P-wave dispersion and T-peak to T-end interval, were not analyzed. These metrics have been shown in recent studies to provide valuable prognostic information regarding arrhythmia risk following ASD closure.

Conclusion

Interventional closure of Secundum ASD induces significant and sustained electrical reverse remodeling, as demonstrated by improvements across nearly all ECG parameters. These changes occur immediately post-procedure and continue to evolve over the long term. Our findings highlight ECG as a simple, widely accessible, and valuable tool for monitoring favorable cardiac remodeling following ASD closure.

List of Abbreviations

aPTT	activated Partial Thromboplastin Time
ASD	Atrial Septal Defect
CMR	Cardiac Magnetic Resonance
ECG	Electrocardiography
INR	International Normalized Ratio
mPAP	mean Pulmonary Artery Pressure
PT	Prothrombin Time
PVRI	Pulmonary Vascular Resistance Index
PVR	Pulmonary Vascular Resistance
RAD	Right Axis Deviation
RAE	Right Atrial Enlargement
RBBB	Right Bundle Branch Block
RVH	Right Ventricular Hypertrophy
SGOT	Serum Glutamate Oxalat Transaminase
SGPT	Serum Glutamate Pyruvat Transaminase
TEE	Trans-esophageal Echocardiography
TTE	Trans-thoracic Echocardiography
WU	Wood Units

Ethical Clearance

All procedures performed in this study involving human participants were in accordance with the ethical standards of the institutional and/or national and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. No studies with animals was performed by any of the authors of this article.

Publication Approval

All authors consent to the publication of this manuscript.

Authors Contributions

YP, AHA, and ARB conceived the initial idea. ARB, YP, and MA conducted data collection. ARB and AQ managed the data, performed data cleaning, and conducted the statistical analysis. ARB prepared the initial manuscript. AQ made critical revisions, additional data analysis, and prepared the final submitted manuscript. YP, AHA, II, MA, TDN, and IM reviewed and provided feedback for further revisions. All contributing authors approved the final draft.

Acknowledgments

This research was supported by the Faculty of Medicine, Hasanuddin University and Dr. Wahidin Sudirohusodo Hospital's Cardiac Center.

Conflict of Interest

All of the authors declare that they have no conflict of interest.

Availability of Data and Materials

The datasets generated and/or analysed during this study are not publicly available since they contain private data owned by multiple centers which were involved in this study. However, subject data are available from the corresponding author on reasonable request.

Funding

This study was self-funded and there were no external funding sources.

Copyright/Permissions for Figures

Not applicable.

Generative AI and AI-Assisted Technologies in the Writing Process

During the preparation of this manuscript, ChatGPT (OpenAI, San Francisco, CA, USA) was used to assist with paraphrasing, proofreading, and

improving language clarity. The authors reviewed and edited all content generated by the tool, and take full responsibility for the final version of the manuscript.

References

1. Baumgartner H, Bonhoeffer P, De Groot NMS, et al. ESC Guidelines for the management of grown-up congenital heart disease (new version 2010). *Eur Heart J*. 2010;31(23):2915-57. doi:10.1093/eurheartj/ehq249
2. Torres AJ. Hemodynamic assessment of atrial septal defects. *Journal of Thoracic Disease*; Vol 10, Supplement 24 (September 2018): *Journal of Thoracic Disease (Atrial Septal Defect)*. Published online 2018. <https://jtd.amegroups.org/article/view/19475>
3. Rosas M, Attie F, Sandoval J, et al. Atrial septal defect in adults ≥ 40 years old: negative impact of low arterial oxygen saturation. *Int J Cardiol*. 2004;93(2):145-55. doi:[https://doi.org/10.1016/S0167-5273\(03\)00192-X](https://doi.org/10.1016/S0167-5273(03)00192-X)
4. Bayar N, Arslan Ş, Köklü E, et al. The importance of electrocardiographic findings in the diagnosis of atrial septal defect. *Kardiol Pol*. 2015;73(5):331-6. doi:10.5603/KP.a2014.0240
5. Di Bernardo S, Berger F, Fasnacht M, Bauersfeld U. Impact of right ventricular size on ECG after percutaneous closure of atrial septal defect with Amplatzer Septal Occluder. *Swiss Med Wkly*. 2005;135(43-44):647-51. doi:10.4414/smw.2005.11067
6. Mansour A, Gamal NM, Alaa Nady M, Demitry SR, Shams-Eddin H, El-Maghraby KM. Comparison of the early cardiac electromechanical remodeling following transcatheter and surgical secundum atrial septal defect closure in adults. *Egypt Heart J*. 2021;73(1):53. doi:10.1186/s43044-021-00174-5
7. Kamphuis VP, Nassif M, Man SC, et al. Electrical remodeling after percutaneous atrial septal defect closure in pediatric and adult patients. *Int J Cardiol*. 2019;285:32-39. doi:10.1016/j.ijcard.2019.02.020
8. Fumanelli J, Garibaldi S, Castaldi B, et al. Mid-Term Electrical Remodeling after Percutaneous Atrial Septal Defect Closure with GCO Device in a Pediatric Population. *J Clin Med*. 2023;12(19). doi:10.3390/jcm12196334
9. Kim G, Kil HR. Changes in reverse cardiac remodeling after percutaneous atrial septal

- defect closure in children and adults. *Congenit Heart Dis.* 2021;16(3):211-20. doi:10.32604/CHD.2021.013724
10. Mostafa S, Abdelhakim A, Aboelazm T, Arafa O, Elemam A. Effect of transcatheter closure of secundum atrial septal defect on cardiac electric remodeling. *International Journal of Heart Rhythm.* 2017;2(1):40. doi:10.4103/2352-4197.208453
 11. Etman I, ElZawawy T, Zaki A, ElEmam AM, AbdelAzeem AM. Echocardiographic insights of right heart dynamics after transcatheter ASD closure: A prospective study. *American Heart Journal Plus: Cardiology Research and Practice.* 2025;59:100632. doi:https://doi.org/10.1016/j.ahjo.2025.100632
 12. Bosshardt D, Voskuil M, Krings GJ, et al. Echocardiographic right ventricular remodeling after percutaneous atrial septal defect closure. *International journal of cardiology Congenital heart disease.* 2023;12:100459. doi:10.1016/j.ijchd.2023.100459
 13. Bigdelu L, Nezhad Biglari N, Ghaderi Y, et al. Trans-thoracic echocardiographic findings after the closure of ostium secundum atrial septal defect: A six-month follow-up study. *J Cardiovasc Thorac Res.* 2025;17(1):27-34. doi:10.34172/jcvtr.025.32997
 14. Santoro G, Pascotto M, Sarubbi B, et al. Early electrical and geometric changes after percutaneous closure of large atrial septal defect. *Am J Cardiol.* 2004;93(7):876-80. doi:10.1016/j.amjcard.2003.12.027
 15. Ahmed SH, Abdel-jaleil MA, Mahmoud SM. The Outcomes of Transcatheter Closure of Secundum Atrial Septal Defects on Cardiac Remodeling by Electrocardiography and Holter Study in Pediatric Population. *Egypt J Hosp Med.* 2024;97(1):4140-6. doi:10.21608/ejhm.2024.393206
 16. Elraouf MGA, Biomy R, Elsheikh R, Moawad A. Electrical Remodeling Following Atrial Septal Defect Closure in Adults: A Prospective Cohort Study. *Journal of Population Therapeutics and Clinical Pharmacology.* 2024;31(6):397-408.
 17. Saedi T, Firouzi A, Saedi S. Cardiac remodeling after atrial septal defects device closure. *Echocardiography.* 2022;39(8):1089-94. doi:10.1111/echo.15421