Electrophysiological predictors of propafenone efficacy in prevention of atrioventricular nodal re-entrant and atrioventricular re-entrant tachycardia

**Aim** To assess the efficacy of propafenone in prevention of atrioventricular nodal reentrant tachycardia (AVNRT) and orthodromic atrioventricular tachycardia (AVRT) based on the clinical results of arrhythmia recurrence and find the electrophysiological predictor of propafenone effectiveness.

**Methods** This retrospective study included 44 participants in a 12-month period, who were divided in two groups: group A – in which propafenone caused complete ventriculo-atrial block and group B – in which propafenone did not cause complete ventriculo-atrial block.

**Results** Group A had significantly lower incidence of tachycardia than group B (95% vs 70.8%, \( P = 0.038 \)), and complete ventriculo-atrial block predicted the efficacy of propafenone oral therapy in the prevention of tachycardia (sensitivity 87.5%, specificity 52.8%, positive predictive value 95%, negative predictive value 29.2%). Patients with AVNRT in group B who did not experience the recurrences of tachycardia had significantly shorter echo zone before intravenous administration of propafenone than the patients who experienced episodes of sustained tachycardia (median 40 ms [range 15-60 ms] vs 79 ms [range 50-180 ms], \( P = 0.008 \)).

**Conclusion** In patients with non-inducible tachycardia, complete ventriculo-atrial block can be used as an electrophysiological predictor of the efficacy of propafenone oral therapy in the prevention of tachycardia. In patients with non-inducible AVNRT, but without complete ventriculo-atrial block, propafenone was more effective in patients with shorter echo zone of tachycardia.

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Nowadays a leading method in treatment of supraventricular re-entrant tachycardias is radio-frequent (RF) catheter ablation (1-4). However, in some patients the method of choice in the prevention of tachycardia recurrence is treatment with antiarrhythmics (5). The medications used can be selected on the basis of the previous clinical experience or the results of the electrophysiological study (EPS) (6). A few groups of antiarrhythmics can be used in prevention of supraventricular re-entrant tachycardias, atrioventricular nodal re-entrant tachycardia (AVNRT) and atrioventricular re-entrant tachycardia (AVRT), such as: class IV (verapamil), lc (propafenone and flecainide), and II (beta-blockers) (7-9).

It is extremely important to establish the characteristics of patients’ clinical tachycardia, as well as the echo zone of the tachycardia (10,11). The medicines effective in patients with long-lasting echo zone are class lc antiarrhythmics (12,13), and in patients with shorter echo zone potassium channel blockers (sotalol, amiodarone), due to their strong effect on the refractory period of the accessory pathway (9,14). Propafenone is a class lc antiarrhythmic agent, with mild beta-blocking activity, affecting both pathways of AVNRT and orthodromic AVRT, thus causing retrograde or antegrade block (9). It has been extensively used in the treatment of a number of supraventricular tachyarrhythmias (7,15). In our previous study, we examined the safety and efficacy of intravenous propafenone in termination of both AVNRT and AVRT, as well as the electrophysiological effects of propafenone on cardiac conductivity (16).

The aim of this study was to examine the effectiveness of propafenone in the prevention of tachycardia (AVNRT and AVRT) recurrence with regard to several electrophysiological parameters. This has been done in previous similar studies, but they included fewer patients and electrophysiological findings were not evaluated as predictors of antiarrhythmic agent effectiveness (17,18).

PATIENTS AND METHODS

Design and settings

This retrospective, single-center study included 44 patients who had previously undergone electrophysiological study (EPS) followed by intravenous administration of propafenone at the Department of Cardiology, Sestre Milosrdnice University Hospital Center, Zagreb, Croatia between January 2004 and December 2005 and continued to receive propafenone oral therapy during a 12-month period after the EPS. These 44 patients were chosen from all the patients (70 patients) who underwent the electrophysiological procedure at the University Hospital Centre Sestre Milosrdnice, Department of Internal Medicine between January 2004 and December 2005 and among whom tachycardia could not be induced after iv. propafenone had been administered.

Electrophysiology study protocol and intervention

The study included a total of 44 patients with symptoms of two most common suparaventricular tachycardias, typical AVNRT and orthodromic AVRT, documented by 12-lead ECG, with minimally four episodes per year during the last three years. After taking their complete medical history, each patient underwent a thorough clinical examination, routine laboratory testing, chest x-ray, standard ECG recording, and echocardiogram.

Since they were diagnosed with tachycardia, all patients were treated with different antiarrhythmic drugs ranging from one to five antiarrhythmics (group A: median 4, range 1-5; group B: median 4, range 1-5, P = 0.256). The most frequently used antiarrhythmic was atenolol (in 47 patients, 67.1%). Thirty-four patients (48.6%) were treated with verapamil, 29 (41.1%) with sotalol, and 28 (40.0%) with amiodarone. Eight patients were previously treated with propafenone (11.4%), 4 with digoxin (5.7%), and one with disopyramide (1.5%). All antiarrhythmic drugs were discontinued at least 30 days before the EPS (19,20).

All patients underwent EPS according to standard protocol (19-21), using four 6-French, quadripolar electrodes positioned under fluoroscopy in the right atrium, right ventricular apex, coronary sinus, and bundle of His. Conduction intervals and refractory periods for EPS were defined in the conventional manner (19). Before starting the EPS, a written informed consent was obtained from each patient.

Tachycardia was induced by standard stimulation protocols using a Mingograph 62 Siemens stimulator (Elema AB, Stockholm, Sweden). Programmed atrial stimulation was the most frequent way of inducing tachycardia, whereas fast atrial and ventricular stimulation was the most frequent way of terminating tachycardia. Programmed ventricular stimulation induced tachycardia in 12 patients with orthodromic AVRT (36.4%) and 4 patients with AVNRT (10.8%), with the difference being significant (P = 0.011, t test proportion for independent samples). There was no significant difference in other types of tachycardia induction, which was induced easily by all applied methods (P > 0.05, t test proportion for independent samples).
Two techniques are used to evaluate retrograde ventriculo-atrial (VA) conduction in the evaluation of SVTs: burst pacing in the right ventricle and premature ventricular stimulation. Retrograde properties were evaluated by noting the longest ventricular paced cycle length that produced VA block and by measuring the ERP period of VA conduction (20).

The antegrade echo zone is defined as the zone of A1A2, at which A2 provokes atrial echoes with or without tachycardia. The average atrial echo zone is an interval from spontaneous atrial activity to atrial premature depolarization producing tachycardia (20,21).

The successful induction of tachycardia and determination of its mechanism were followed by intravenous administration of propafenone (2 mg/kg over 10 minutes) (19,20). The same electrophysiological protocol was repeated to assess tachycardia inducibility. During the procedure, vital parameters including blood pressure, ECG, and oxygen saturation were monitored. After the completion of EPS, standard ECG was recorded and compared to the one obtained prior to propafenone administration.

**Study protocol**

The exclusion criteria for EPS and intravenous administration of propafenone were the following: bradycardia, AV block, sinus node dysfunction, hypotension, coronary artery disease, cerebrovascular disease, left ventricular hypertrophy, reduced left ventricular systolic function, renal or liver failure, hypokalemia, and pregnancy.

After the completion of EPS, each patient was prescribed individualized per os antiarrhythmic therapy aimed to prevent tachycardia. Forty-four participants were included into a retrospective study after a 12-month period after EPS and were divided in two groups: group A – in which propafenone caused complete VA block and group B – in which propafenone did not cause complete VA block.

Propafenone was considered to be a drug of choice as continuous oral therapy in patients in whom tachycardia was non-inducible after its intravenous administration during the EPS. In patients in whom iv. propafenone during EPS did not prevent tachycardia (22/66 patients, 33%) per os therapy with atenolol, sotalol, or amiodarone was prescribed.

Patients were divided into two groups according to VA block because it was shown that completing VA block could represent a significant electrophysiological predictor of propafenone efficiency in tachycardia prevention (16,22,23).

During the period of 12 months, patients had regular controls every 3 months, which included medical history taking, physical examination, ECG recording, and 24-hour continuous ECG recording. Our focus was on the recurrence of clinical tachycardia, that is, on the efficacy of propafenone in its prevention, as well as possible side-effects of propafenone. If the patient was asymptomatic during this period, propafenone was considered to be effective. Recurrence of clinical tachycardia required discontinuation of propafenone. Moreover, several electrophysiological parameters were identified as predictors of a good clinical outcome (absence of tachycardia).

**Statistical analysis**

Quantitative data are presented as median values and ranges and qualitative data by contingency tables as absolute values and percentages. The differences in quantitative variables between the groups were tested by Mann-Whitney U-test (non-parametric test for independent samples). The relation between qualitative variables and patients’ groups was tested by χ² test with Yates corrections for Tables 2 × 2. The differences in frequencies of certain variables between the groups were tested by t test proportion for small independent samples. The differences in quantitative variables before and after drug treatment were tested by Wilcoxon test for pair samples (non-parametric test for dependent samples). The effect of certain electrophysiological parameters on the prevention of tachycardia by propafenone treatment was evaluated on the basis of sensibility and reliability test. The effect of variables on relative risk of the prevention of tachycardia by propafenone treatment was evaluated by logistic regression and presented as odds ratio (OR) with 95% confidence intervals (CI). Statistical analysis was carried out using the Statistica 6.0 software (24). A value of P < 0.05 was considered statistically significant.

**RESULTS**

The study included 44 participants who continued to receive propafenone oral therapy (450-900 mg) after they had undergone EPS. Patients in group A received on average 669 ± 79 mg of propafenone in comparison with patients in group B who received 678 ± 168 mg (P = 0.569). After a period of one year, 36 patients...
(82%) continued to receive propafenone therapy, which was prescribed if tachycardia was non-inducible after its intravenous administration during the EPS. Nineteen patients in group A (95%) were asymptomatic and continued to receive antiarrhythmic therapy (propafenone), whereas 1 patient had tachycardia and therapy was terminated. In group B, 7 patients (29%) had palpitations leading to the termination of recommended propafenone therapy, while 17 patients (71%) were asymptomatic (Table 1). Group A had significantly lower incidence of tachycardia than group B ($P = 0.038$; $t$ test proportion). Complete VA block predicted the efficacy of propafenone in the prevention of tachycardia (sensitivity 87.5%, specificity 52.8%, positive predictive value 95%, negative predictive value 29.2%) (Table 2).

Despite recommendations, 6 out of 22 patients in whom iv. propafenone during EPS did not prevent tachycardia (27%, 2 with AVNRT and 4 with AVRT) continued with the propafenone therapy. One out of 6 patients during the monitoring period of per os propafenone therapy (2 $\times$ 300 mg/per day) did not show the symptoms of tachycardia. Propafenone therapy was interrupted for the remaining 5 patients considering the recurrence of tachycardia.

After the patients with AVNRT in the group B (16 patients) were studied in isolation, we noticed that 11 patients (69%) did not have recurrent tachycardia. Compared with patients with AVNRT who had palpitations (5/16 patients, 31%), they had shorter echo zone during EPS before the intravenous propafenone administration, 40 ms (15-60 ms) vs 79 ms (50-180 ms) ($P = 0.008$).

Side-effects of propafenone therapy occurred in 9 out of 44 patients (21%) and included the following: mild gastrointestinal upset, blurred vision, and constipation. However, there was no need to discontinue propafenone in any patient because of the side-effects. No patient showed proarhythmic effect.

**DISCUSSION**

The retrospective, single-center study studied a sample of patients with the two most common supraventricular tachycardias.

### Table 1. Demographic and clinical parameters in the group A (with complete ventriculo-atrial block) and group B (without complete ventriculo-atrial block)

| Demographic and clinical data | Group A | Group B | $P$  |
|------------------------------|---------|---------|------|
| Age in years (median, range) | 44 (18-70) | 47 (20-67) | 0.301* |
| Symptom duration in months (median, range) | 146 (44-450) | 99 (38-480) | 0.126* |
| Sex, n (%): | | | |
| male | 7 (35) | 10 (42) | 0.197† |
| female | 13 (65) | 14 (58) | 0.197† |
| Heart disease (any, except those mentioned in exclusion criteria), n (%) | | | |
| Therapy (antiarrhythmics) | 10 (50) | 11 (46) | 0.425‡ |
| Symptons, n (%): | | | |
| presyncope | 8 (40) | 6 (25) | 0.191† |
| syncope | 5 (25) | 5 (21) | 0.098§ |
| chest pain | 0 | 1 (4) | 0.234‡ |
| palpitations | 18 (90) | 23 (96) | 0.321§ |

*Mann-Whitney U test. †$t$ test. ‡$t$ test. §$t$ test.

### Table 2. Sensitivity and specificity of complete ventriculo-atrial (VA) block as a predictor of efficacy of propafenone in prevention of tachycardia*

| Tachycardia is non-inducible, complete VA block | Propafenone effective | Propafenone ineffective | Total |
|-----------------------------------------------|-----------------------|-------------------------|-------|
| Tachycardia is non-inducible, without complete VA block | 19 | 1 | 20 |
| Total | 17 | 7 | 24 |

*Sensitivity = 87.5%; specificity = 52.8%; positive predictive value = 95%; negative predictive value = 29.2%.
Electrophysiological predictors of propafenone efficacy in AVNRT and AVRT during the EPS, propafenone was more effective in the prevention of tachycardia in patients with achieved complete VA block. Therefore, the complete VA block can be used as an electrophysiological predictor of propafenone efficacy in the prevention of tachycardia recurrence.

Furlanello et al performed transesophageal EPS in 58 patients, with administration of class Ic antiarrhythmics (propafenone and flecainide) (27). Their study showed that these medications were extremely effective in the prevention of supraventricular tachycardias, if arrhythmia was non-inducible during the EPS after the drug application, with effectiveness of prevention of 65.5%, which is less than in our study (81.8%). Breithardt et al reported that propafenone was a very potent agent in the prevention of supraventricular tachycardias in patients with Wolf-Parkinson-White syndrome, independently of the results of EPS (28).

It is noteworthy that in adults with supraventricular re-entrant tachycardias, RF ablation during EPS has become the first-line treatment approach with a high acute success and low complication rate (3,4,29,30). However, Vassilikos et al showed that non-cardiologists (general practitioners and internists) who are involved in the acute and long-term management of SVT rely more on antiarrhythmic drugs and tend to underestimate the role of ablation therapy for the long-term management of SVT (31). RF ablation therapy is increasingly used in pediatric patients with SVTs, however, antiarrhythmic therapy is still the first choice (usually guided on the results of transesophageal atrial pacing technique), in which propafenone is one of the first choices and has good efficacy in termination of tachycardias (32,33). All this points to the importance of antiarrhythmics for the treatment of supraventricular tachycardias, and therefore to the importance of propafenone, which has proven to be efficient in our study as well as several previous studies, although they included a small number of patients (23,27,28). According to the literature overview, indication for continuous antiarrhythmic therapy is non-inducibility of tachycardia after antiarrhythmic agent administration (15,17,18).

Regarding the effectiveness of other groups of antiarrhythmics, Komatsu et al tested the effectiveness of four antiarrhythmics on the induction and termination of supraventricular re-entrant tachycardias (22). Verapamil was effective in about 65% patients, procainamide in somewhat more (around 75%), whereas propranolol and disopyramide were not particularly effective (under 40% overall). Our study showed better effectiveness for propafenone.

In addition, we tried to find the electrophysiological parameter that predicted propafenone effectiveness in patients in whom the complete VA block during EPS was not achieved. According to our results, in patients with AVNRT and without achieved complete VA block, propafenone was more effective in the prevention of tachycardia in patients with shorter echo zone of tachycardia prior to intravenous propafenone administration during EPS.

There are certain limitations of the study: it is a retrospective single-center study performed on a small group of patients. Moreover, the data are largely descriptive and due to the small number of patients, statistical analysis could not be performed between certain groups.

In conclusion, oral therapy with propafenone is more effective in the prevention of tachycardia recurrence in patients with achieved complete VA block during EPS. Complete VA block, achieved after intravenous administration of propafenone during the EPS, can be used as an electrophysiological predictor of efficacy of propafenone in the prevention of tachycardia recurrence.

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Declaration of authorship HP substantially contributed to study conception and design as well as data acquisition, analysis, and interpretation, wrote all parts of the manuscript and gave the final approval. ZB participated in conception and design of the study, data interpretation, data acquisition, revision of the results, and gave the final approval. LV contributed to data collection, revision of the results, making the conclusions, revision of the manuscript, and gave the final approval. LZ participated in study design, data acquisition, data interpretation, drafting of the manuscript, contributed substantially to the literature search, and gave the final approval. NP participated in designing the methods, analyzing and interpreting the results, preparing the manuscript, and gave the final approval. HP contributed to data collection, participated in interpretation of data, revision of the manuscript, and gave the final approval. IZ participated in study design, data acquisition, data interpretation, drafting of the manuscript, contributed substantially to the literature search, and gave the final approval. DH contributed to data collection, participated in interpretation of data, revision of the manuscript, and gave the final approval. IZ participated in study design, data acquisition, data interpretation, drafting of the manuscript, contributed substantially to the literature search, and gave the final approval.

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References

1. Sorbora C, Cohen M, Woolfe P, Kalapatapu SR. Atrioventricular nodal reentry tachycardia: Slow pathway ablation using the transeptal approach. Pacing Clin Electrophysiol. 2000;23:1281-4. Medline:11025889 doi:10.1111/j.1540-8159.2000.tb00961.x

2. Altemose GT, Scott LR, Miller JM. Atrioventricular node reentrant tachycardia requiring ablation of the miter annulus. J Cardiovasc Electrophysiol. 2000;11:1281-95. Medline:11083250 doi:10.1046/j.1540-8167.2000.01281.x

3. Iyer R, Hameed S, Vora AM, Lokhandwala Y. Radiofrequency ablation: a cure for tachyarrhythmias. J Indian Med Assoc. 2000;98:684-7. Medline:11265797

4. Feldman A, Viskoboinik A, Kumar S, Spence S, Morton JB, Kistler PM, et al. Predictors of acute and long-term success of slow pathway ablation for atrioventricular nodal reentrant tachycardia: a single center series of 1,419 consecutive patients. Pacing Clin Electrophysiol. 2011;34:927-33. Medline:21569056 doi:10.1111/j.1540-8167.2011.03092.x

5. Wyse DG, Talajic M, Hafley GE, Buxton AE, Mitchell LB, Kus TK, et al. Antiarrhythmic drug therapy in the Multicenter UnSustained Tachycardia Trial (MUSTT). Drug testing and as-treated analysis. J Am Coll Cardiol. 2001;38:344-51. Medline:11499722 doi:10.1016/S0735-1097(01)01402-4

6. Ludertiz B, Manz M. Pharmacologic treatment of supraventricular tachycardia: the German experience. Am J Cardiol. 1992;70:66A-73A. Medline:1510002 doi:10.1016/0002-9149(92)91081-e

7. Trohman RG. Supraventricular tachycardia: implications for the intensivist. Crit Care Med. 2000;28:N129-35. Medline:11055681 doi:10.1097/00003156-200001001-00004

8. Blomström-Lundqvist C, Scheinman MM, Aliot EM, Alpert JS, Calkins H, Camm AJ, et al. ACC/AHA/ESC guidelines for the management of patients with supraventricular arrhythmias-executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and the European Society of Cardiology Committee for Practice Guidelines (Writing Committee to Develop Guidelines for the Management of Patients With Supraventricular Arrhythmias). Circulation. 2003;108:1871-909. Medline:14557344 doi:10.1161/01.CIR.0000091380.04100.84

9. Opie LH, Gersh BJ. Drugs for the heart. 7th ed. Saunders Elsevier Inc.Philadelphia. 2009.

10. Stanton MS. Class I antiarrhythmic drugs: Quinidine, procainamide, disopyramide, lidocaine, mexiletine, flecainide and propafenone. In: Zipes DP, Jalife J, editors. Cardiac Electrophysiology: from cell to bedside, 3rd ed. Philadelphia (Pa): WB Saunders; 2004. p. 911-7.

11. Kuck KH, Cappato R, Siebels J, Ruppel R. Randomized comparison of antiarrhythmic drug therapy with implantable defibrillators in patients resuscitated from cardiac arrest: The Cardiac Arrest Study Hamburg (CASpH). Circulation. 2000;102:748-54. Medline:10942742 doi:10.1161/01.CIR.102.7.748

12. Members of the Sicilian Gambit. The concept of the vulnerable parameter and its application to specific arrhythmias. In: Members of the Sicilian Gambit, editors. Antiarrhythmic therapy: a pathophysiological approach. Armonk (NY): Futura Publishing Co Inc.; 1994. p. 121-7.

13. Jazwinska-Tarnawska E, Orzechowska-Juzwenko K, Niewinski P, Rzemislawska Z, Luboz-Grudzien K, Dmochowska-Perz M, et al. The influence of CYP2D6 polymorphism on the antiarrhythmic efficacy of propafenone in patients with paroxysmal atrial fibrillation during 3 months propafenone prophylactic treatment. Int J Clin Pharmacol Ther. 2001;39:288-92. Medline:11471772

14. Sarubbi B, Ducceschi V, Esposito R, Briglia N, Manzo G, Mayer MS, et al. The pharmacological treatment of supraventricular atrioventricular nodal reentry or accessory pathway reentry tachycardias: the usefulness of transesophageal electrophysiological study in the selection of therapy. Cardiologia. 1996;41:543-9. Medline:8766417

15. Connors S, Dorian P. Management of supraventricular tachycardia in the emergency department. Can J Cardiol. 1997;13:19A-24A. Medline:9117922

16. Pintaric H, Manola S, Szavits Nossan J, Pavlovic N, Delic-Birkljacic D, Radijelic V. Electrophysiological effects, efficacy and safety of intravenous propafenone in termination of atrioventricular nodal reentrant tachycardia and atrioventricular reentrant tachycardia: a prospective non-randomized interventional study. Acta Clin Croat. 2010;49:25-31. Medline:20635581

17. Manz M, Steinbeck G, Ludertiz B. Usefulness of programmed stimulation in predicting efficacy of propafenone in long-term antiarrhythmic therapy for paroxysmal supraventricular tachycardia. Am J Cardiol. 1985;56:593-7. Medline:4050693 doi:10.1016/0002-9149(85)91017-3

18. Manolis AS, Katsaros C, Cokkinos DV. Electrophysiological and electropharmacological studies in pre-excitation syndromes: results with propafenone therapy and isoproterenol infusion testing. Eur Heart J. 1992;13:1489-95. Medline:1361174

19. Griffin BP, Topol EJ. Manual of cardiovascular medicine 3rd edition, Philadelphia (Pa): Lippincott, Williams and Wilkins, Wolters Kluwer Health; 2009. p. 673-99.

20. Petrač D. Electrophysiological testing of the heart. U: Barić lJ, Petrač D, Birtić K, editors. Electrocardiography in practice (in Croatian). Zagreb: Tiskara Znanje; 2003. p. 417-28.

21. Kou HC, Hung JS, Lee YS, Wu D. Effects of oral disopyramide phosphate on induction and sustenance of atrioventricular reentrant tachycardia incorporating retrograde accessory pathway conduction. Circulation. 1982;66:454-62. Medline:6807567 doi:10.1161/01.CIR.66.2.454

22. Komatsu C, Ishinaga T, Tateishi O, Tokuhisa Y, Yoshimura S. Effects of four antiarrhythmic drugs on the induction and termination of
paroxysmal supraventricular tachycardia. Jpn Circ J. 1986;50:961-72. Medline:3806932 doi:10.1253/jcj.50.961

23 Hammill SC, McLaran CJ, Wood DL, Osborn MJ, Gersh BJ, Holmes DR. Double-blind study of intravenous propafenone for paroxysmal supraventricular reentrant tachycardia. J Am Coll Cardiol. 1987;9:1364-8. Medline:3584724 doi:10.1016/S0735-1097(87)80479-5

24 StatSoft. Inc.: STATISTICA data analysis software system version 6.0 (for Windows). Tulsa, OK: StatSoft Inc; 2000.

25 Campbell RW. Supraventricular tachycardia. Occasional nuisance or frequent threat? Eur Heart J. 1996;17 Suppl C:21-5. Medline:8809535 doi:10.1093/eurheartj/17.suppl_C.21

26 Orejarena LA, Vidalilet H Jr, DeStefano F, Nordstrom DL, Vierkant RA, Smith PN, et al. Paroxysmal supraventricular tachycardia in the general population. J Am Coll Cardiol. 1998;31:150-7. Medline:9426034 doi:10.1016/S0735-1097(97)00422-1

27 Furlanello F, Guanerino M, Inama G, Vergara G, Del Greco M, Bertoldi A, et al. Long-term follow-up of patients with inducible supraventricular tachycardia treated with flecainide of propafenone: therapy guided by transesophageal electropharmacologic testing. Am J Cardiol. 1992;70:19A-25A. Medline:1509994 Medline:1509994 doi:10.1016/0002-9149(92)91073-D

28 Breithardt G, Borggreve M, Weibinghaus E, Seipel L. Effect of propafenone in the Wolf-Parkinson-White syndrome: electrophysiologic findings and long-term follow-up. Am J Cardiol. 1994;74:29D-39D. Medline:6496367 doi:10.1016/S0002-9149(84)80283-0

29 Parizek P, Haman L, Dostalova H, Duda J, Popelka J. Long-term outcome of catheter ablation therapy of supraventricular tachyarrhythmias. Vnitr Lek. 2011;57:546-50. Medline:21751540

30 Ballin SJ, Korthas MA, Weers NJ, Hoffman CJ. Direct visualization of the slow pathway using voltage gradient mapping: a novel approach for successful ablation of atrioventricular nodal reentry tachycardia. Europace. 2011;13:1188-94. Medline:21508003 doi:10.1093/europace/eur112

31 Vassilikos VP, Mantziari L, Goudis CA, Paraskevaidis S, Dakos G, Stavropoulos G, et al. Practice patterns of cardiologists, general practitioners, and internists for managing supraventricular tachycardias in Greece. Eur J Emerg Med. 2011;18:153-6. Medline:21099433 doi:10.1097/MEJ.0b013e328341ee61

32 Drago F, Silvetti MS, De Santis A, Marcara S, Fazio G, Anacletio S, et al. Paroxysmal reciprocating supraventricular tachycardia in infants: electrophysiologically guided medical treatment and long-term evolution of the re-entry circuit. Europace. 2008;10:629-35. Medline:18388122 doi:10.1093/europace/eun069

33 Hafez M, Abu-Elkheir M, Shokier M, Al-Marsafawy H, Abo-Haded H, El-Maaty MA. Radiofrequency catheter ablation in children with supraventricular tachycardias: intermediate term follow up results. Clin Med Insights Cardiol. 2012;6:7-16. Medline:22259261 doi:10.4137/CMI.S8578