

# Transesophageal electrophysiologic study in children and young patients

Sema Özer, Alpay Çeliker, Tevfik Karagöz, Engin Melek

Section of Cardiology, Department of Pediatrics, Hacettepe University Faculty of Medicine, Ankara, Turkey

**SUMMARY:** Özer S, Çeliker A, Karagöz T, Melek E. Transesophageal electrophysiologic study in children and young patients. Turk J Pediatr 2007; 49: 45-51.

Transesophageal electrophysiologic study (TEEPS) is a semi-invasive method of atrial stimulation and recording. The aim of the study was to report our experience with TEEPS in children and young adults.

A total of 153 TEEPS were performed in 147 consecutive patients aged between 26 days to 26 years (mean 9.8 years) with the following indications: evaluation of symptoms that may be signs of any arrhythmias in 89 procedures (Group A), risk assessment of Wolff-Parkinson-White syndrome (WPW) in 17 procedures (Group B), determination of the mechanism of previously detected or ongoing tachycardia on ECG or Holter monitoring in 22 procedures (Group C), assessment of antiarrhythmic therapy effectiveness in 17 procedures (Group D), and follow-up of radiofrequency ablation procedure (RFA) in 8 procedures (Group E). A similar pacing protocol was performed for induction of tachycardia in each patient.

Tachycardia was induced in a total of 72 procedures (72/153, 47%): 32/89 (36%) in Group A, 13/17 (76.5%) in Group B, 12/22 (54.5%) in Group C, 12/17 (70.6%) in Group D and 3/8 (37.5%) in Group E. In Group A, the ventriculoatrial (VA) interval of inducible tachycardia was found to be shorter than 70 msec in 16/32 (50%) and longer than 70 msec in 12/32 (37.5%) patients and these patients were diagnosed as having atrioventricular nodal reentrant tachycardia (AVNRT) and atrioventricular reentrant tachycardia (AVRT), respectively. In this group, 1 atrial tachycardia, 2 junctional ectopic tachycardia, 1 sinus node reentrant tachycardia and 1 permanent junctional reciprocating tachycardia (PJRT) were also diagnosed.

In conclusion, transesophageal atrial stimulation is a valuable tool in the initial evaluation of patients with symptoms possibly related with arrhythmia or in the management of patients who have any arrhythmia.

*Key words:* transesophageal atrial pacing, palpitation, syncope, children.

Transesophageal atrial pacing is a simple and semi-invasive method that is useful for the diagnosis of supraventricular tachycardias (SVT) and for many other indications<sup>1,2</sup>. It allows precise assessment and management of supraventricular arrhythmias without using the endocavitary route. The inducibility and causative mechanism of the induced tachycardia has shown a good correlation to findings at a subsequent invasive electrophysiological study<sup>3-10</sup>. We report our results of transesophageal atrial pacing routinely performed as a screening procedure on patients with suspected SVT,

for risk stratification of Wolff-Parkinson-White (WPW) syndrome, in the follow-up of patients who have undergone radiofrequency ablation (RFA) procedure, and to assess effectiveness of antiarrhythmic therapy in children.

## Material and Methods

Between July 2002 and July 2004, we performed a total of 153 transesophageal electrophysiologic studies (TEEPS) on 147 patients aged between 27 days to 26 years (mean 9.8 years). Indications were: evaluation of symptoms that may be the signs of any arrhythmias

in 89 (58.2%) procedures (Group A), risk assessment of WPW syndrome in 17 procedures (Group B), determination of the mechanism of previously detected or ongoing tachycardia on ECG or Holter monitoring in 22 procedures (Group C), control of antiarrhythmic therapy effectiveness in 17 procedures (Group D), and follow-up of RFA procedure in 8 procedures (Group E) (Table I). Fourteen patients had structural abnormalities of the heart. A standard electrocardiogram was obtained in all patients. Dysrhythmia was detected on previous or current standard 12-lead ECG or Holter monitoring in 22 patients. Seventeen patients had WPW syndrome pattern. Holter monitoring was performed in 73 patients - 3 had SVT, 4 had frequent supraventricular ectopy (SVE) and 1 had rare ventricular ectopy (VE). Exercise testing was performed in 65 patients - all were normal. In 1 patient with WPW syndrome, preexcitation disappeared during exercise testing.

lidocaine in all patients. Cardiac stimulation was accomplished with a Fiab Programmable Cardiac Stimulator 8817 with a pulse width and amplitude capacity between 5-20 msec and 5-45 mA consecutively. A standard ECG machine was used for recording. A similar pacing protocol was performed in each patient. The protocol consisted of 1) 1 extrastimulus at progressively closer intervals after an 8 beat pacing train at 500 and 430 msec cycle lengths and 2) incremental atrial pacing to the point of second-degree atrioventricular block. If tachycardia was not initiated, the protocol was repeated after atropine and/or isoproterenol (0.05-0.1 µg/kg/min) infusion. The end point of the procedure was either induction of tachycardia or completion of the protocol. Apart from standard programmed stimulation, burst pacing in high rates was also performed, especially in patients with WPW syndrome, to induce atrial fibrillation.

**Table I.** Clinical Characteristics of the Patients and TEEPS Results

Indications for TEEPS	Number of TEEPS in 147 patients	Number of inducible tachycardia (%)
Palpitation	61	27 (44.3)
Palpitation-syncope	18	4 (22.2)
Palpitation-chest pain	10	1 (10)
Risk assessment of WPW	17	13 (76.5)
Evaluation of the tachycardia mechanism	22	12 (54.5)-inducible 10 (45.5)-spontaneous
Control of drug efficacy	17	12 (70.6)
Evaluation of recurring palpitation after RFA	8	3 (37.5)
Total	153	72 (47)-inducible 10 (6.5)-spontaneous

TEEPS: Transesophageal electrophysiologic study. WPW: Wolff-Parkinson-White syndrome. RFA: Radiofrequency ablation.

A TEEPS was performed as previously described by Benson et al.<sup>6</sup> in all patients as an outpatient procedure after at least four hours of fasting. Before starting the procedure, all patients were given oral (0.3 to 1.0 mg/kg) or i.v. (0.05-0.1 mg/kg) midazolam for relief of anxiety and sedation. The possible discomfort induced by programmed stimulation was explained to all patients and/or family. A 6 Fr quadripolar electrode catheter (Fiab, Esokid 4) with electrode spaced at 10 mm was introduced through the nose into the esophagus and fixed in a position where the largest atrial electrogram was recorded. Before insertion, the tip of the catheter was coated with 1%

We also looked for the sinus node function in patients presenting with syncope. This was determined by performing atrial stimulation at a rate slightly faster than the sinus rate. Pacing was continued at a constant rate for at least 30 sec and then was abruptly stopped. The recovery interval (the interval from the last atrial pacing to the first spontaneous sinoatrial nodal depolarization) represented the degree of overdrive suppression induced by pacing<sup>11</sup>.

#### **Mechanism of Tachycardia**

Atrioventricular reentrant tachycardia (AVRT) was presumed to be present under the condition of regular rhythm, no evidence of

AV dissociation, and a ventriculoatrial (VA) interval  $\geq 70$  msec. Atrioventricular nodal reentrant tachycardia (AVNRT) was presumed to be present under the condition of regular rhythm without evidence of AV dissociation and a VA interval  $< 70$  msec.

**Results**

In addition to midazolam, ketamine anesthesia was used in 5 (3.2%) patients since the procedure was not tolerated. As a result, TEEPS could be successfully performed in all patients. Patient ages were between 0-2 years in 14, 2-6 years in 22, 7-12 years in 63, 13-18 years in 42, and over 18 years in 6 patients. There were 78 females and 69 males. The mean ( $\pm$ SD) procedure time was 22.5 (7.7) minutes. The pacing duration and amplitude were  $16.5 \pm 2.4$  msec and  $16.4 \pm 2.5$  mA, respectively.

Tachycardia was induced in a total of 72 procedures (72/153, 47%): 32/89 (36%) in Group A, 13/17 (76.5%) in Group B, 12/22 (54.5%) in Group C, 12/17 (70.6%) in Group D and 3/8 (37.5%) in Group E (Table I) (Fig. 1).

(20.2%), and tachypnea, irritability, fatigue and pallor in 2 (2.2%) patients of Group A. Tachycardia was induced in only 10% and 22.2% of the patients presenting with chest pain plus palpitation and syncope plus palpitation, respectively, while it was induced in 45.8% of the patients with palpitation only. In only 2 patients - 1 with Ebstein’s anomaly having AVRT and 1 with PJRT - cardiomegaly was detected by telecardiogram.

In all those presenting with syncope accompanying palpitation, the tilt test was performed and found to be normal. The QTc intervals obtained from ECG and Holter recordings in all of them were also normal. The mean sinus node recovery time (SNRT) and corrected sinus node recovery time (CSNRT) values were found to be  $731.7 \pm 191.1$  and  $175.5 \pm 112.4$  msec, respectively. The SNRT and CSNRT were normal in all except 1, and this patient was diagnosed as having sick sinus syndrome.

In Group B, while 10 of them were asymptomatic, 6 had palpitation and 1 had syncope. Tachycardia was induced in 76.5% of

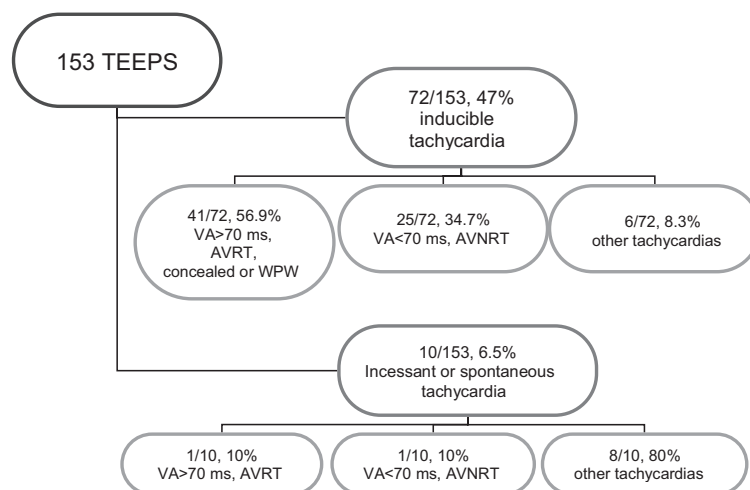


Fig. 1. Mechanism of the tachycardias determined by transesophageal electrophysiologic study (TEEPS). VA: Ventriculoatrial interval during tachycardia. AVRT: Atrioventricular reentrant tachycardia. AVNRT: Atrioventricular nodal reentrant tachycardia. WPW: Wolff-Parkinson-White syndrome.

In Group A, inducible tachycardias were diagnosed as AVNRT in 16/32 (50%), AVRT in 12/32 (37.5%) patients, 1 atrial tachycardia, 2 junctional ectopic tachycardia (JET) and 1 permanent junctional reciprocating tachycardia (PJRT). The presenting symptom was palpitation in 59 (66.3%), chest pain with palpitation in 10 (11.2%), syncope with palpitation in 18

these patients. In all of them, the VA interval was found to be longer than 70 msec, and atrial fibrillation was induced in none. In 5 of the patients, the accessory pathway effective refractory period (APERP) was found to be shorter than 250 msec. In 1 of these 5 patients, who presented with syncope, tachycardia could not be induced. Including this patient,

RFA was performed successfully in 2. In the intracardiac electrophysiologic study prior to ablation, the APERPs of these 2 patients were found to be similar with the results of TEEPS. Three of these 5 patients were followed with antiarrhythmic therapy since 2 of them were in the infantile age group and the parents of 1 did not accept the ablation.

In Group C, the presenting symptom was palpitation in 15, breath holding in 1, post-operative tachycardia in 1, palpitation with syncope in 1, chest pain with palpitation in 1 and tachypnea and pallor in 2 patients, while 1 had no complaint. In addition to inducible tachycardia in 12 patients in this group, spontaneous dysrhythmia was observed in the remaining 10 patients (45.5%) during the procedure. Five of these inducible tachycardias were diagnosed as AVNRT, while 6 were diagnosed as AVRT and 1 as atrial flutter. Patients with spontaneous dysrhythmias were diagnosed as AVNRT in 1, JET in 2, ectopic atrial tachycardia in 2, atrial flutter in 3, AVRT in 1 and accelerated idioventricular rhythm in 1 patient.

In Group D, tachycardia was induced in 12 of 17 patients; however, in 4 of them the tachycardia cycle length was prolonged significantly when compared to that at the initial procedure or clinically documented tachycardia, and in 2 of them tachycardia was non-sustained.

In Group E, TEEPS was performed in 8 patients to evaluate palpitation recurring after RFA performed for AVNRT in 4, WPW in 1 and AVRT in 3 patients, and tachycardia was induced by TEEPS in 3 of them. One was the patient with WPW syndrome, in whom a concealed accessory pathway was discovered. The second was the patient with AVNRT, in whom a non-sustained (5 sec) atrial tachycardia was induced. The third was the patient with another AVNRT, in whom a non-sustained AVNRT was induced.

Of all of our patients, in 6 (42.9%) from the infantile age group (n=14), tachypnea, irritability, fatigue and pallor indicating congestive heart failure were noted by the parents. In 12 of these 14 patients, tachycardia was induced. Ten of these inducible tachycardias (83.3%) were AVRT while the remaining 2 were JET. One of these JET diagnoses was made in a patient immediately after an operation for congenital heart disease.

## Discussion

Transesophageal electrophysiologic study is a semi-invasive method of diagnosing and treating arrhythmias. Though formerly used frequently in the treatment and diagnosis of SVT, it is currently used to assess the function of the sinus and AV nodes, to diagnose, initiate and bring to an end supraventricular and some ventricular tachycardia, to assess the effectiveness of the antiarrhythmic drugs and finally to evaluate those patients whose symptoms could be due to arrhythmias<sup>2</sup>. In the present study, in addition to the indications mentioned above, TEEPS was also used for evaluation of patients with recurrent symptoms after RFA.

If we look at the present study and the literature, although heart failure findings can be seen in infants more than older children during SVT, the most frequent presenting complaint was palpitation in older children. It is important to take an ECG recording in a patient presenting with palpitation during the complaint. Since the duration of a single SVT attack is usually quite short and infrequent, the possibility of recording a SVT attack on a standard ECG recording is very low. In many patients with possible SVT attack, it is sometimes impossible to document etiology even after detailed work-up including surface ECG, echocardiography, telecardiogram, Holter monitoring, exercise test, tilt test and event recorder for months and years. Therefore, in such patients, it is common to have repeated and extensive investigations without precise diagnosis for a long period. As a result, this kind of approach may be time-consuming and costly. By using TEEPS in the present and previously published studies, in 36-71% of the patients presenting with symptoms including palpitation alone and palpitation plus chest pain and syncope, SVT was induced<sup>2,4</sup>. In other words, etiology can be clarified precisely in a short time using TEEPS.

The information gathered from the present and previously published studies shows that in pediatric patients, SVT due to an accessory pathway is more common than through the AV nodes, and the number of individuals with AVNRT dramatically increases with age<sup>12-14</sup>. The long-term effect of RFA on coronary function and the risk involved in initiating arrhythmias is not well known. Considering that 40% of the atrioventricular

accessory pathways are functionally abolished by the end of the first year of life, SVT in the newborn and infants should initially be treated pharmacologically. In choosing the most appropriate antiarrhythmic agent for its treatment, identifying the mechanism of the tachycardia is important. In the Boston series<sup>2</sup>, a total of 393 TEEPS procedures were performed on 270 patients and the diagnoses of AVRT and AVNRT were made in 74% and 21%, respectively, by measuring the VA intervals. In the present study, in all the study groups, tachycardia was induced in 72 out of 153 procedures. The diagnosis based on the VA interval being less than 70 msec or longer than 70 msec was AVNRT in 25 (34.7%) and AVRT in 41 (56.9%), respectively (Fig. 1).

In general, many studies show that, whether symptomatic or not, initial presentation in patients with WPW syndrome may be sudden death<sup>17-20</sup>. The risk varies from 0.1% to 0.6%. The underlying etiology is generally a very high ventricular rate and atrial fibrillation; thus, in the long-term treatment planning, it is important to identify patients with WPW syndrome with a particularly increased risk of developing ventricular fibrillation<sup>19,21</sup>. Since the APERP in children is shorter than in adults, the probability of initially presenting with ventricular fibrillation or sudden death is higher in children with WPW syndrome<sup>17-20,22,23</sup>. The most important indicator of the ventricular fibrillation development during atrial fibrillation is the length of APERP<sup>24-28</sup>. The longer the refractory period of the accessory pathway, the lower the risk of developing VT. The intermittent observation of preexcitation with rest ECG, the disappearance of delta waves during exercise test, especially with low heart rate and after injection of high- dose intravenous procainamide, all imply that APERP is long<sup>29-32</sup>. The best means to assess APERP is the use of electrophysiologic study. In our study as well, of the 17 patients followed for WPW syndrome, in whom TEEPS was performed to assess the risk of sudden death, an exercise test was performed in 8. During the exercise test it was observed that in 1 of the patients, preexcitation disappeared intermittently and reappeared during the recovery phase. APERP was found to be shorter than 250 msec in 5 patients.

In our study, TEEPS was performed to assess the effectiveness of the treatment in a total of 17 patients. Though in 12 of the 17 patients tachycardia was induced, in 4 of them, the tachycardia cycle length was prolonged significantly when compared to that at the initial procedure or clinically documented tachycardia, and in 2 of them tachycardia was non-sustained. Kulakowski et al.<sup>15</sup>, in assessing the effectiveness of medical treatment, performed TEEPS before and during treatment with oral antiarrhythmic drugs on 37 patients with narrow QRS complex tachycardia. After initiation of medical treatment, and during the follow-up, a total of 9 of 12 patients (75%) with inducible and only 1 patient with non-inducible tachycardia by TEEPS had a recurrence of tachycardia attacks. At the end of the study, the negative and positive predictive values of TEEPS in assessing the effectiveness of medical treatment were found to be 96% and 75%, respectively. Santinelli et al.<sup>16</sup> gave i.v. propafenone to 3 and oral propafenone to 7 of the 10 symptomatic patients with WPW syndrome, and atrial fibrillation was induced with TEEPS in all those given i.v. propafenone and in 4 of those given oral propafenone. Nevertheless, they found the RR interval to be longer in all those patients in whom atrial fibrillation was induced compared to the measurements before the treatment, and associated this to the effectiveness of the treatment given. In conclusion, if tachycardia is not inducible during the TEEPS in a patient under treatment or if prolongation in the tachycardia cycle is found when compared to the values before the treatment, then the given treatment can be said to be quite effective. The induction of tachycardia does not automatically imply that the treatment in question is a total failure.

In the present study, we also used TEEPS in a small group of patients with recurrent symptoms after RFA. Tachycardia recurrence after successful RFA is possible in some patients during the follow-up. Non-invasive recording methods may be valuable to document both the presence and absence of the arrhythmia during the symptoms. Due to disadvantages of the methods mentioned before, especially in patients with infrequent and short symptoms, TEEPS may be useful in making a precise and immediate diagnosis and in relieving patient and family anxiety in the short-term.

Since non-inducibility is not sufficiently sensitive for excluding an arrhythmic origin, the fact that in 64% of the symptomatic patients in whom TEEPS was performed SVT was not induced indicates that the reasons for the presentation of most patients with these types of complaints is not of cardiac origin. Though it is important to note that, in adolescent patients, such complaints are likely to be psychological, arrhythmic work-up should be done before associating palpitations to psychiatric problems. In light of this study, we think that in those cases where complaints are not strongly suspected to be of cardiac origin, TEEPS, as a semi-invasive procedure, could be performed for the purpose of relieving family anxiety.

Though TEEPS is useful in a wide variety of situations as described above, it has some limitations, which include the sometimes painful nature of the investigation and the impossibility of recording the electrical activity of the His bundle. This may limit detailed evaluation of atrioventricular node and accessory pathways. In addition to these limitations, it is also not generally effective for ventricular pacing by standard stimulator and catheter systems. Therefore, it should be kept in mind that possible ventricular arrhythmia cannot be ruled out by this technique. Despite these limitations, we think that TEEPS is a semi-invasive, effective and rapid method in the initial evaluation and management of patients with a variety of arrhythmias, except those originating from the ventricles, in evaluation of risk assessment in WPW syndrome and of antiarrhythmic therapy effectiveness, and in the follow-up of patients with recurring symptoms after RFA. In patients whose complaints cannot be explained by non-invasive procedures like ECG and Holter monitoring, a semi-invasive procedure like TEEPS should be preferred instead of intracardiac electrophysiologic study.

#### REFERENCES

1. Knick BJ, Saul JP. Immediate arrhythmia management. In: Zeigler VL, Gillette PC (eds). *Practical Management of Pediatric Cardiac Arrhythmias*. New York: Futura Publishing Co. Inc.; 2001: 161-230.
2. Saul JP. Transesophageal atrial recording and pacing. In: Walsh EP, Saul JP, Triedman JK (eds). *Cardiac Arrhythmias in Children and Young Adults with Congenital Heart Disease*. Philadelphia: Lippincott Williams and Wilkins; 2001: 33-55.
3. Hessling G, Brockmeier K, Ulmer HE. Transesophageal electrocardiography and atrial pacing in children. *J Electrocardiol* 2002; 35: 143-149.
4. Pongiglione G, Saul JP, Dunnigan A, Strasburger JF, Benson DW Jr. Role of transesophageal pacing in evaluation of palpitation in children and adolescents. *Am J Cardiol* 1988; 62: 566-570.
5. Kugler JD, Danford DA. Management of infants, children and adolescents with paroxysmal supraventricular tachycardia. *J Pediatr* 1996; 129: 324-338.
6. Benson DW Jr, Dunnigan A, Sterba R, Benditt DG. Atrial pacing from the esophagus in the diagnosis and management of tachycardia and palpitations. *J Pediatr* 1983; 102: 40-46.
7. Ozer S, Schaffer M. Sinus node reentrant tachycardia in a neonate. *Pacing Clin Electrophysiol* 2001; 24: 1038-1040.
8. Ko JK, Ryu SJ, Ban JE, Kim YH, Park IS. Use of transesophageal atrial pacing for documentation of arrhythmias suspected in infants and children. *Jpn Heart J* 2004; 45: 63-72.
9. Kesek M, Sheikh H, Bastani H, Bloomstrom P, Lundqvist CB. The sensitivity of transesophageal pacing for screening in atrial tachycardias. *Int J Cardiol* 2000; 72: 239-242.
10. Pehrson SM, Blomstrom-Lundqvist C, Ljungstrom E, Blomstrom P. Clinical value of transesophageal atrial stimulation and recording in patients with arrhythmia-related symptoms or documented supraventricular tachycardia -- correlation to clinical history and invasive studies. *Clin Cardiol* 1994; 17: 528-534.
11. Kugler JD. Electrophysiologic studies. In: Allen HD, Gutgesell HP, Clark EB, Driscoll DJ (eds). *Moss and Adams Heart Disease in Infants, Children, and Adolescents* (6<sup>th</sup> ed) Vol 1. Philadelphia: Lippincott Williams and Wilkins; 2001: 452-467.
12. Perry JC. Supraventricular tachycardia. In: Garson A Jr, Bricker JT, Fisher DJ, Neish S (eds). *The Science and Practice of Pediatric Cardiology* (2<sup>nd</sup> ed). Baltimore: Williams and Wilkins; 1998: 2059-2101.
13. Ro PS, Rhodes LA. Atrioventricular node reentry tachycardia in pediatric patients. *Prog Pediatr Cardiol* 2001; 13: 3-10.
14. Brembilla-Perrot B, Marcon F, Bosser G, Lucron H. Junctional tachycardia in adolescents: nodal reentry is the most frequent cause. *Ann Cardiol Angeiol (Paris)* 2000; 49: 8-12.
15. Kulakowski P, Dluzniewski M, O'Nunain S, Camm AJ, Wardzynska M. The value of transesophageal atrial pacing in predicting the efficacy of antiarrhythmic drugs in patients with paroxysmal narrow QRS complex tachycardia. *Pacing Clin Electrophysiol* 1992; 15: 895-904.
16. Santinelli V, Turco P, De Paola M, et al. Propafenone in Wolff-Parkinson-White syndrome at risk. *Cardiovasc Drugs Ther* 1990; 4: 681-685.
17. Munger TM, Packer DL, Hammill SC, et al. A population study of the natural history of Wolff-Parkinson-White syndrome in Olmsted County, Minnesota, 1953-1989. *Circulation* 1993; 87: 866-873.

18. Deal BJ, Dick M, Beerman L, et al. and Members of the Pediatric Electrophysiology Society. Cardiac arrest in young patients with Wolff-Parkinson-White syndrome (Abstract). *Pacing Clin Electrophysiol* 1995; 18: 815.
19. Blaufox AD, Saul JP. Accessory-pathway-mediated tachycardias. In: Walsh EP, Saul JP, Triedman JK (eds). *Cardiac Arrhythmias in Children and Young Adults with Congenital Heart Disease*. Philadelphia: Lippincott Williams and Wilkins, 2001: 175-199.
20. Bromberg BI, Lindsay BD, Cain ME, Cox JL. Impact of clinical history and electrophysiologic characterization of accessory pathways on management strategies to reduce sudden death among children with Wolff-Parkinson-White syndrome. *J Am Coll Cardiol* 1996; 27: 690-695.
21. Saul JP, Walsh EP, Triedman JK. Mechanisms and therapy of complex arrhythmias in pediatric patients. *J Cardiovasc Electrophysiol* 1995; 6: 1129-1148.
22. Chang RK, Wetzel GT, Shannon KM, Stevenson WG, Klitzner TS. Age- and anesthesia-related changes in accessory pathway conduction in children with Wolff-Parkinson-White syndrome. *Am J Cardiol* 1995; 76: 1074-1076.
23. 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: 1229-1233.
24. Klein GJ, Bashore TM, Sellers TD, Pritchett EL, Smith WM, Gallagher JJ. Ventricular fibrillation in the Wolff-Parkinson-White syndrome. *N Engl J Med* 1979; 301: 1080-1085.
25. Wellens HJ, Rodriguez LM, Timmermans C, Smeets JP. The asymptomatic patient with the Wolff-Parkinson-White electrocardiogram. *Pacing Clin Electrophysiol* 1997; 20: 2082-2086.
26. Morady F, Sledge C, Shen E, Sung RJ, Gonzales R, Scheinman MM. Electrophysiologic testing in the management of patients with the Wolff-Parkinson-White syndrome and atrial fibrillation. *Am J Cardiol* 1983; 51: 1623-1628.
27. Milstein S, Sharma AD, Klein GJ. Electrophysiologic profile of asymptomatic Wolff-Parkinson-White pattern. *Am J Cardiol* 1986; 57: 1097-1100.
28. Montoya PT, Brugada P, Smeets J, et al. Ventricular fibrillation in the Wolff-Parkinson-White syndrome. *Eur Heart J* 1991; 12: 144-150.
29. Klein GJ, Gulamhusein SS. Intermittent preexcitation in the Wolff-Parkinson-White syndrome. *Am J Cardiol* 1983; 52: 292-296.
30. Wellens HJ, Braat S, Brugada P, Gorgeis AP, Bar FW. Use of procainamide in patients with the Wolff-Parkinson-White syndrome to disclose a short refractory period of the accessory pathway. *Am J Cardiol* 1982; 50: 1087-1089.
31. Strasberg B, Ashley WW, Wyndham CR, et al. Treadmill exercise testing in the Wolff-Parkinson-White syndrome. *Am J Cardiol* 1980; 45: 742-748.
32. Bricker JT, Porter CJ, Garson A Jr, et al. Exercise testing in children with Wolff-Parkinson-White syndrome. *Am J Cardiol* 1985; 55: 1001-1004.