JACC Vol. 5, No. 1
January 1985:130–5

Wolff-Parkinson-White Syndrome and Supraventricular Tachycardia During Infancy: Management and Follow-Up

BARBARA J. DEAL, MD, JOHN F. KEANE, MD, PAUL C. GILLETTE, MD, FACC, ARTHUR GARSON, JR., MD, FACC

Boston, Massachusetts and Houston, Texas

The records of 90 patients with Wolff-Parkinson-White syndrome who presented with supraventricular tachycardia in the first 4 months of life were reviewed. Among these, 63% were male. Structural heart disease was present in 20%, most commonly Ebstein's anomaly. All patients presented with a regular narrow QRS tachycardia, and pre-excitation became evident only when normal sinus rhythm was established. Only one infant had atrial flutter and none had atrial fibrillation. Type A Wolff-Parkinson-White syndrome was most common (49%), with heart disease occurring in only 5% of these patients. In contrast, heart disease was identified in 45% of those with type B syndrome.

Initially, normal sinus rhythm was achieved in 88% of the 66 infants treated with digoxin with no deaths. Normal sinus rhythm resumed after electrical countershock in 87% of the 15 infants so treated. Maintenance digoxin therapy was used in 85 patients. The Wolff-Parkinson-White pattern disappeared in 36% of the patients. Four infants died of cardiac causes during the mean follow-up period of 6.5 years. Two of these four infants had congenital heart disease; the third, with a normal heart initially, developed ventricular fibrillation and died from a cardiomyopathy considered related to resuscitation. The remaining infant, with a normal heart, died suddenly at 1 month of age. All were receiving digoxin. A wide QRS tachycardia later appeared in three patients, all with heart disease, one of whom died.

In the initial treatment of narrow regular QRS supraventricular tachycardia in this group of patients, digoxin was a safe and effective (88%) drug for restoration of normal sinus rhythm, with electrical cardioversion being equally effective (87%) in those critically ill. After age 1 year, 33% of the infants experienced recurrent tachycardias; these were more frequent in patients with type B Wolff-Parkinson-White syndrome ($p < 0.05$) and in those requiring more than one drug to maintain normal sinus rhythm during the initial hospitalization ($p < 0.001$).

(J Am Coll Cardiol 1985;5:130–5)

The Wolff-Parkinson-White syndrome is present in 10 to 50% of infants with supraventricular tachycardia (1–5). Digoxin has been the standard recommended therapy for these infants (2,6), in contrast to adult patients (7–9). Among patients presenting with this syndrome in early infancy, recurrences after the first 18 months of life are thought unlikely (2,5,6,10), and previous investigators (6) have stressed the benign course of the syndrome in patients without structural heart disease. Only limited information is available concerning the long-term outcome in such infants, including the persistence of the electrocardiographic pattern, the incidence of recurrent supraventricular tachycardia and the results of therapy (3,4,6,11,12).

Because controversy exists about the use of digoxin, we reviewed the data on all infants presenting with supraventricular tachycardia and Wolff-Parkinson-White conduction to our institutions over the last 31 years. In addition, we attempted to evaluate prognostic factors and the outcome of various treatment regimens, particularly digitalis therapy.

Methods

Patients. The records of all patients with the diagnosis of Wolff-Parkinson-White syndrome observed at the Texas Children's Hospital, Houston and the Children's Hospital, Boston between 1951 and 1982 were reviewed. Among these, 90 infants were identified who had a prolonged episode of tachycardia in the first 4 months of life and are the subject of this report. The electrocardiographic diagnosis of Wolff-Parkinson-White syndrome in normal sinus rhythm

From the Department of Cardiology, Children's Hospital, Harvard Medical School, Boston, Massachusetts and the Lilly Frank Abercombie Section of Cardiology, Department of Pediatrics, Texas Children's Hospital, Baylor College of Medicine, Houston, Texas. This study was supported in part by Training Grant 07193-05 from the National Institutes of Health, Bethesda, Maryland. Manuscript received February 21, 1984; revised manuscript received July 24, 1984, accepted August 10, 1984.

Address for reprints: John F. Keane, MD, Department of Cardiology, Children's Hospital, 300 Longwood Avenue, Boston, Massachusetts 02115.

0735-1097/85/$3.30

was based on a short PR interval for age (≤0.08 second), a prolonged QRS complex (>0.08 second) and a delta wave. The electrocardiograms were classified into type A (positive delta wave and positive QRS complex in right precordial leads, simulating right bundle branch block), type B (positive delta wave and positive QRS complex in left precordial leads, resembling left bundle branch block) and type C (positive delta wave, positive QRS complex in leads V_1 to V_6) (12,13).

Among the 90 infants, 48 were seen at Texas Children's Hospital and the other 42 at Children's Hospital in Boston. A complete history, physical examination, chest roentgenogram and electrocardiogram were obtained for each patient; more thorough investigations were made in those patients with evidence of underlying heart disease. Complete 12 lead electrocardiograms taken during the initial episode of tachycardia were available for analysis in 65 patients; rhythm strips were available in the remaining 25. All patients had complete electrocardiograms after conversion to normal sinus rhythm.

Follow-up. This included a detailed history of the frequency of tachycardia episodes, a record of all medications taken, electrocardiograms (obtained in 89 patients) and further tests as indicated. Contact by telephone or letter was attempted for any patient not seen within the last 3 years.

Statistics. Statistical analysis of continuous data was performed using the Student's *t* test; categorical data were evaluated using chi-square analysis.

Results

Clinical features. Of the 90 infants, 57 (63%) were male and 33 were female ($p < 0.05$). No structural heart disease was present in 72 patients (80%), while heart disease was identified in 18 (20%) (Table 1). Among the latter, 10 were female and Ebstein's anomaly was the most common lesion encountered (5 patients).

Pattern of presentation. All patients presented initially with tachycardia, and Wolff-Parkinson-White conduction was first noted only after conversion to normal sinus rhythm. The mean age at presentation was 29 days, with 69% presenting in the first month of life. Tachycardia was detected in utero in seven patients (8%), two of whom were delivered by emergency cesarean section for congestive heart failure. Signs of congestive heart failure were the presenting features in 54% of the patients (49 of 90); most asymptomatic infants had the arrhythmia identified during a routine evaluation. Three patients presented in shock that in two of the three progressed to cardiac arrest from which they were successfully resuscitated. In addition, two infants underwent an exploratory laparotomy for unexplained neonatal ascites before the tachycardia and then the Wolff-Parkinson-White pattern became manifest on electrocardiogram.

Electrocardiogram. During the initial episode of tachycardia, the heart rate was regular in all infants with rates ranging from 215 to 350 beats/min (mean 280); the QRS complex was narrow in all. There was no correlation between heart rate during tachycardia and the presence of heart disease. No arrhythmia other than supraventricular tachycardia was identified with the exception of one occurrence of atrial flutter. Subsequently, three patients developed a wide QRS tachycardia (QRS interval >0.12 second).

Classification of Wolff-Parkinson-White pattern. A summary of the types of Wolff-Parkinson-White pattern on the electrocardiogram for all patients and for those with heart disease is presented in Table 2. It can be seen that although type A was most common (49%), only 11% of patients with heart disease had this pattern. In contrast, type B (present in 32% of all patients and in 22% of those with a normal heart) was by far the most common pattern seen in those with heart disease (72%). Among all patients with type B Wolff-Parkinson-White conduction, 45% were found to have heart disease, compared with 5% of patients with type A ($p < 0.001$) and 13% of patients with type C ($p < 0.05$).

Initial treatment. Table 3 outlines the methods used to obtain normal sinus rhythm during the initial episode of supraventricular tachycardia. Spontaneous conversion to sinus rhythm occurred in eight patients, whereas normal sinus rhythm was achieved in 58 (88%) of 66 infants treated with digoxin and 13 (87%) of 15 patients treated with electrical countershock. After conversion to normal sinus rhythm, five

Table 1. Cardiac Lesions in Infants With Wolff-Parkinson-White Syndrome and Supraventricular Tachycardia

Lesion	No. of Patients
Ebstein's anomaly	5
Patent ductus arteriosus	3
Cardiomyopathy	3
Corrected transposition of great arteries	2
Mitral insufficiency + VSD	1
Single ventricle + PS	1
Aortic stenosis	1
VSD	1
PAPVC	1

PAPVC = partial anomalous pulmonary venous connection; PS = pulmonary stenosis; VSD = ventricular septal defect.

Table 2. Classification of Wolff-Parkinson-White Patterns

Type	All Patients (n = 90) No. (%)	Patients With Normal Heart (n = 72) No. (%)	Patients With Heart Disease (n = 18) No. (%)
A	44(49)	42(58)	2(11)
B	29(32)	16(22)	13(72)
C	16(18)	14(20)	2(11)
A + B	1(1)	0	1(6)

JACC Vol. 5, No. 1
January 1985:130–5

Table 3. Methods of Conversion to Normal Sinus Rhythm During Initial Supraventricular Tachycardia

	No. of Patients (%)
Spontaneous conversion	8(9)
Digoxin administration	58(65)
Electrical cardioversion	13(14)
Other*	11(12)
Total	90(100)

*Includes administration of multiple drugs (six patients), vagal maneuvers (one patient), ice to forehead (one patient), verapamil (one patient), methoxamine (one patient) and phenylephrine (one patient).

of the eight patients whose arrhythmia converted spontaneously were discharged taking no medication; all of the remaining 85 infants received digoxin. Among these infants, 15 required the addition of one or more drugs to maintain sinus rhythm during their initial hospitalization. No deaths occurred during treatment of this initial episode of supraventricular tachycardia.

Follow-Up

The 90 patients were followed up from 0.5 months to 27 years (mean 6.5 years) (Fig. 1). The Wolff-Parkinson-White pattern persisted in 64% (57 of 89) of the patients, and was not seen again in the other 36%.

Mortality. There were five deaths during this period (Table 4); all five patients were receiving digoxin. The first patient presented with tachycardia at birth and was maintained on digoxin therapy after successful conversion to normal sinus rhythm with this drug. This infant with type A conduction was found dead in his crib at 1 month of age. Postmortem examination was unrevealing. The second patient had severe cyanotic congenital heart disease, type B Wolff-Parkinson-White conduction and had undergone a Potts shunt 10 days before dying during an episode of tachycardia on the way to the hospital. The patient was receiving digoxin and quinidine.

Figure 1. Duration of follow-up (in years) of patients after first documented episode of supraventricular tachycardia. Median = 4.5 years.

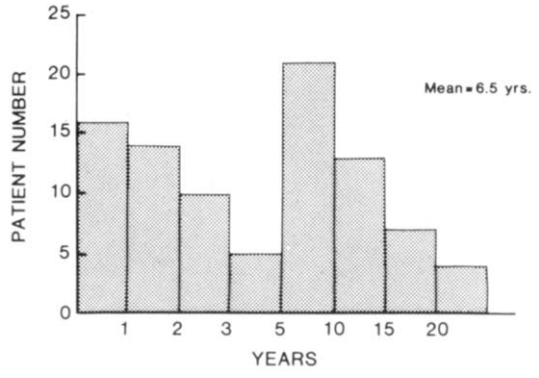

Table 4. Mortality in the Patients Studied

Age at Death	Circumstance	Cardiac Lesions
1 mo	Dead in crib	None
4 mo	During tachycardia	Single left ventricle (d-loop) and PS
6 mo	SVT, VF	Cardiomyopathy
18 yr	SVT, VF	Ebstein's anomaly
15 yr	Accidental	None

PS = pulmonary stenosis; SVT = supraventricular tachycardia; VF = ventricular fibrillation.

In the third patient, with type B conduction, supraventricular tachycardia at birth was converted by treatment with digoxin, which was continued as maintenance therapy. On the basis of physical examination and chest roentgenogram, there was no evidence of heart disease at this time. At 2 months of age, she had a cardiac arrest outside the hospital and received several electrical countershocks up to 75 watt-s/dose for recurrent ventricular fibrillation for the next 2 days. She died at 6 months of age and was found to have a cardiomyopathy thought to be related to the high electrical cardioversion doses.

The fourth patient, with Ebstein's anomaly and type B Wolff-Parkinson-White conduction, developed frequent symptomatic supraventricular tachycardia with a wide QRS complex; this represented the antidromic form of accessory pathway conduction. He was treated with digoxin and quinidine and had multiple admissions for electrical cardioversion. During one of these admissions, supraventricular tachycardia degenerated to ventricular fibrillation and the patient died at 18 years of age. Neither digoxin nor quinidine serum levels were available in these four patients; although they were receiving standard doses, it is not possible to exclude drug toxicity as a contributing factor in their deaths. The remaining patient died because of an accident.

Wide QRS tachycardia. Three patients, each with heart disease, developed a wide QRS tachycardia during subsequent episodes of supraventricular tachycardia; the first is the fourth patient just described. A second patient, also with Ebstein's anomaly, had brief recurrent episodes of narrow complex supraventricular tachycardia in the first year of life and was maintained on digoxin therapy for 8 years. At 9 years of age, without digoxin treatment, she developed a wide QRS tachycardia (antidromic conduction) with chest pain, requiring electrical cardioversion. Digoxin therapy was resumed but she continued to have frequent symptomatic episodes of wide QRS tachycardia, later accompanied by atrial fibrillation at age 24 years. Digoxin was discontinued, and after failure of quinidine and propranolol therapy, she is now well controlled with amiodarone treatment alone.

The third patient was born with atrial flutter, the only patient with this arrhythmia in this series. Later, while receiving digoxin, she developed a wide QRS tachycardia along with a variety of atrial arrhythmias including atrial

fibrillation-flutter. This patient was successfully treated with and maintained on procainamide after failure of cardioversion and propranolol therapy.

Recurrent supraventricular tachycardia. Among the 90 infants, 37 (41%) experienced supraventricular tachycardia as a single episode or as brief recurring episodes only during their initial hospitalization. Another 30 patients (33%) continued to have recurrent supraventricular tachycardia after 1 year of age. All 30 patients also continued to have recurrences beyond 18 months. Twenty-five (42%) of 60 patients followed up for more than 2 years continued to have recurrent episodes of tachycardia. Recurrence of tachycardia after 1 year of age was not related to sex, presence of heart disease (with the exception of Ebstein's anomaly) or persistence of Wolff-Parkinson-White pattern on the electrocardiogram (chi-square analysis). Recurrences were significantly more likely to occur in patients with type B Wolff-Parkinson-White conduction than in those with type A ($p < 0.05$) or type C ($p < 0.05$) and in those requiring two or more drugs to maintain normal sinus rhythm during presentation ($p < 0.001$). Among patients with type B conduction, 45% had recurrent supraventricular tachycardia after 1 year of age, compared with 25% of patients with type A and 20% with type C conduction. Three-fourths of patients requiring multiple drugs initially continued to have late recurrences, as did four of the five with Ebstein's anomaly.

Maintenance therapy. Two patients had no further episodes of supraventricular tachycardia after spontaneous conversion to normal sinus rhythm and received no medication. Digoxin was the only medication used in 55 patients (61%); 33 patients (37%) required additional therapy to control recurrent supraventricular tachycardia. Among the 15 patients receiving multiple drugs to maintain constant normal sinus rhythm at presentation, 11 continued to have recurrent supraventricular tachycardia beyond 1 year of age; 6 required additional medical therapy and 2 eventually underwent surgical division of the bypass tract. In contrast, only 15 of the 70 infants managed initially with digoxin alone required additional medical therapy.

In patients experiencing frequent recurrent supraventricular tachycardia while receiving digoxin, various combinations of digoxin, quinidine and propranolol were used to maintain normal sinus rhythm. In addition, one patient with Ebstein's anomaly with recurrent symptomatic episodes of wide QRS supraventricular tachycardia while taking these medications is now well controlled with amiodarone alone. Verapamil was successful in one of three patients in whom it was tried after failure of digoxin, quinidine and propranolol therapy.

Three patients underwent surgery for division of Kent bundles after failure of medical therapy. Postoperatively, two patients have had no recurrences of supraventricular tachycardia without medication, with disappearance of the Wolff-Parkinson-White pattern on the electrocardiogram. The third patient has had a decrease in episodes of tachycardia, but continues to have the pre-excitation pattern.

Electrophysiologic studies. Twenty patients underwent 22 studies at Texas Children's Hospital; most have been previously reported (14). The anterograde effective refractory periods of the accessory connection and the atrioventricular node were measured in 14 and 5 patients, respectively; in eight more recent studies, the response of the refractory period to ouabain administration was determined as a guide to future therapy with digoxin. The anterograde effective refractory period of the accessory connection was longer than 200 ms in all but one (170 ms) and exceeded that of the atrioventricular node in three patients in whom both measurements were available. After ouabain administration, the anterograde effective refractory period of the accessory connection decreased in four (<200 ms in one), increased in three and was unchanged in one patient. One patient with Ebstein's anomaly developed rapid anterograde conduction over the accessory pathway with atrial fibrillation during the study after receiving ouabain; her therapy was therefore changed from digoxin to quinidine.

No accessory pathway could be identified in two patients in whom the electrocardiographic pattern of Wolff-Parkinson-White conduction had disappeared; in one of these, an accessory pathway had been documented previously.

Discussion

Analysis of this series of 90 infants with Wolff-Parkinson-White conduction and supraventricular tachycardia confirms the previously reported male preponderance (3,6,10) and the incidence of congenital heart disease in approximately 20% of patients (6,10,11). Although classification of Wolff-Parkinson-White conduction into three types may be of some use in localizing the bypass tracts (7,15,16), it has not been thought to be clinically helpful in patient management (12,17). In our patients with Wolff-Parkinson-White syndrome presenting with supraventricular tachycardia in early infancy, type A conduction was most common (49%), was associated with heart disease in only 5% of patients and had a relatively low risk of recurrent supraventricular tachycardia (25%) after 1 year of age. Type B conduction occurred in about one-third of our infants and was associated with a high incidence of both heart disease (45%) and recurrent supraventricular tachycardia (45%). Overall, the Wolff-Parkinson-White pattern disappeared on subsequent electrocardiograms in 36% of the patients; however, recurrent supraventricular tachycardia was not significantly less in these children, perhaps because of the small number of patients in this group or because retrograde conduction only over the accessory pathway persisted. In one patient, the accessory pathway could not be detected at a subsequent electrophysiologic study. Therefore, at least in

the infants and children, classification of Wolff-Parkinson-White conduction into types may provide useful diagnostic and prognostic information.

Treatment of Supraventricular Tachycardia: Use of Digitalis and Recommendations

It is well documented that in adults, atrial fibrillation may be associated with rapid anterograde conduction over the accessory pathway, leading to ventricular fibrillation (7,9,17–19). It has also been shown (9,18,20) that digitalis may shorten the anterograde refractory period of the accessory pathway and result in ventricular fibrillation in both adults and children.

Initial episode of supraventricular tachycardia. All of our patients presented initially with tachycardia, and Wolff-Parkinson-White conduction became evident only after conversion to normal sinus rhythm. No patient was found to have either atrial fibrillation or a wide QRS tachycardia at onset, although a single neonate with these features was recently reported (21). Digoxin, administered to 66 infants in stable condition, was successful in restoring normal sinus rhythm in 58 (88%) and no deaths occurred. Therefore, in the initial management of infants in stable condition with a regular narrow QRS supraventricular tachycardia, digoxin and electrical cardioversion (0.5 watt-s/kg) were both safe and equally effective in this selective series of patients. Direct current countershock remains the treatment of choice for urgent conversion of life-threatening tachycardia. In the future, the technique of esophageal pacing (22,23) may become more widely available for use in the infant and provide an alternative to electrical cardioversion.

The choice of medication for maintenance therapy in these infants presents a more difficult problem. Mortality associated with the use of digoxin in this patient group may be at least 2%, and could be as high as 5%. In addition, excessive amounts of digoxin may precipitate ventricular fibrillation in these infants (24). Therefore, on the basis of this experience, the use of digoxin in these patients may carry a small but significant risk; the results of a multicenter investigation now in progress may help clarify this issue. Thus, in the absence of electrophysiologically demonstrated safety of digoxin, the use of propranolol should be considered, particularly in the setting of recurrent supraventricular tachycardia.

Recurrent supraventricular tachycardia. Among those patients with significant recurrent episodes of typical supraventricular tachycardia, other drugs such as propranolol, quinidine or procainamide alone or in combination may be necessary. Verapamil, effective in older patients, may be used in older children, but experience with its use in infants is limited. Bradycardia and hypotension have been reported with use of verapamil in this age group (25); thus, if this agent is used, pacing capabilities should be readily available.

In patients who manifest a wide QRS complex tachycardia or atrial fibrillation, or both, the use of digitalis is contraindicated. Wide QRS tachycardia appeared in three of our patients, two of whom had Ebstein's anomaly. Atrial fibrillation occurred in only two patients in this series, in one, a patient with Ebstein's anomaly, at age 24 years. Drugs other than digitalis should be used in these patients and electrophysiologic studies should be strongly considered.

We recommend electrophysiologic studies in addition for those children older than 1 year of age in whom supraventricular tachycardia is not adequately controlled by medical management, to locate the bypass tract, to measure its refractory period as well as that of the atrioventricular node and to determine the response to medications, including ouabain. Surgical division of the accessory connection is a very effective method of treatment (26,27) and should be considered as an alternative to life-long drug therapy in older children.

On the basis of our experience, overall mortality, related in part to arrhythmias, in patients with Wolff-Parkinson-White syndrome and supraventricular tachycardia is 5% and may be associated with heart disease in some cases. Approximately one-third of the patients will continue to have recurrent supraventricular tachycardia after 1 year of age; all patients presenting in infancy deserve long-term follow-up.

We extend our thanks to Jane Newburger, MD, for statistical analysis and to Barbara Cesaro and Mary Durkin for preparation of the manuscript.

References

1. Nadas AS, Daeschner CW, Roth A, Blumenthal SL. Paroxysmal tachycardia in infants and children: study of 41 cases. Pediatrics 1952;9:167–81.
2. Schiebler GL, Adams P, Anderson P, Anderson RC. The Wolff-Parkinson-White syndrome in infants and children. A review and a report of 28 cases. Pediatrics 1959;24:585–603.
3. Lundberg A. Paroxysmal tachycardia in infancy: follow-up study of 47 subjects ranging in age from 10–26 years. Pediatrics 1973;51:26–35.
4. Wolff GS, Han J, Curran J. Wolff-Parkinson-White syndrome in the neonate. Am J Cardiol 1978;41:559–63.
5. Garson A Jr, Gillette PC, McNamara DG. Supraventricular tachycardia in children: clinical features, response to treatment and long term follow up in 217 patients. J Pediatr 1981;98:875–82.
6. Giardina CV, Ehlers KH, Engle MA. Wolff-Parkinson-White syndrome in infants and children: a long-term follow up study. Br Heart J 1972;34:839–46.
7. Gallagher JJ, Gilbert M, Svenson RH, Sealy WC, Kasell J, Wallace AG. Wolff-Parkinson-White syndrome: the problem, evaluation, and surgical correction. Circulation 1975;51:767–85.
8. Smith WM, Gallagher JJ, Kerr CR, et al. The electrophysiologic basis and management of symptomatic recurrent tachycardia in patients with Ebstein's anomaly of the tricuspid valve. Am J Cardiol 1982;49:1223–34.
9. Wellens HJJ, Durrer D. Effect of digitalis on atrioventricular conduction and circus-movement tachycardias in patients with Wolff-Parkinson-White syndrome. Circulation 1973;47:1229–33.

10. Swiderski J, Lees MH, Nadas AS. The Wolff-Parkinson-White syndrome in infancy and childhood. N Engl J Med 1962;267:968–74.

11. Mantakas ME, McCue CM, Miller WW. Natural history of Wolff-Parkinson-White syndrome discovered in infancy. Am J Cardiol 1978;41:1097–103.

12. Gillette PC, Garson A Jr. The preexcitation syndromes. In: Gillette PC, Garson A Jr, eds. Pediatric Cardiac Dysrhythmias. New York: Grune & Stratton, 1981:155.

13. Rosenbaum FF, Hecht HH, Wilson FN, Johnston FD. The potential variations of the thorax and the esophagus in anomalous atrioventricular excitation (Wolff-Parkinson-White syndrome). Am Heart J 1945;29:281–326.

14. Gillette PC, Garson A Jr, Kugler JD. Wolff-Parkinson-White syndrome in children: electrophysiologic and pharmacologic characteristics. Circulation 1979;60:1487–95.

15. Tonkin AM, Wagner GS, Gallagher JJ, Cope GD, Kasell J, Wallace AG. Initial forces of ventricular depolarization in the Wolff-Parkinson-White syndrome. Circulation 1975;52:1030–5.

16. Becker AE, Anderson RH, Durrer D, Wellens HJJ. The anatomical substrates of Wolff-Parkinson-White syndrome: a clinicopathologic correlation in seven patients. Circulation 1978;57:870–9.

17. Mehta AV, Casta A, Wolff GS. Supraventricular tachycardia. In: Roberts NK, Gelband H, eds. Cardiac Arrhythmias in the Neonate, Infant and Child. Norwalk: Appleton-Century-Crofts, 1983:126.

18. Klein GJ, Bashore TM, Sellers TD, Pritchett ELC, Smith WM, Gallagher JJ. Ventricular fibrillation in the Wolff-Parkinson-White syndrome. N Engl J Med 1979;301:1080–5.

19. Cosio FG, Benson DW Jr, Anderson RW, et al. Onset of atrial fibrillation during antidromic tachycardia associated with sudden cardiac arrest and ventricular fibrillation in a patient with Wolff-Parkinson-White syndrome. Am J Cardiol 1982;50:353–9.

20. Jedeiken R, Gillette PC, Garson A Jr, et al. Effect of ouabain on the anterograde effective refractory period of accessory atrioventricular connections in children. J Am Coll Cardiol 1983;1:869–72.

21. Belhassen B, Pauzner D, Blieden L, et al. Intrauterine and postnatal atrial fibrillation in the Wolff-Parkinson-White syndrome. Circulation 1982;66:1124–8.

22. Gallagher JJ, Smith WM, Kerr CR, et al. Esophageal pacing: a diagnostic and therapeutic tool. Circulation 1982;65:336–41.

23. Benson DW, Dunnigan A, Benditt DG, Pritzker MR, Thompson TR. Transesophageal study of infant supraventricular tachycardia: electrophysiologic characteristics. Am J Cardiol 1983;52:1002–6.

24. Byrum CH, Wahl RA, Behrendt DM, Dick M. Ventricular fibrillation associated with use of digitalis in a newborn infant with Wolff-Parkinson-White syndrome. J Pediatr 1982;101:400–3.

25. Soler-Soler J, Sagrista-Sauleda J, Cabrera A, et al. Effect of verapamil in infants with paroxysmal supraventricular tachycardia. Circulation 1979;59:876–9.

26. Sealy WC, Gallagher JJ, Wallace AG. The surgical treatment of Wolff-Parkinson-White syndrome: evolution of improved methods for identification and interruption of the Kent bundle. Ann Thorac Surg 1976;22:443–57.

27. Gillette PC, Garson A Jr, Kugler JD, Cooley DA, Zinner A, McNamara DG. Surgical treatment of supraventricular tachycardia in infants and children. Am J Cardiol 1980;46:281–4.