The Journal of Emergency Medicine, Vol. 24, No. 3, pp. 295–301, 2003

0736-4679/03 $–see front matter

ELSEVIER

doi:10.1016/S0736-4679(02)00764-3

Selected Topics: Cardiology Commentary

ELECTROCARDIOGRAPHIC MANIFESTATIONS: WIDE COMPLEX TACHYCARDIA DUE TO ACCESSORY PATHWAY

James A. Nelson, MD,* Kirk U. Knowlton, MD,† Richard Harrigan, MD,‡ Marc L. Pollack, MD, PhD,§ and Theodore C. Chan, MD*

*Department of Emergency Medicine and †Division of Cardiology, Department of Internal Medicine, University of California, San Diego Medical Center, San Diego, California, ‡Department of Emergency Medicine, Temple University, Philadelphia, Pennsylvania, and §Department of Emergency Medicine, York Hospital, York, Pennsylvania

Reprint Address: Theodore C. Chan, MD, Department of Emergency Medicine, UC San Diego Medical Center, 200 West Arbor Drive #8676, San Diego, CA 92103

□ **Abstract—Tachycardia with a wide QRS complex is usually due to ventricular tachycardia (VT), supraventricular tachycardia (SVT) with aberrant intraventricular conduction, or an accessory pathway-mediated dysrhythmia. The most common type of accessory pathway causing a wide complex tachycardia is the atrioventricular bypass tract. Distinguishing the accessory pathway-mediated tachycardia from VT or SVT with aberrancy is often difficult, but has important clinical consequences. This article will review the diagnosis of wide complex tachycardia due to an accessory pathway and its related management in the emergent setting.**

□ **Keywords—accessory pathway; electrocardiogram; wide complex tachycardia; EKG manifestations**

INTRODUCTION

Tachycardia with a wide QRS complex implies that one ventricle is being activated before another, usually due to ventricular tachycardia (VT), supraventricular tachycardia (SVT) with aberrant intraventricular conduction, or an accessory pathway-mediated dysrhythmia. The latter can be difficult to distinguish from the two former. In one study of 150 patients presenting with wide complex tachycardia, only one of the seven with an accessory pathway was correctly identified initially. Three were misdiagnosed as having ventricular tachycardia, and three were misdiagnosed as having aberrant conduction (1). The present review addresses the difficult process of recognizing the accessory pathway in wide complex tachycardia.

CASE PRESENTATIONS

Case 1

A 44-year-old man with a history of Wolff-Parkinson-White syndrome presented to the Emergency Department (ED) with a racing heart rate and dizziness. His symptoms had started suddenly 90 min earlier without provocation. The patient had experienced this 3 years prior, successfully treated with adenosine, and then again 15 months ago, resolving spontaneously. The blood pressure was 170/76 mm Hg and the heart rate 280 beats/min. An electrocardiogram (EKG) showed a wide irregular tachycardia (Figure 1). The patient was given adenosine 6 mg, followed by two boluses of 12 mg each, with no response. Lidocaine 100 mg i.v. was then given, and the systolic blood pressure dropped to 96 mm Hg. Serial

Selected Topics: Cardiology Commentary is coordinated by *Theodore Chan*, MD, of the University of California San Diego Medical Center, San Diego, California and *William Brady*, MD, of the University of Virginia, Charlottesville, Virginia

RECEIVED: 19 June 2002; ACCEPTED: 6 August 2002

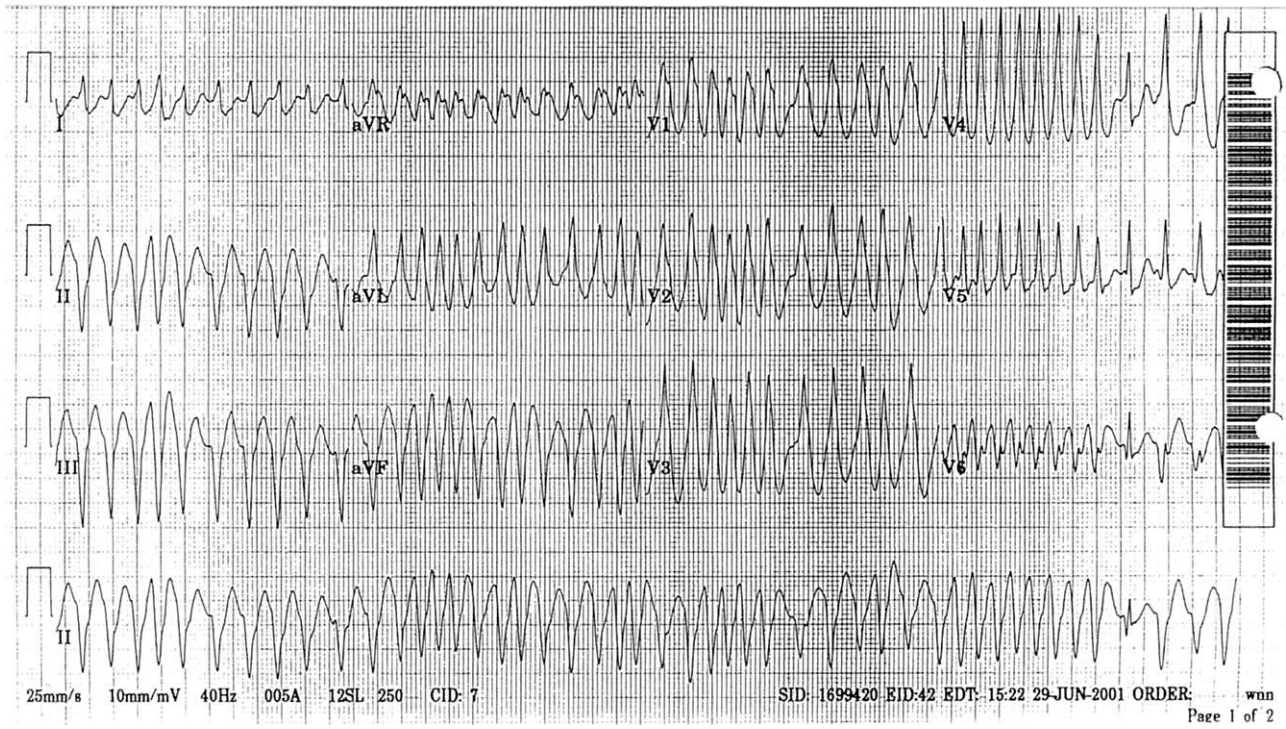

Figure 1. Irregular wide QRS-complex tachycardia seen in a patient with known WPW.

cardioversions were attempted at 50, 75, 100, and then 150 J, which finally cardioverted the rhythm. The patient's rhythm, however, degenerated into ventricular fibrillation. Cardioversion with 360 J returned the patient to sinus rhythm with non-specific ST-T changes and a shortened PR interval (Figure 2).

Case 2

A 20-year-old woman with no past cardiac or other medical history awoke with abdominal pain, palpitations and lightheadedness after drinking alcohol the night before. She presented to an outside clinic at which time a 12-lead EKG was obtained, revealing an irregular wide complex tachycardia (Figure 3). She received adenosine and lidocaine intravenously with no change in her rhythm. Cardioversion was attempted with 100 J, again with no change in her rhythm. She was subsequently transported to the Emergency Department (ED).

On arrival, the patient's vital signs were pulse 200 beats/min and irregular, blood pressure of 112/76 mm Hg, and respiratory rate of 12 breaths/min. In the ED, the patient received amiodarone followed by procainamide. The patient was cardioverted at 200 J into sinus rhythm and the EKG at that time revealed likely pre-excitation (Figure 4). The patient was admitted to the cardiology service and subsequently underwent successful ablation of a left posterior accessory pathway.

DISCUSSION

An accessory pathway is a tract of anomalous conducting tissue in the heart, often causing dysrhythmias. There are many different types of accessory pathways, such as nodoventricular, atriofascicular, and intranodal bypass tracts, but the most common is the atrioventricular (AV) bypass tract (2). This anomalous tract represents an embryologic defect in atrioventricular septation, in which the fibrous skeleton of the heart has not completely sealed atrial from ventricular myocardium (3). The tract, also called the bundle of Kent, is the anatomical substrate of the Wolff-Parkinson-White syndrome, which is characterized by pre-excitation and episodic tachydysrhythmias (4–7).

Pre-excitation is defined as the "premature activation of part of the ventricular myocardium by an impulse that travels by an anomalous path and so avoids physiological delay in the atrioventricular junction" (8). It is manifested by early excitation of the ventricles, resulting in a shortened PR interval and slurred upstroke of the QRS wave. Pre-excitation is seen in about 1.5 per 1000 people (9). Estimates of the prevalence of tachydysrhythmias in this group vary from 12–85% (10).

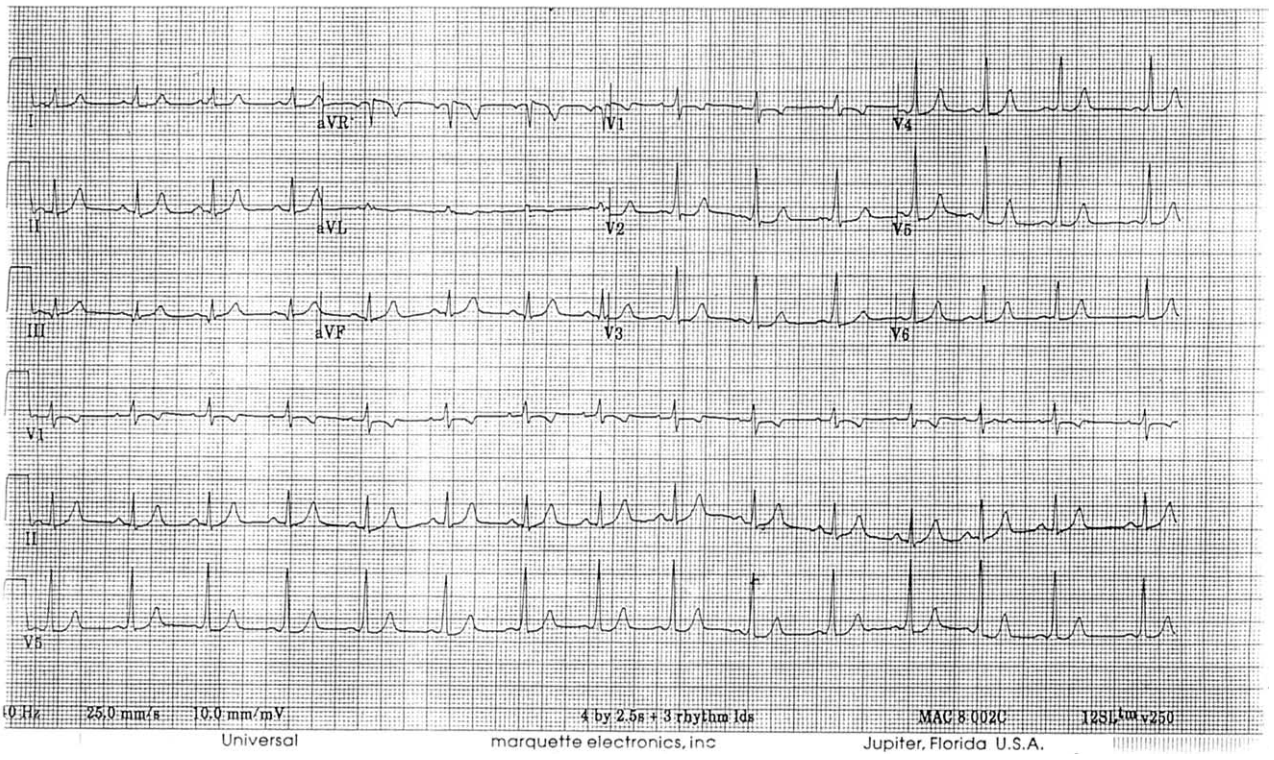

Figure 2. EKG after synchronized electrical cardioversion to sinus rhythm. Note the shortened PR interval and delta wave indicative of WPW.

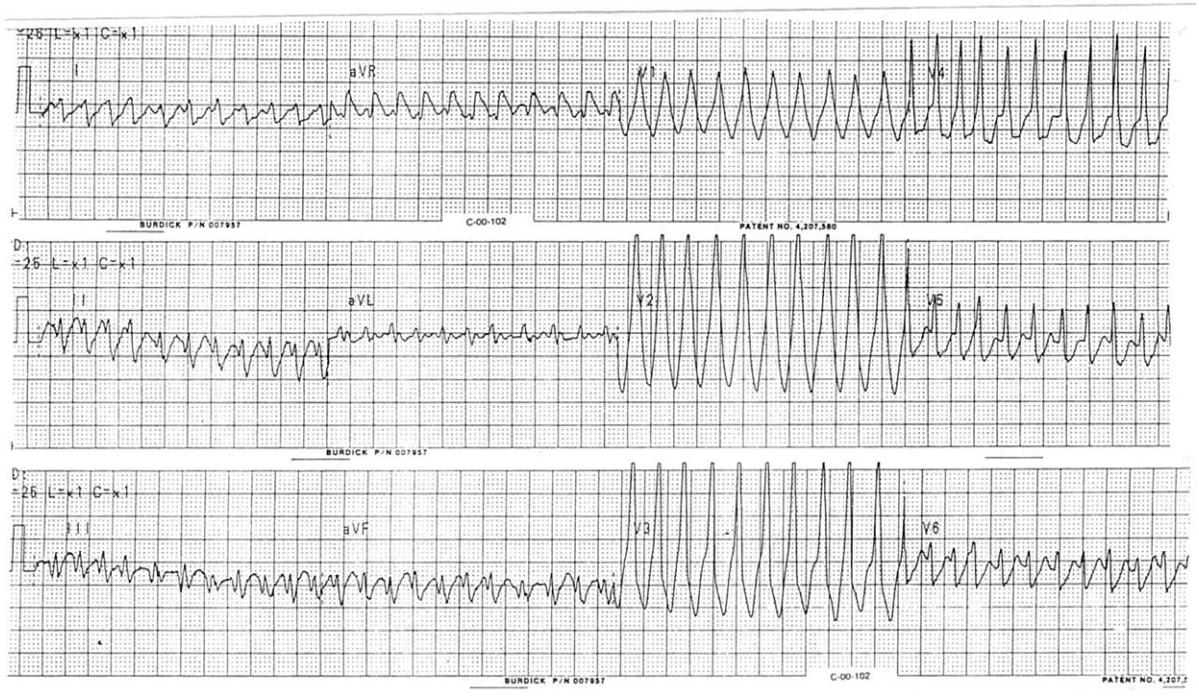

Figure 3. Irregular wide QRS-complex tachycardia seen in 20-year-old woman with no prior cardiac history.

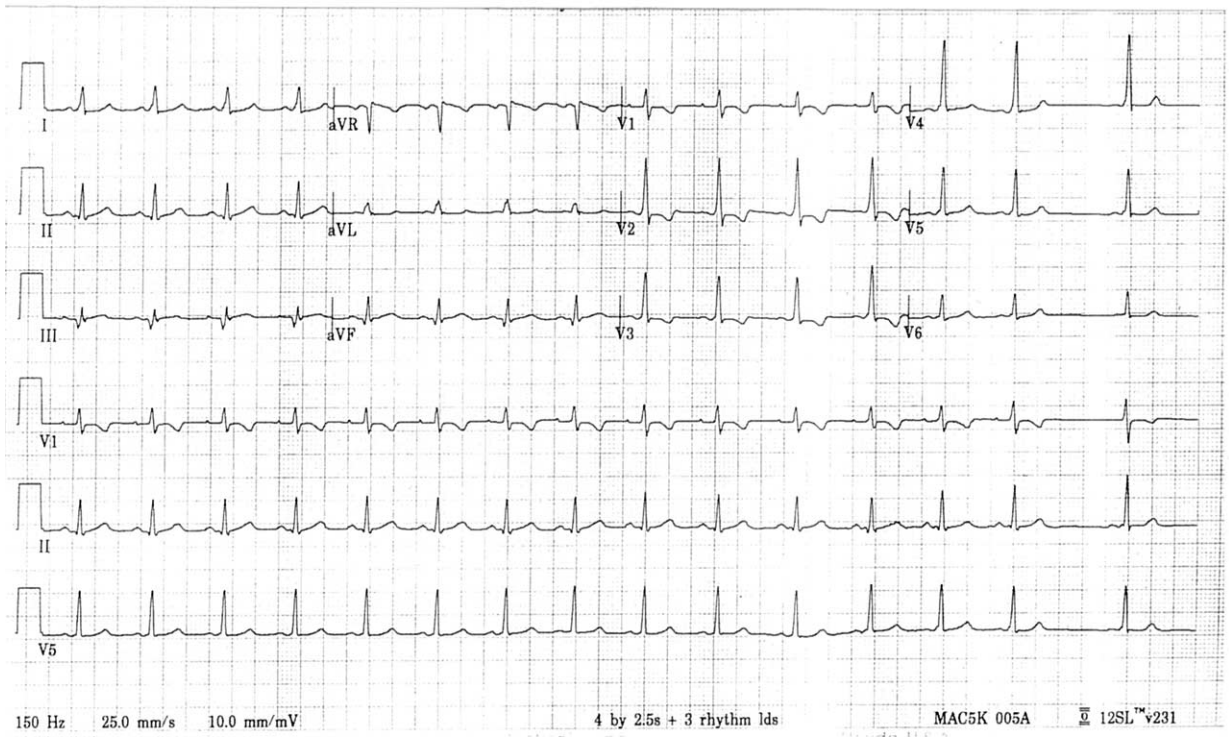

Figure 4. EKG after pharmacologic cardioversion with amiodarone and procainamide to sinus rhythm. The classic findings of shortened PR interval, delta wave, prolonged QRS interval, and ST repolarization abnormalities suggest the diagnosis of pre-excitation.

Most of the tachydysrhythmias caused by the Wolff-Parkinson-White syndrome will be narrow complex, due to atrioventricular reentrant tachycardia (also called orthodromic circus movement tachycardia), with the atrioventricular node used as the antegrade and the accessory pathway as the retrograde limb of a reentry circuit. If the accessory pathway is used as the antegrade limb and the atrioventricular node as the retrograde limb of a reentry circuit, a wide complex tachycardia will be produced, known as antidromic circus movement tachycardia (11). This diagnosis is often difficult.

A more common cause of wide complex tachycardia in patients with Wolff-Parkinson-White syndrome is an atrial tachydysrhythmia such as atrial fibrillation. Atrial fibrillation occurs in 10–39% of patients with Wolff-Parkinson-White syndrome (12,13).

An accessory pathway will conduct an atrial tachydysrhythmia directly to the ventricles (although the AV node continues to be used for conduction as well), creating abnormally high ventricular response rates. These high rates may provoke ventricular fibrillation, a danger that increases in proportion to rate (14,15). Hypotheses to explain this degeneration include myocardial ischemia from poor cardiac output as well as activation of the ventricles during their vulnerable phase (the R-on-T phenomenon) (9). Ventricular fibrillation has been found to be more likely when the RR intervals are less than 205 ms (15).

Failure to recognize an accessory pathway may lead to the misdiagnosis of ventricular tachycardia, often treated with lidocaine. Although early studies suggested that lidocaine would slow conduction through the accessory pathway, subsequent studies have shown lidocaine to either have no effect or to actually increase the ventricular response rate, increasing the risk of ventricular fibrillation (16–18). Lidocaine is no longer recommended as a treatment for wide complex tachycardia due to an accessory pathway (9). It is therefore important to identify the accessory pathway in cases that might resemble ventricular tachycardia.

Misdiagnosis of the tachydysrhythmia as SVT with aberrant intraventricular conduction may lead to the use of AV nodal slowing agents. In the presence of an accessory pathway, these agents will paradoxically increase the heart rate. Digitalis, calcium channel blockers, beta-blockers, and even the short-acting adenosine have this effect, increasing the risk of ventricular fibrillation (19–25). Indeed, AV nodal slowing agents are used in electrophysiology laboratories to artificially enhance conduction through the accessory pathway (26).

Findings Suggesting an Accessory Pathway

History. Patients who are young and free of heart disease are more likely to have processes of congenital origin such as an accessory pathway. They may be able to report a prior diagnosis of Wolff-Parkinson-White syndrome. A history of prolonged paroxysmal palpitations is suggestive.

Rate. Heart rates above 200–220 suggest that the AV node is not controlling the rate, implying either an accessory pathway or ventricular ectopy. The atrioventricular node normally keeps the heart rate below 150 with low sympathetic tone, and below about 200 with high sympathetic tone (27). Heart rates above 200 should lead to a search for an accessory pathway, although some authors have proposed rates as low as 180 or as high as 300 (28,29). A high rate is useful in discriminating aberrant intraventricular conduction from accessory pathway, as aberrancy will usually have a rate below 150.

Rhythm. Irregular rhythm in wide complex tachycardia in most cases will be due to atrial fibrillation, made wide by either aberrant intraventricular conduction or an accessory pathway. The distinction usually can be made on the basis of the EKG, using the discussed differences in rate as well as morphologic criteria (30). Although irregular rhythm in wide complex tachycardia is an important clue for accessory pathway, regular rhythm does not eliminate its possibility. Atrial flutter has a lifetime prevalence of about 5% in those with the Wolff-Parkinson-White syndrome (9).

Morphologic criteria. The usefulness of morphologic criteria has been assessed in a study addressing the discrimination of aberrant intraventricular tachycardia from accessory pathway (31). In patients with irregular wide complex tachycardia, ruling out bundle branch block in leads V1 and V6 was used to assume accessory pathway. This method identified the accessory pathway with 100% sensitivity and a specificity between 79% and 85%. Criteria for right bundle branch block include rSR' in V1 (where R' > r) or RS in V6 (where R > S, small Q waves allowed). Criteria for left bundle branch block include QS in V1 with an intrinsicoid deflection of less than 70 ms (small r is allowed) or a monophasic R in V6 (no Q allowed).

EKG in sinus rhythm. The presence of an accessory pathway can be confirmed by either a baseline or post-cardioversion EKG showing pre-excitation. The criteria for pre-excitation are the following: first, an initial slurring of the upstroke of the QRS, called the delta wave; second, the PR interval will be accordingly shortened, less than 0.12 s. Third, the QRS will be prolonged, at least 0.10 s, although some authors think 0.12 s are necessary for the diagnosis (9,32). For equivocal cases, these findings of pre-excitation during sinus rhythm may be enhanced by adenosine or vagal maneuvers, which tend to slow conduction over the AV node, favoring conduction over the accessory pathway (33).

The classic features are not present on the baseline EKG in 25% of those with accessory pathways, either because the accessory pathway is located close to the AV node or because antegrade conduction does not occur in sinus rhythm (26). The latter is known as a concealed accessory pathway. Diagnosis in these patients is more difficult.

The slow upstroke of the delta wave will, from the opposite leads of the heart, be seen as a slow downstroke. These Q waves may mimic myocardial infarction in as many as 70% of patients (34). Secondary ST-T changes may be present as well, and are generally opposite the delta wave (32).

TREATMENT

If the patient with wide complex tachycardia is unstable, no diagnostic deliberation should delay prompt synchronized cardioversion, 50–100 J (with sedation, if time permits). Immediate cardioversion also should be considered in those with abnormally high heart rates, such as above 200–250 (32,35).

In the stable patient, after the presence of an accessory pathway has been established, procainamide is a reasonable choice for first-line therapy (36,37). Sotalol has been shown, like procainamide, to prolong the refractory period of the accessory pathway, and its use in patients with Wolff-Parkinson-White syndrome and atrial fibrillation has been supported (38–40). Other pharmacologic interventions, such as disopyramide, dofetilide, propafenone, flecainide, amiodarone, and ibutilide have been proposed to be effective, but none has been proved superior to procainamide, nor has any been shown to be effective after the failure of procainamide (10,13,41–45). It is thought that if procainamide fails, further pharmacologic interventions are not likely to be helpful (30). Instead, the patient should be treated immediately with synchronized cardioversion (32).

SUMMARY

Recognizing the presence of an accessory pathway in patients with wide complex tachycardia can be difficult. The entity should be sought in patients with a history of

prolonged intermittent palpitations or a rate greater than 200 beats/min. Diagnosis can be confirmed by evidence of preexcitation on a baseline or post-cardioversion electocardiogram. In patients with irregular wide complex tachycardia, aberrancy often can be distinguished from accessory pathway by its slower rate and its distinct morphologic criteria. Once diagnosed and treated, patients with an accessory pathway should be referred for ablation (46–48).

REFERENCES

1. Akhtar M, Shenasa M, Jazayeri M, Caceres J, Tchou PJ. Wide QRS complex tachycardia. Reappraisal of a common clinical problem. Ann Intern Med 1988;109:905–12.
2. Wellens HJJ. Preexcitation. In: Willerson JT, Cohn JN, eds. Cardiovascular medicine. Philadelphia: Churchill Livingstone; 2000: 1620–31.
3. Dunnigan A. Developmental aspects and natural history of preexcitation syndromes. In: Benditt DG, Benson BW, eds. Cardiac preexcitation syndromes: origins, evaluation, and treatment, 1st edn. Boston: Martinus Nijhoff Publishing; 1986:21–9.
4. Becker AE, Anderson RH, Durrer D, Wellens HJ. The anatomical substrates of Wolff-Parkinson-White syndrome. A clinicopathologic correlation in seven patients. Circulation 1978;57:870–9.
5. Wolferth CC, Wood FC. The mechanism of production of short PR intervals and prolonged QRS complexes in patients with presumably undamaged hearts: hypothesis of an accessory pathway of auriculoventricular conduction (bundle of Kent). Am Heart J 1933; 8:297–311.
6. Holzman M, Scherf D. Uber Elektrokardiogramme met verkurtzer varhof-kammer distanz und positiven P zachen. Z Klin Med 1932; 121:404–23.
7. Wolff L, Parkinson J, White PD. Bundle Branch block with short PR interval in healthy young people prone to paroxysmal tachycardia. Am Heart J 1930;5:685–704.
8. Spraycar M. Stedman's medical dictionary, 26th edn. Baltimore: Williams and Wilkins; 1995.
9. Braunwald E, Zipes D, Libby P. Heart disease, 6th edn. Philadelphia: W.B. Saunders; 2001.
10. Wellens HJJ, Brugada P, Penn OC. The management of preexcitation syndromes. JAMA 1987;257:2325–33.
11. Atie J, Brugada P, Brugada J, et al. Clinical and electrophysiologic characteristics of patients with antidromic circus movement tachycardia in the Wolff-Parkinson-White syndrome. Am J Cardiol 1990;66:1082–91.
12. Campbell RWF, Smith RA, Gallagher JJ, Pritchett ELC, Wallace AG. Atrial fibrillation in the preexcitation syndrome. Am J Cardiol 1977;40:514–20.
13. Al-Khatib SM, Pritchett ELC. Clinical features of Wolff-Parkinson-White syndrome. Am Heart J 1999;138:402–13.
14. Dreifus LS, Haiat R, Watanabe Y, Arriaga J, Reitman N. Ventricular fibrillation: a possible mechanism of sudden death in patients with Wolff-Parkinson-White syndrome. Circulation 1971;43: 520–7.
15. 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.
16. Josephson ME, Kastor JA, Kitchen JG III. Lidocaine in Wolff-Parkinson-White syndrome with atrial fibrillation. Ann Intern Med 1976;84:44–5.
17. Barrett PA, Laks MM, Mandel WJ, Yamaguchi I. The electrophysiologic effects of intravenous lidocaine in the Wolff-Parkinson-White syndrome. Am Heart J 1980;100:23–33.
18. Akhtar M, Gilbert CJ, Shenasa M. Effect of lidocaine on atrioventricular response via the accessory pathway in patients with Wolff-Parkinson-White syndrome. Circulation 1981;63:435–41.
19. Sellers TD Jr, Bashore TM, Gallagher JJ. Digitalis in the preexcitation syndrome. Analysis during atrial fibrillation. Circulation 1977;56:260–7.
20. Gulamhusein S, Ko P, Carruthers SG, Klein GJ. Acceleration of the ventricular response during atrial fibrillation in the Wolff-Parkinson-White syndrome after verapamil. Circulation 1982;65:348–54.
21. Strasberg B, Sagie A, Rechavia E, et al. Deleterious effects of intravenous verapamil in Wolff-Parkinson-White patients and atrial fibrillation. Cardiovasc Drugs Ther 1989;2:801–6.
22. Garatt CJ, Griffith MJ, O'Nunain S, Ward DE, Camm AJ. Effects of intravenous adenosine on antegrade refractoriness of accessory atrioventricular connections. Circulation 1991;84:1962–8.
23. Exner DV, Muzyka T, Gillis AM. Proarrhythmia in patients with the Wolff-Parkinson-White syndrome after standard doses of intravenous adenosine. Ann Intern Med 1995;122:351–2.
24. McGovern B, Garan H, Ruskin JN. Precipitation of cardiac arrest by verapamil in patients with Wolff-Parkinson-White syndrome. Ann Intern Med 1986;104:791–4.
25. Jacob AS, Nielsen DH, Gianelly RE. Fatal ventricular fibrillation following verapamil in Wolff-Parkinson-White syndrome with atrial fibrillation. Ann Emerg Med 1985;14:159–60.
26. Wagner G. Marriott's practical electrocardiography, 9th edn. New York: Lippincott Williams and Wilkins; 1994.
27. Wagner GS, Waugh RA, Ramo BW. Cardiac dysrhythmias. New York: Churchill Livingstone; 1983.
28. Janeira LF. Differentiating wide complex tachycardias. Am Fam Physician 1996;54:1573–84.
29. Tintinalli J. Emergency medicine: a comprehensive study guide. San Francisco: McGraw-Hill; 2000.
30. Marriott HJL, Conover MB. Advanced concepts in dysrhythmias, 3rd edn. St Louis: Mosby; 1998.
31. Lau EW, Pathamanathan RK, Andreng G, Griffith MJ. Electrocardiographic criteria for diagnosis of irregular broad complex tachycardia with a high sensitivity for preexcited atrial fibrillation. Pacing Clin Electrophysiol 2000;23:2040–5.
32. Rosen P, Barkin R, Yealy DMZ, Delbridge TR. Emergency medicine: concepts and clinical practice, 4th edn.. St. Louis: Mosby; 1998.
33. Cohen TJ, Tucker KJ, Abbott JA, et al. Usefulness of adenosine in augmenting ventricular preexcitation for noninvasive localization of accessory pathways. Am J Cardiol 1992;69:1178–85.
34. Ruskin JN, Akhtar M, Damato AN, Ticzon AR, Lau SH, Caracta AR. Abnormal Q waves in Wolff-Parkinson-White syndrome. Incidence and clinical significance. JAMA 1976;235:2727–30.
35. Mandell WJ. Cardiac dysrhythmias. Philadelphia: Lippincott Company; 1995.
36. Sellers TD Jr, Campbell RWF, Bashore TM, Gallagher JJ. Effects of procainamide and quinidine sulfate in the Wolff-Parkinson-White syndrome. Circulation 1977;55:15–22.
37. Mandel WJ, Laks MM, Obayashi K, et al. The Wolff-Parkinson-White syndrome: pharmacologic effects of procainamide. Am J Heart 1975;90:744–54.
38. Bennett DH. Acute prolongation of myocardial refractoriness by sotalol. Br Heart J 1992;47:521–6.
39. Mitchell LB, Wyse DG, Duff HJ. Electropharmacology of sotalol in patients with Wolff-Parkinson-White syndrome. Circulation 1987;76:810–8.
40. Fitton A, Sorkin EM. Sotalol. An updated review of its pharmacological properties and therapeutic use in cardiac dysrhythmias. Drugs 1993;46:678–719.
41. Krahn AD, Klein GJ, Yee R. A randomized, double-blind, placebo-controlled evaluation of the efficacy and safety of intravenously administered dofetilide in patients with Wolff-Parkinson-White syndrome. Pacing Clin Electrophysiol 2001;24:1258–60.
42. Camm AJ, Katritsis D, Nunain SO. Effects of flecainide on atrial electrophysiology in the Wolff-Parkinson-White syndrome. Am J Cardiol 1992;70:33A–37A.
43. Feld GK, Nademanee K, Stevenson W, Weiss J, Klitzner T, Singh BN. Clinical and electrophysiologic effects of amiodarone in patients with atrial fibrillation complicating the Wolff-Parkinson-White syndrome. Am Heart J 1988;115:102–7.
44. Tuzcu EM, Gilbo J, Masterson M, Maloney JD. The usefulness of

amiodarone in management of refractory supraventricular tachydysrhythmias. Cleve Clin J Med 1989;56:238–42.
45. Varriale P, Sedighi A, Mirzaietehrane M. Ibutilide for termination of atrial fibrillation in the Wolff-Parkinson-White syndrome. Pacing Clin Electrophysiol 1999;22:1267–9.
46. Morady F. Radio-frequency ablation as treatment for cardiac arrhythmias. N Engl J Med 1999;340:534–44.
47. Jackman WM, Wang X, Friday KJ, et al. Catheter ablation of accessory atrioventricular pathways (Wolff-Parkinson-White syndrome) by radiofrequency current. N Engl J Med 1991;324:1605–11.
48. Leather RA, Leitch JW, Klein GJ, Guiraudon GM, Yee R, Kim YH. Radiofrequency catheter ablation of accessory pathways: a learning experience. Am J Cardiol 1991;68:1651–5.