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Lack of Prognostic Value of Syncope in Patients With Wolff-Parkinson-White Syndrome

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Syncope in patients with Wolff-Parkinson-White syndrome may be considered a premonitory event heralding the future development of sudden death. Therefore, the clinical and electrophysiologic data of 101 patients with Wolff-Parkinson-White syndrome referred for invasive evaluation of known arrhythmias were reviewed to assess the incidence and clinical relevance of syncope.

Thirty-six patients reported the occurrence of one or more syncopal episodes (group 1) and 65 patients had no syncope (group 2). These two groups did not differ significantly with regard to age, gender, incidence and characteristics of arrhythmia, clinical history, frequency of arrhythmic events and presence of associated cardiac disease. There were 10 patients in group 1 and 12 in group 2 who had ventricular fibrillation.

There were no statistical differences between the two groups with respect to the effective refractory period of the right atrium, atrioventricular node, accessory pathway and right ventricle. Furthermore, no differences between the two groups were noted with respect to cycle length of circus movement tachycardia, mean heart rate during atrial fibrillation, and minimum RR interval during atrial fibrillation. In addition, the accessory pathway location was not significantly different between group 1 and group 2.

The occurrence of syncope could not be predicted from any electrophysiologic finding and this symptom had a low sensitivity and specificity for recognition of dangerous rapid heart rates. Furthermore, the prognostic value of syncope was less accurate and predictive than the shortest RR interval during atrial fibrillation and the anterograde effective refractory period of the accessory pathway for aborted sudden death occurrence.

These data show that syncope is a relatively common clinical finding in patients with Wolff-Parkinson-White syndrome referred for electrophysiologic testing and its occurrence does not identify patients with a risk for sudden death. Furthermore, there is no electrophysiologic variable that could account for syncope occurrence suggesting that other factors may play a role in the genesis of this event in symptomatic patients with Wolff-Parkinson-White syndrome.

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Patients with the Wolff-Parkinson-White syndrome frequently experience palpitation, chest pain or dyspnea resulting from paroxysmal tachyarrhythmias (1). Syncope is a less-often described symptom but its occurrence is regarded as an alarming event (2,3). In fact, syncope suggests the occurrence of rapid tachyarrhythmias and the possibility that they might result in sudden death unless effectively managed. In this respect the syncope occurrence in patients with Wolff-Parkinson-White syndrome could be considered a warning event. Therefore, any patient who has experienced this symptom should be promptly and thoroughly evaluated and managed more aggressively (3). However, to date the reason for syncope occurring in patients with Wolff-Parkinson-White syndrome is not clear and previous studies (4–8) have yielded no unusual electrophysiologic findings in such patients. Also, the clinical relevance and the incidence of syncope in patients with Wolff-Parkinson-White syndrome are still a matter of debate (4,6,8) and the prediction of syncope in such patients by electrophysiologic testing has been assessed only to a minimal extent (8). Furthermore, the sensitivity and specificity of syncope alone and in comparison with other high-risk markers for identifying patients at risk for sudden death have not been evaluated.

The purpose of this retrospective study was to determine the incidence of syncope in a population of symptomatic patients with Wolff-Parkinson-White syndrome referred for medical or nonpharmacologic treatment and to compare the electrophysiologic findings of patients with a history of syncopal episodes with those of patients without syncope. In addition, this study was designed to define the predictability of the occurrence of syncope by electrophysiologic variables and to define the sensitivity and the specificity of syncope as a risk factor for sudden cardiac death in the event of atrial fibrillation. Finally, this study compares the additional prognostic value of the symptom "syncope" in patients with Wolff-Parkinson-White syndrome and atrial fibrillation demonstrating a short RR interval ≤250 ms and in patients with an anterograde effective refractory period of the accessory

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pathway <270 ms as the accepted reference standard in determining the risk for sudden death in these patients (9–12).

Methods

Study patients. One hundred one consecutive patients (66 male, 35 female) with a mean age of 35 years (range 3.3 to 65) with Wolff-Parkinson-White syndrome were consecutively included in the study. Syncope was defined as a sudden transient loss of consciousness with an inability to maintain postural tone. There were 36 patients (36%) who reported at least one episode of syncope (group 1) and 65 patients (64%) without a history of syncope (group 2). Twenty-two patients (22%) presented an episode of cardiac arrest, most likely due to atrial fibrillation degenerating into ventricular fibrillation. This arrhythmic event was considered as an aborted sudden death because of the prompt and successful management of the tachyarrhythmia.

Patients with sick sinus syndrome, right or left ventricular dysplasia, or both, or coronary artery disease were excluded. Medical history, physical examination, 12-lead electrocardiogram (ECG), echocardiogram, 24-h ambulatory ECG recording and a symptom-limited graded treadmill or bicycle exercise test (modified Bruce protocol) were performed in all patients. Cardiac catheterization was carried out, if warranted, for associated cardiac lesions or if the patient was a potential candidate for surgical ablation of the accessory pathway. A neurologic assessment was obtained in all patients experiencing syncope.

Electrophysiologic testing. The study was performed in the nonsedated, postabsorptive state after informed consent was obtained in writing. All antiarrhythmic drugs were discontinued at least five drug half-lives before study. Two bipolar catheters and one quadripolar catheter were introduced transvenously and positioned in the high right atrium, right ventricular apex and coronary sinus, respectively. A tripolar catheter was placed across the tricuspid valve for His bundle recording. The 12-lead surface ECG was recorded simultaneously with intracardiac signals on a 16-channel recorder (Mingograf, Siemens) at a paper speed between 100 and 200 mm/s. Programmed stimulation was performed according to standard technique (13) using a stimulator capable of delivering square-wave stimuli at twice diastolic threshold. The effective refractory period was measured after an 8-beat drive at a basic cycle length of 600 or 500 ms.

The presence and location of an accessory pathway, as well as the mechanism of tachycardias observed, were determined by criteria previously described (3,14). Accessory pathway location was exactly assessed in 82 patients undergoing surgical ablation of the accessory pathway. The accessory pathway location was restricted to four main areas: left, septal, right and multiple. The left and right areas included accessory pathways located laterally and posteriorly and the septal area included paraseptal and posteroseptal accessory pathways. The so-called multiple area collected all accessory pathways located at different sites unrelated to the actual position.

An anterograde effective refractory period of the accessory pathway of <270 ms and a shortest RR interval between two consecutive pre-excited QRS complexes during atrial fibrillation of ≤250 ms were considered variables of higher risk for sudden death according to previous studies (10–12).

Statistical analysis. All variables measured are reported as mean values ± SD. Statistical analysis of data was performed using the unpaired Student's *t* test, chi-square test and Pearson correlation test, when appropriate. A p value <0.05 was considered significant.

Results

Patient characteristics (Table 1). No patients enrolled in this study suffered from a neurologic disease that could explain the syncopal event and the majority of patients who had syncope reported palpitation or chest discomfort before the syncope.

The distribution of the conduction modality via the accessory pathway was not statistically significant between the groups (Table 1). Similarly, no statistical difference in ECG-documented arrhythmic symptoms was noted between the two groups and there was no relation between the syncopal episode and an arrhythmic event.

The incidence of arrhythmic events was similar in the two groups (Table 1). However, from these calculations two patients of group 1 and three of group 2 were excluded because they had a high monthly incidence of reciprocating tachycardia which would distort the whole group analysis.

Most patients had a history of orthodromic reciprocating tachycardia, that is, 25 patients of group 1 (69%) and 52 patients of group 2 (80%, p = NS), and in 3 patients of group 1 (8%) and in 2 patients of group 2 (3%) an antidromic reciprocating atrial tachycardia occurred (p = NS).

The heart rate during spontaneous episodes of tachycardia before study, the mean average RR interval during documented episodes of atrial fibrillation and the minimal RR interval between two maximally pre-excited QRS complexes during atrial fibrillation were similar in the two groups (Table 1). In eight patients experiencing syncope (Table 2), it was possible to record an ECG during the syncopal episode demonstrating atrial fibrillation in five and orthodromic reciprocating tachycardia in the other three patients. The mean cycle length of the atrial reciprocating tachycardia was 329 ms and the mean shortest RR interval between two consecutive pre-excited beats was 252 ms.

Antiarrhythmic therapy employed to suppress known or suspected arrhythmias in the majority of patients before referral was not statistically significantly different (Table 1). However, quantitative information regarding drug dosage at the time of syncope could not be obtained.

Associated cardiac diseases. The most common cardiac diseases associated with the Wolff-Parkinson-White syn-

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Table 1. Clinical Characteristics of 101 Patients

	Group 1 (n = 36)	Group 2 (n = 65)
Age (yr)		
Mean	35 ± 16	34 ± 15
Range	3–60	9–60
Gender		
Men	24 (67%)	43 (66%)
Women	12 (33%)	22 (34%)
Conduction over the accessory pathway		
Manifest	32 (89%)	60 (92%)
Intermittent	1 (3%)	3 (5%)
Concealed	3 (8%)	2 (3%)
History of tachyarrhythmias		
RT	12 (33%)	23 (35%)
A fib	8 (22%)	11 (17%)
RT and A fib	16 (44%)	31 (47%)
VT–VF (cardiac arrest)	10 (28%)	12 (18%)
Incidence of arrhythmic events (events/month)		
RT	2.7	2.5
A fib	0.6	0.7
Heart rate during arrhythmia		
RT (beats/min)	188 ± 35	186 ± 22
Average RR interval A fib (ms)	364 ± 49	370 ± 76
Shortest RR interval A fib (ms)	245 ± 40	250 ± 44
No. of drugs by history	2.4	2.3
Associated cardiac anomalies		
Ebstein's anomaly	3 (8%)	1 (2%)
MVP	7 (19%)	9 (14%)
Others	5 (15%)	5 (8%)

None of the differences between groups are statistically significant. A fib = atrial fibrillation; MVP = mitral valve prolapse; RT = reciprocating atrial tachycardia; VF = ventricular fibrillation; VT = ventricular tachycardia.

drome, identified according to the Feigenbaum echocardiographic criteria (15), were manifest or forme fruste Ebstein's anomaly (4%), mitral valve prolapse (16%), tricuspid valve prolapse (2%) and ostium secundum atrial septal defect (1%). There was no difference in the incidence of these diseases in the two patient groups with or without syncope; furthermore, the syncopal episode occurrence was unrelated to any of these cardiac diseases (r = 0.35, p = NS).

Electrophysiologic variables (Table 3). No patient enrolled in this study presented a PA or AH interval duration, or both, out of the normal range. The effective refractory period of the right atrium, left atrium (assessed through the coronary sinus), AV node and right ventricle demonstrated no significant difference between the two groups. The accessory pathway refractoriness, evaluated in the anterograde as well as in the retrograde direction, did not distinguish patients with syncope from those without. A comparable number of patients had an anterograde effective refractory period of the accessory pathway <270 ms. Exact determinations were obtained in 24 patients of group 1 (67%) and in 40 patients of group 2 (62%). In the remaining patients only estimated values were assigned where atrial refractoriness was reached before the effective refractory period of the accessory pathway. Furthermore, considerable similarity of both groups was found in the percentage of patients demonstrating a shortest RR interval during atrial fibrillation of ≤250 ms.

The cycle length during spontaneous episodes of reciprocating tachycardia before this study was very similar to that attained during tachycardia induced in the laboratory (Fig. 1). Furthermore, the cycle length of the reciprocating tachycardia induced during electrophysiologic testing was comparable in group 1 and group 2 (Table 3).

A good correlation between spontaneous episodes of atrial fibrillation and those induced during electrophysiologic study was found with respect to the mean heart rate (Fig. 2) and the minimum RR interval between two maximally pre-excited QRS complexes (Fig. 3). There was a considerable overlap in the average RR intervals during atrial fibrillation (Table 3), as well as in the shortest RR interval between two maximally pre-excited QRS complexes during the atrial fibrillation. In addition, considerable agreement was found between the groups in the percentage of patients demonstrating a shortest RR interval of ≤250 ms during atrial fibrillation.

The two groups could not be distinguished on the basis of accessory pathway location. In fact, a left-sided accessory pathway was present in 61% of patients of group 1 and in 65% of patients of group 2 and a right-sided connection in

Table 2. Arrhythmia Profile of the Eight Patients With an ECG-Documented History of Syncope

Patient	Gender	Age (yr)	ECG-Arrhythmia RT (ms)	ECG-Arrhythmia A fib (ms)	EPS-Arrhythmia RT (ms)	EPS-Arrhythmia A fib (ms)	AP Location
1	M	25	—	240	353	200	Left lateral
2	M	34	353	—	375	[illegible]	Left lateral
3	F	48	—	280	—	[illegible]	[illegible] posterior
4	M	36	—	200	333	190	Left lateral + septal
5	M	58	—	280	316	320	Right anterior
6	F	25	353	—	353	270	Left lateral
7	F	29	—	260	316	250	Left posteroseptal
8	M	47	280	—	210	—	Left lateral

AP = accessory pathway; ECG = clinically documented; EPS = electrophysiologic study. Other abbreviations as in Table 1.

Table 3. Electrophysiologic Variables in 101 Patients

	Group 1 (n = 36)	Group 2 (n = 65)
PA interval	33 ± 9	32 ± 7
AH interval	95 ± 33	91 ± 40
ERP, right atrium	241 ± 33	231 ± 40
ERP, AV node, ante	269 ± 51	246 ± 43
ERP, AV node, retro	247 ± 38	250 ± 51
ERP, accessory pathway, ante	252 ± 56	246 ± 54
ERP, accessory pathway, retro	227 ± 30	246 ± 42
Right ventricle	220 ± 31	230 ± 34
ERP-AP <270 ms	24 (67%)	40 (62%)
Induced arrhythmias		
Cycle length RT	326 ± 58	333 ± 46
Average RR interval during A fib	347 ± 48	366 ± 43
Minimum RR interval during A fib	249 ± 48	246 ± 43
SRR-A fib ≤250 ms	27 (75%)	43 (66%)

All values are expressed in ms. None of the differences between the two groups are statistically significant. ante = anterograde; ERP = effective refractory period; retro = retrograde; RT = reciprocating tachycardia; SRR = shortest RR interval during atrial fibrillation. Other abbreviations as in Tables 1 and 2.

14% of patients of group 1 and in 12% of patients of group 2. A septal pathway was present in 10% and 14% of group 1 and 2, respectively. Five patients (14%) in group 1 and six patients (9%) in group 2 had multiple-sided accessory pathways.

Prediction of syncope by electrophysiologic variables (Table 4). There seems to be no electrophysiologic variable enabling prediction of syncope occurrence. In fact, the cycle length of induced reciprocating tachycardia and the accessory pathway refractoriness of <270 ms showed little specificity and sensitivity. Similarly, a minimal RR interval ≤250 ms during atrial fibrillation could not account for the syncope; sensitivity, specificity and positive and negative predictive values were comparable with those obtained from the analysis of cycle length during the tachycardia or anterograde accessory pathway refractoriness.

Prognostic value of electrophysiologic variables and syncope (Table 5). There were 10 patients of group 1 (28%) and 12 patients of group 2 (18%) who had a history of aborted sudden death. A shortest RR interval during induced atrial fibrillation of ≤250 ms was present in 20 of these 22 patients with a history of cardiac arrest. Furthermore, 17 of the group who experienced aborted sudden death showed an anterograde refractory period of the accessory pathway of <270 ms in the electrophysiologic study. However, one patient who presented with cardiac arrest was excluded because it was not possible to determine exactly the anterograde effective refractory period of his accessory pathway.

There was no correlation between syncopal episodes and very rapid ventricular rates occurring during atrial fibrillation. In fact, syncope was a less sensitive and specific marker for recognition of a dangerous rapid ventricular rate (Table 5); only 10 patients with a syncopal episode had a history of life-threatening rapid ventricular rates during atrial fibrillation.

The symptom of "syncope," in combination with the two accepted risk markers for sudden death in Wolff-Parkinson-White syndrome patients, failed to improve the prognostic value of these two markers. In fact, only 8 of the 22 patients with cardiac arrest had both a syncopal episode and a shortest RR interval during atrial fibrillation of ≤250 ms and

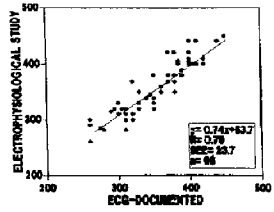

Figure 2. Correlation analysis of mean heart rate (cycle length in ms) during atrial fibrillation induced during electrophysiologic study and during ECG-documented spontaneous episodes.

Figure 1. Correlation analysis of heart rate (cycle length in ms) during tachycardia induced during electrophysiologic study and during ECG-documented spontaneous episodes.

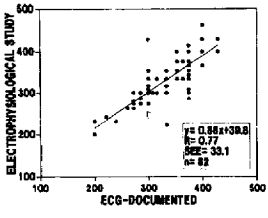

Figure 3. Shortest RR interval between two maximally pre-excited QRS complexes during atrial fibrillation in the clinically documented arrhythmia in relation to the data during electrophysiologic study.

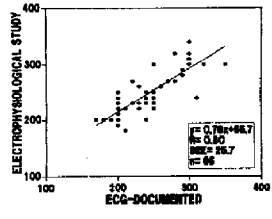

Table 4. Prediction of Syncope by Electrophysiologic Variables

	Sensitivity (%)	Specificity (%)	Positive Predictive Value (%)	Negative Predictive Value (%)
Cycle length RT <300 ms	58	68	50	75
SRR-A fib ≤250 ms	77	34	38	76
ERP-AP <270 ms	80	32	37	76

Abbreviations as in Tables 1 to 3.

only 9 patients presented both with syncope and a refractory period of accessory pathway of <270 ms (Table 5).

Discussion

Prognostic role of syncope. Natural history studies (1) suggest that many patients with Wolff-Parkinson-White syndrome are asymptomatic or largely symptom free. A syncopal or near syncopal episode is a less common event in these patients. The occurrence of this symptom suggests the possibility of very rapid reciprocating tachycardia or atrial fibrillation with a rapid ventricular response due to anomalous conduction. In this case the syncope could be regarded as a warning event heralding the future development of sudden cardiac death. Because of the potential risk of sudden death in patients with an accessory pathway capable of a sustaining rapid ventricular rate during atrial fibrillation, syncope might be a marker of this high-risk patient subgroup. In that event individuals with a history of syncope warrant complete electrophysiologic testing and aggressive therapy as well.

However, in contrast to this opinion, our data show that syncope is a more benign clinical symptom in patients with Wolff-Parkinson-White syndrome. In fact, according to Yee et al. (4) and Paul et al. (8), who reported a syncope incidence of 22% and 19%, respectively, the symptom "syncope" was a relatively common characteristic in the history of our patients, present in 36% of our study population. Consistent with the findings of Yee et al. (4), all patients of this study with syncope had clinical characteristics, arrhythmic events and considerable similarities in heart rate during tachyarrhythmia episodes comparable with those not reporting syncope.

Prediction of syncope occurrence by electrophysiologic study. Assuming that a relation exists between syncope and episodes of tachyarrhythmias in Wolff-Parkinson-White patients, a particular electrophysiologic pattern might be expected. However, no patient of our study population showed abnormal intraatrial or atrioventricular electrophysiologic variables and, significantly, exceptional rapid anterograde conduction over the accessory pathway was not found. In addition, there was considerable agreement in mean heart rate during reciprocating tachycardia or atrial fibrillation between the patients' inherent tachycardia and those induced during electrophysiologic study. The good relation between clinical arrhythmia characteristics and electrophysiologic findings in our patients is in agreement with Rinne et al. (16). These similarities suggest that tachyarrhythmias are probably not the cause of syncope. This is partially supported by our findings that eight patients of this study who had experienced syncope showed electrophysiologic variables similar to those recorded at the time of their syncopal event.

Therefore, these data suggest the "primum movens" of syncope occurring in Wolff-Parkinson-White patients to be classified as unclear. We consider it important that many patients in our study were able to sustain rapid ventricular rates during atrial fibrillation or fast reciprocating tachycardia and yet were not subject to syncope. On the other hand, a similar percentage of patients with and without syncope showed a shortest interval of preexcited beats during atrial fibrillation of ≤250 ms or an anterograde refractory period of the accessory pathway of <270 ms. Although our and Yee's (4) data are in disagreement with the report of Paul et al. (8) who were able to demonstrate a strong relation and predictability of syncope and the shortest RR interval during atrial

Table 5. Prediction of Sudden Death by Syncope and Electrophysiologic Variables

	Sensitivity (%)	Specificity (%)	Positive Predictive Value (%)	Negative Predictive Value (%)
Syncope	45	67	38	67
SRR-A fib ≤250 ms	91	36	29	93
SRR-A fib ≤250 ms + syncope	26	81	36	81
ERP-AP <270 ms	81	31	27	84
ERP-AP <270 ms + syncope	43	79	39	82

Abbreviations as in Tables 1 to 3.

fibrillation, it must be noted that they studied a younger patient population and, in addition, selected a lower value of the RR interval (≤220 ms) during atrial fibrillation.

Predictive value of electrophysiologic testing for the risk of sudden death. There is general agreement in considering the shortest RR interval between two consecutive pre-excited complexes during atrial fibrillation and the anterograde effective refractory period of accessory pathway as high-risk markers (9–11,17). Although accessory pathway refractoriness tends to correlate with the shortest RR interval (17), the effective refractory period cannot always be measured and other variables, such as heart rate, may also affect the refractoriness (17). Therefore, the accessory pathway refractoriness lacks sensitivity and is less predictive than the shortest RR interval during atrial fibrillation (10); in this respect, our data confirm previous reports (10–12) on the superiority of the shortest RR interval between two consecutive pre-excited beats during atrial fibrillation of ≤250 ms as a better predictor of risk of sudden death than the anterograde effective refractory period of accessory pathway of <270 ms.

Prediction of sudden death by syncope. The data of this study support the hypothesis that the clinical finding of "syncope" has a poor predictive accuracy for identifying patients with Wolff-Parkinson-White syndrome who are at risk for sudden death. One reason for this is suggested by Bayesian analysis and relates to the relatively low incidence of sudden death in patients with Wolff-Parkinson-White syndrome. Nevertheless, because syncope has been regarded as an alarming event in such patients, a higher sensitivity of this event in respect to sudden death should be expected than we actually found. The failure of the symptom "syncope" to be a marker for risk of sudden death is evidence by its comparison with other risk markers, in particular, the shortest RR interval during atrial fibrillation. In fact, using "syncope" in combination with the other two risk markers examined improves the specificity of the test, demonstrating that patients with a long RR interval during atrial fibrillation (>250 ms) or with an anterograde effective refractory period >270 ms, or both, but without experiencing syncope have a better prognosis.

The potential reason for this failure of syncope to predict sudden death may lie in the indistinguishability of patients who really experienced unconsciousness because of rapid conduction through the accessory pathway from other patients in whom the cause of syncope could be related to extracardiac mechanisms. Furthermore, other speculative mechanisms of syncope in Wolff-Parkinson-White syndrome patients could be related to abnormal catecholamine response and activation of cardiac vagal afferents, as recently suggested by Alquist et al. (18) or to mechanical activation either by cardiac distension and stretching or by vigorous, forceful and rapid systolic contraction generating "ventricular syncope," as discussed by Abboud (19) resulting both in marked vasodilatation and bradycardia.

Limitation of study. A retrospective study is always subject to several limitations because of lack of collected data; in this way, this study cannot cover the role of psychologic, metabolic and autonomic factors active at the time of syncope occurrence. Furthermore, absence of a statistically significant difference between the two patient groups could be related to the sample size (type 2 error). Our relatively high incidence of syncope (36%) compared with the 22% and 19% incidence previously reported (4,8) could be attributed to different selection criteria or to a preselected patient population referred to our institution especially for surgical treatment of Wolff-Parkinson-White syndrome. However, even considering the syncope group alone, these patients do not present more risk features than other symptomatic patients with Wolff-Parkinson-White syndrome referred for electrophysiologic study.

Another possible limitation of this study ensues from the patient's supine position during the electrophysiologic study that could drastically mask the hemodynamic mechanism of failure to compensate for the sudden rate increase during tachyarrhythmia. In fact, previous studies (20) reported complex adjustments in contractility, vascular resistance, stroke volume and atrioventricular timing during tachycardia, the interrelation of which is still poorly understood, and one or more of these factors could contribute to impair the patient's hemodynamic state. Although it would be worthwhile to evaluate the symptom of "syncope" in a control study population of normal subjects, this is not easy to do because the majority of subjects with syncope seem to have some form of organic heart disease.

Conclusions. The results of this study suggest that syncope is a relatively common feature in patients with Wolff-Parkinson-White syndrome referred for electrophysiologic testing; its occurrence alone fails to correctly identify patients at risk of sudden death. The main cause of syncope occurring in patients with Wolff-Parkinson-White syndrome remains obscure and further studies are needed to elucidate the mechanism of such events. Furthermore, the absence of clinical relevance of syncope in these patients and the inability to improve the risk stratification by electrophysiologic variables suggest a more restrictive management in patients with Wolff-Parkinson-White syndrome and syncope. The decision to proceed with invasive electrophysiologic evaluation or aggressive treatment, or both, of patients with Wolff-Parkinson-White syndrome should be based on other clinical and electrophysiologic findings instead of emphasizing the occurrence of syncope.

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