

Inducible multiform ventricular tachycardia in Wolff-Parkinson-White syndrome

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SUMMARY The induction of ventricular tachycardia by ventricular stimulation was investigated in 46 patients with isolated Wolff-Parkinson-White syndrome (10 concealed) and 36 control patients with normal electrocardiograms and conduction systems. None of those studied had spontaneous ventricular arrhythmias or myocardial or valve disease. Single and double ventricular extrastimuli were delivered at 3 cycle lengths (sinus, 600 ms, 400 ms). In the controls ventricular stimulation induced one episode (3%) of non-sustained ventricular tachycardia. Ventricular stimulation in patients with Wolff-Parkinson-White syndrome induced two episodes of ventricular fibrillation and 15 episodes of non-sustained multiform ventricular tachycardia (37%). Ventricular arrhythmias were induced only in patients with overt Wolff-Parkinson-White syndrome. In 14 patients the conformation of the electrocardiogram at the start of ventricular tachycardia resembled that of major pre-excitation. The absence of inducible ventricular tachycardia in patients with concealed Wolff-Parkinson-White syndrome suggests that anterograde conduction via an atrioventricular accessory pathway is required to initiate the ventricular arrhythmias: the ventricular tachycardia may be associated with reentry of impulses via atrioventricular connection during the phase of ventricular vulnerability. The similarity between the start of ventricular tachycardia and pre-excitatory complexes may also indicate local reentry into the ventricular area occupied by the bypass tracts.

Patients with Wolff-Parkinson-White syndrome and anterograde pre-excitation are more likely to have inducible multiform ventricular tachycardia than individuals without Wolff-Parkinson-White syndrome.

Patients with Wolff-Parkinson-White syndrome may present with syncope which is often assumed to be related to atrial fibrillation with a rapid ventricular response caused by conduction through an atrioventricular connection. Ventricular tachycardia has also been regarded as the probable cause of neurological symptoms in patients with ventricular pre-excitation.¹

We have compared the occurrence of ventricular tachycardia induced by programmed electrical stimulation in patients with Wolff-Parkinson-White syndrome with that in control patients.

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Patients and methods

PATIENTS

We studied 46 patients with the Wolff-Parkinson-White syndrome (28 men and 18 women, aged 8 to 62 years (36 (14))). Wolff-Parkinson-White syndrome was diagnosed in 36 patients who had the typical delta wave recorded in electrocardiograms taken during sinus rhythm. Ten patients had an accessory pathway that conducted only in the retrograde direction. Electrophysiological studies were performed to measure the effective refractory period of the bypass tracts or the mechanism of paroxysmal junctional tachycardia. Twenty four hour Holter monitoring and exercise test had established the absence of spontaneous ventricular arrhythmias. No evidence of ischaemic, myocardial, valvar, hyper-

tensive, or congenital heart disease was found in any of the patients by examination, chest x ray, cross sectional echocardiography, or coronary angiography (in men who were ≥ 50). Seventeen patients presented with dizziness or syncope.

We also studied 36 control patients (20 men and 16 women, aged 18 to 80 years (47 (17))). None of the controls had Wolff-Parkinson-White syndrome or ventricular arrhythmias on Holter monitoring. The case history, physical examination, chest x ray, electrocardiogram, echocardiography, and electrophysiological study established that none of the control patients had underlying heart disease. Eleven control patients had an electrophysiological examination to investigate the cause of syncope which was later found to be related to a non-cardiac cause, and 25 had it to evaluate paroxysmal sinus bradycardia or first degree atrioventricular block which were reversed by atropine.

ELECTROPHYSIOLOGICAL TESTING

All patients were studied in the non-sedated post-absorptive state after they had given their informed consent. The right heart was catheterised via the femoral vein with three 6 or 7 French multi-electrode catheters. Electrograms were routinely recorded from the right atrium, atrioventricular junction (His bundle electrogram), and distal coronary sinus. Surface electrocardiographic leads I, III, V1, V6, and intracardiac electrograms were recorded at paper speed of 25 and 100 mm/s on a Siemens multi-channel oscilloscopic recorder. Pacing stimuli were given by a programmable stimulator (Medtronic 1325) for 1.8 s at a strength that was approximately twice the diastolic threshold. The following electrophysiological variables were measured during the study:

- (a) AH and HV basic intervals.
- (b) Anterograde atrioventricular conduction, assessed by atrial pacing at incremental rates until second degree atrioventricular block occurred.
- (c) The refractory periods of the anterograde conduction system determined by extrastimuli techniques; premature atrial stimulation (A/2) was introduced during sinus rhythm and during driven atrial rhythm at a cycle length of 600 ms.
- (d) Retrograde ventricular atrial conduction assessed by ventricular pacing at an incremental rate up to 200 beats/min.
- (e) In patients with Wolff-Parkinson-White syndrome in whom atrial fibrillation had not been induced by any of the preceding procedures rapid atrial pacing was performed until atrial fibrillation was induced.
- (f) Programmed ventricular stimulation was given

at the apex and, in some patients, at the infundibulum of the right ventricle. It was performed as follows: single premature ventricular extrastimulus (S2) was introduced in late diastole during sinus rhythm and paced cycle lengths (S1-S2 intervals of 600 and 400 ms) and the interval was shortened until the ventricle became refractory. Double ventricular extrastimuli (S2 and S3) were introduced during sinus rhythm and during paced cycle lengths starting with an S1-S2 interval that was 10-20 ms longer than the ventricular refractory period; the S2-S3 interval was shortened until S3 did not depolarise the ventricles.

The reproducibility of the initiation of ventricular tachycardia (except for induced ventricular fibrillation) was assessed by performing the stimulation on three occasions after an interval of a few minutes.

DEFINITIONS

The following definitions were used:

Sustained ventricular tachycardia—This lasted for more than 1 minute or required termination by programmed ventricular stimulation or cardioversion before that time.

Non-sustained ventricular tachycardia—A run of at least five consecutive ventricular complexes which terminated spontaneously in < 60 s.

Multiform ventricular tachycardia—Ventricular tachycardia with an unstable (continuously varying) QRS complex configuration recorded in any electrocardiographic lead.

Repetitive ventricular response—One to four intra-ventricular reentries.

Concealed accessory atrioventricular connection was diagnosed during reciprocating tachycardia if one of the following criteria was satisfied:

- (a) Demonstration of eccentric activation by endocardial catheter mapping.
- (b) If premature ventricular depolarisation during reciprocating tachycardia when the bundle of His was refractory caused pre-excitation of the atrium.

Results

RESPONSES TO PROGRAMMED VENTRICULAR STIMULATION

Ventricular tachycardia was induced in 17 (37%) of the 46 patients with Wolff-Parkinson-White syndrome and in only one of the 36 control patients without Wolff-Parkinson-White syndrome. In patients with Wolff-Parkinson-White syndrome all the ventricular tachycardias were multiform, rapid (cycle length 150 to 200 ms), and they could be

reproduced by repeat testing. The diagnosis of ventricular tachycardia was based on the absence of His bundle deflection preceding the complexes of ventricular tachycardia and the existence of complete atrioventricular dissociation during ventricular tachycardia in all patients (fig).

Fifteen ventricular tachycardias were non-sustained. Two were ventricular fibrillation and required cardioversion to stop them. Ventricular arrhythmia was induced in 15 patients by the introduction of two ventricular extrastimuli during paced cycle length and in two patients by the introduction of two ventricular extrastimuli during sinus rhythm. The configuration of the beginning of ventricular tachycardia was similar to major ventricular pre-excitation in 14 patients (fig); this similarity was noted for the first, the second, or the third complex. All these ventricular arrhythmias were induced in patients with overt Wolff-Parkinson-White syndrome; ventricular tachycardia was not induced in patients with a concealed accessory pathway.

In the control group, only one episode of non-sustained, multiform ventricular tachycardia was induced by the S1, S2, S3 technique.

Intraventricular reentry was induced in 21 patients with Wolff-Parkinson-White syndrome and in 20 control patients.

CORRELATIONS BETWEEN CLINICAL AND ELECTROPHYSIOLOGICAL FINDINGS AND INDUCIBILITY OF MULTIFORM VENTRICULAR TACHYCARDIA IN PATIENTS WITH THE WOLFF-PARKINSON-WHITE SYNDROME

Tables 1 and 2 summarise the results. The 17 patients who had inducible ventricular tachycardia were not statistically different in terms of age (31 (14) vs 38 (14)), sex, or location of the atrioventricular connection from the 29 patients in whom ventricular arrhythmia was not induced (table 2).

The occurrence of dizziness or syncope was similar in both groups. In the ten patients with syncope and without inducible ventricular tachycardia, however, syncope could be explained by a short refractory period in the Kent bundle in three patients, carotid sinus hypersensitivity in four patients, and associated conduction disturbances in two patients. One patient had unexplained syncope. In the seven patients with syncope and inducible ventricular tachycardia, three had syncope associated with a short Kent bundle refractory period and in four the cause of the syncope was not known.

The occurrence of atrial fibrillation or flutter was not statistically different in the two groups; however, atrial fibrillation was more common in patients with inducible ventricular tachycardia (5/17 (29%))

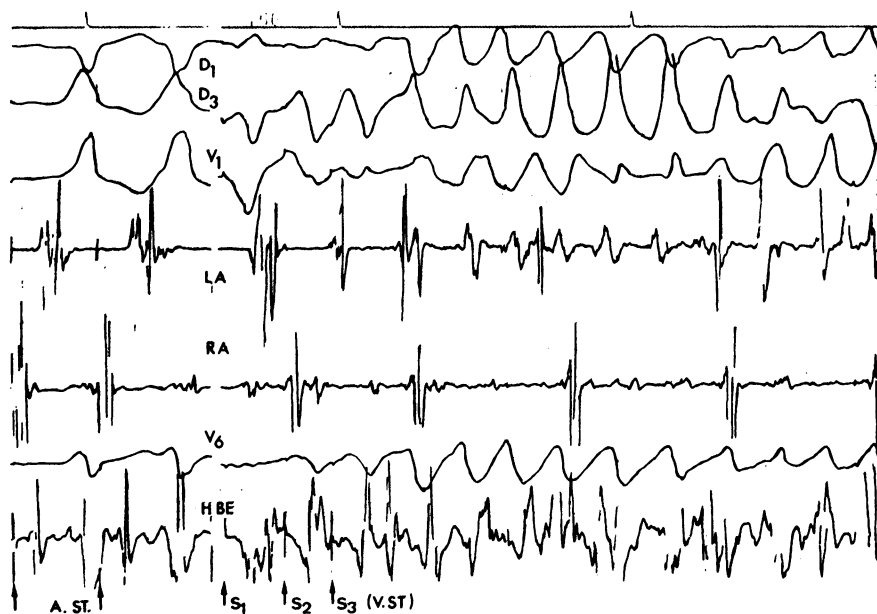


Figure Electrocardiogram obtained during programmed ventricular stimulation in a patient (case 10) with type A Wolff-Parkinson-White syndrome. One atrial premature extrastimulus (left) caused a major pre-excitation complex. Ventricular extrastimuli (S1, S2, S3) induced a multiform ventricular tachycardia which degenerated into ventricular fibrillation (panel 2); the first complexes resemble those of pre-excitation complexes. A ST, atrial stimulation; V ST, ventricular stimulation; HBE, His bundle electrogram; RA, right atrium; LA, left atrium; D1, V1; D3, V6.

Table 1 Data on patients and their electrophysiological characteristics

Patient	Age (yr)	Symptoms	Location of pathway	Anterograde ERP of AP at PCL (600 ms)	Shortest R-R during atrial fibrillation (ms)	Shortest PCL with 1:1 retrograde conduction (ms)	Induced SV arrhythmia	Induced ventricular arrhythmia and number of ventricular reentries
1	25	D-T	S	280	375	430	AF	NS VT (17)
2	36	S	L	450	375	350	AF	NS VT (12)
3	29	T	L	250	260	< 300	PJT-AF	NS VT (7)
4	59	S-T	S	350	750	315	PJT	NS VT (16)
5	32	T	R	230	350	< 300	PJT	NS VT (11)
6	22	T	L	500	> 600	< 300	PJT	NS VT (7)
7	42	T	S	290	545	< 300	PJT	NS VT (5)
8	15	S-T	S	210	170	< 300	PJT-AF	NS VT (11)
9	56	T	L	240	250	< 300	PJT	VF
10	15	D-T	L	230	330	< 300	PJT	VF
11	8	0	R	230	230	< 300	PJT	NS VT (62)
12	15	D	S	330	545	< 300	PJT	NS VT (6)
13	34	0	S	350	315	350	PJT	NS VT (11)
14	39	D	S	220	220	< 300	PJT-AF	NS VT (7)
15	25	T	L	330	250	< 300	PJT-AF	NS VT (6)
16	32	T	L	240	200	< 300	PJT-AF	NS VT (14)
17	47	T	L	280	> 600	< 300	PJT	NS VT (37)
18	21	T	R	230	210	< 300	PJT-AF	—
19	31	0	R + S	300	375	350	PJT	—
20	40	0	R	380	285	400	PJT	3
21	50	T	L	340	400	300	PJT	1
22	27	T	L	200	180	< 300	PJT-AF	3
23	24	T	L	400	1000	< 300	PJT	3
24	37	0	S	300	400	600	—	2
25	40	D	S	180	250	< 300	PJT	—
26	57	S-T	L	600	1000	300	PJT	1
27	33	S	S	450	375	330	—	—
28	33	S	L	600	1000	375	PJT	3
29	55	S-T	L	210	250	< 300	PJT	—
30	35	0	S	400	1000	460	—	—
31	15	0	S	200	300	< 300	—	—
32	39	0	S	250	260	< 300	PJT-AF	3
33	25	S-T	S	240	270	400	AF	3
34	67	S-T	L	310	285	< 300	PJT	1
35	22	T	L	500	600	< 300	PJT	2
36	25	S	S	550	1000	375	—	—
37	29	T	L	Concealed	—	330	PJT	1
38	29	T	L	Concealed	—	430	PJT	3
39	54	T	L	Concealed	—	330	PJT	3
40	62	D-T	L	Concealed	—	315	PJT-AF	1
41	39	T	L	Concealed	—	< 300	PJT	2
42	58	T	L	Concealed	—	330	PJT	0
43	37	T	L	Concealed	—	460	PJT	1
44	28	D-T	S	Concealed	—	< 300	PJT	2
45	47	T	S	Concealed	—	333	PJT	2
46	32	T	L	Concealed	—	300	PJT	2

Symptoms: D, dizziness; S, syncope; T, tachycardia. Location of pathway: L, left lateral; R, right lateral; S, septal. Anterograde ERP of AP at PCL: AP, accessory pathway; ERP, effective refractory period; PCL, paced cycle length. Induced SV arrhythmia: AF, atrial fibrillation or flutter; PJT, paroxysmal junctional tachycardia; SV, supraventricular. Induced ventricular arrhythmia: NS, non-sustained; VF, ventricular fibrillation; VT, ventricular tachycardia.

than in patients without inducible ventricular tachycardia (5/29 (17%)). Only atrial fibrillation developing spontaneously during an induced episode of reciprocating tachycardia or during atrial pacing at an incremental rate of less than 200 beats/min was regarded as abnormal. The shortest paced cycle length associated with 1:1 retrograde conduction was significantly shorter in patients with inducible ventricular tachycardia (291 (45) vs 355 (125 ms)) ($p < 0.05$).

Ventricular arrhythmias were not induced in patients who had concealed accessory atrioventricular pathways that conducted only retrogradely.

In patients with overt Wolff-Parkinson-White syndrome, the anterograde refractory period of the accessory pathway(s) was short (< 250 ms) in the basal state in seven (41%) patients with inducible ventricular tachycardia and in five (26%) without inducible ventricular tachycardia; the values of the

Table 2 Characteristics of patients with Wolff-Parkinson-White syndrome with and without induced ventricular tachycardia (VT)

Characteristics	Patients with VT (17 patients)	Patients without VT (29 patients)
Age (yr) (mean (SD))	31 (14)	38 (14)
Sex	12 M, 5 F	16 M, 13 F
Pathway location:		
Right	2	2
Left	8	16
Septal	7	10
Multiple	0	1
Associated atrial fibrillation	5	5
Syncope	7	10
Kent bundle refractory period:		
< 250 ms	7	5
≥ 250 ms	10	14
Concealed		10

refractory periods of both subgroups were not significantly different (299 (83) vs 349 (137 ms)).

Three patients with induced non-sustained ventricular tachycardia who have been followed up for more than three years are free of ventricular arrhythmia.

Discussion

We found inducible multiform ventricular tachycardia in 37% of patients with Wolff-Parkinson-White syndrome and in 3% of controls. In patients with Wolff-Parkinson-White syndrome the ventricular arrhythmias were only induced in those with the overt syndrome.

Ventricular tachycardia is uncommon in patients with ventricular pre-excitation.^{2,3} Although the occurrence of abnormally widened QRS complexes during paroxysmal arrhythmias in patients with accessory atrioventricular pathways has been noted in several reports, differentiation of ventricular tachycardia from supraventricular tachycardia with a wide QRS complex caused by ventricular pre-excitation can be difficult. The differential diagnosis is usually complex and only resolved by electrophysiological clinical study. In 26 patients with Wolff-Parkinson-White syndrome and a regular tachycardia with a wide QRS complex, Benditt *et al* found only one case of ventricular tachycardia.⁴ A patient reported by Krikler *et al* who had concealed accessory bypass tracts died from ventricular fibrillation which complicated a ventricular tachycardia induced by atrial stimulation.³ Lloyd *et al* considered the cause of the neurological symptoms in four patients with Wolff-Parkinson-White syndrome to be ventricular tachycardia.¹ This was

because the electrophysiological characteristics of the accessory pathways were considered unlikely to support a tachycardia of a rate sufficient to result in syncope and programmed stimulation in the right ventricle induced non-sustained ventricular tachycardia. In the present study we found no correlation between syncope and induced multiform ventricular tachycardia, although ventricular tachycardia could explain the cause of neurological symptoms in four patients in whom the refractory period of the accessory pathway was long.

Milstein *et al* reported inducible multiform ventricular tachycardia in 10.1% of patients with Wolff-Parkinson-White syndrome.⁵ The induction of this clinical ventricular tachycardia was not correlated with a specific electrophysiological finding. The patients were alive and well after a follow up of 20 (11) months.

The significance of induced ventricular tachycardias in patients without spontaneous ventricular arrhythmia is uncertain. Multiform ventricular tachycardia is the most frequent ventricular arrhythmia initiated by programmed electrical stimulation in patients without spontaneous sustained arrhythmias and without structural heart disease. The occurrence of this arrhythmia increases with the number of extrastimuli used in the stimulation protocol.⁶ In most reports the occurrence of non-sustained ventricular tachycardia induced by two extrastimuli was low in normal subjects (0.7% to 2%).^{7,8} In the study of Morady *et al* non-sustained ventricular tachycardia was not induced by stimulation with up to three extrastimuli in patients without structural disease who had no spontaneous ventricular tachycardia, but was frequently induced in patients with structural disease who had no spontaneous ventricular tachycardia.⁹ None the less, a high frequency of multiform ventricular tachycardia was reported by Wellens *et al* in 32 patients without structural heart disease who were evaluated for suspected supraventricular arrhythmias; this arrhythmia was induced by up to two extrastimuli during sinus rhythm and ventricular pacing in 38% of this group.¹⁰

In our study, non-sustained ventricular tachycardia was induced in only one (3%) of the control group. On the other hand, inducible non-sustained ventricular tachycardia was common (37%) in patients with Wolff-Parkinson-White syndrome and without other heart disease.

The high incidence of multiform ventricular tachycardia associated with the preexcitation syndrome resembles the high incidence of atrial fibrillation in Wolff-Parkinson-White syndrome (from 1% to 32%).¹¹ Atrial fibrillation in Wolff-Parkinson-White syndrome has been

related¹¹⁻¹³ either to atrial reentry by an accessory pathway¹³ or to a primary atrial vulnerability.¹⁴ In the present study, ventricular tachycardia was not induced in patients who did not have anterograde conduction via accessory atrioventricular connection; and it may be that such a pathway is required to initiate ventricular tachycardia. Retrograde conduction to atria, which is present in all cases of Wolff-Parkinson-White syndrome, could be responsible for reentry of impulses through the atrioventricular connection and for the occurrence of ventricular depolarisation in the vulnerable phase. Hence atrial fibrillation in Wolff-Parkinson-White syndrome could lead to ventricular fibrillation.¹⁵ In the present study only two patients had no retrograde conduction and ventricular tachycardia was not induced in them. Local reentry into the ventricular area containing the origin of the accessory pathway could also explain the similarity between the first QRS complexes of ventricular tachycardia and those of the pre-excitation QRS complex. All cases of pre-excitation with overt or concealed Wolff-Parkinson-White syndrome may be at high risk of inducible ventricular arrhythmias. This possibility could not be excluded because of the small number of patients with concealed Wolff-Parkinson-White syndrome. In the present study the appearance of the first premature ventricular contraction itself was related to ventricular stimulation. But there could have been automaticity in the accessory ventricular connection¹⁶ or a ventricular premature complex caused by associated organic heart disease. Most spontaneous ventricular tachycardias in the Wolff-Parkinson-White syndrome are reported in patients with underlying heart disease, hypertrophic cardiomyopathy¹³ or previous myocardial infarction.¹ Moreover, it has been shown that the prognosis of the Wolff-Parkinson-White syndrome is better in patients without associated organic heart disease.¹⁷ In the present study, in a 56 year old patient with induced ventricular fibrillation, underlying heart disease was excluded by cross sectional echocardiography, right and left catheterisation, and coronary angiography. In a 15 year old boy who also presented with inducible ventricular fibrillation there was evidence of incipient hypertrophic cardiomyopathy on cross sectional echocardiography; however, this patient was a trained athlete. In the other patients with inducible non-sustained ventricular tachycardia, cross sectional echocardiograms were normal, but incipient associated right ventricular dysplasia could not be excluded. Two young women presenting a ballooning mitral valve without enlargement of left atrium and ventricle, did not have inducible ventricular tachycardia. No patient had protracted arrhyth-

mias that could have caused a disorder indistinguishable from dilated cardiomyopathy.^{18 19}

The worsening of prognosis when the Wolff-Parkinson-White syndrome is associated with another organic heart disease might be explained by mechanisms such as an increased atrial and ventricular vulnerability, leading to atrial fibrillation, followed by a ventricular fibrillation. Associated heart diseases are frequent in Wolff-Parkinson-White syndrome,^{20 21} particularly ventricular diseases which could facilitate ventricular vulnerability, such as operated congenital heart disease, hypertrophic cardiomyopathy, or mitral valve prolapse.

Deaths related to Wolff-Parkinson-White syndrome are rare, however, and generally prognosis is considered to be good.²² Eleven follow up studies of Wolff-Parkinson-White syndrome cases were published between 1962 and 1985. The incidence of deaths related to this syndrome ranged from 0 to 2%.²³ In a recent personal study, the follow up of 195 patients with Wolff-Parkinson-White syndrome for more than three years, there were only two deaths that could be related to this syndrome.²³ One of these was due to an atrial fibrillation, conducted through a Kent bundle with a short refractory period, and the other was a case of sudden death in a 12 year old girl during competitive sport. Neither a clinical examination nor a necropsy was performed in the latter case. There was a high frequency of associated heart disease (24%) and six deaths were related to a complex congenital heart disease.

Studies of the causes of deaths in the Wolff-Parkinson-White syndrome generally report that death was associated with atrial fibrillation with rapid ventricular response via the accessory pathway leading to ventricular fibrillation. The inducibility of ventricular tachycardia in a patient with Wolff-Parkinson-White syndrome therefore is probably not clinically important. Underlying heart disease, however, should always be sought. If it is found, the patient should be considered to be at risk of ventricular arrhythmias because electrical instability is being caused by the accessory pathway and by the associated heart disease.

We conclude that inducible multiform ventricular tachycardia is more common in patients with Wolff-Parkinson-White syndrome who have no organic heart disease but have anterograde pre-excitation than in patients without organic heart disease and without Wolff-Parkinson-White syndrome. Both advanced organic heart disease and the Wolff-Parkinson-White syndrome can lead to the induction of multiform ventricular tachycardia.

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