JACC Vol. 13, No. 4
March 15, 1989:911–6

Ventricular Fibrillation in Six Adults Without Overt Heart Disease

ROBERT LEMERY, MD, PEDRO BRUGADA, MD, PAOLO DELLA BELLA, MD, THIERRY DUGERNIER, MD, HEIN J. J. WELLENS, MD, FACC

Maastricht, The Netherlands

Findings are described in six patients with no clinical evidence of heart disease who had documented ventricular fibrillation (five patients) or ventricular flutter (one patient). The mean age of the six patients, all men, was 34 years (range 26 to 43). Cardiovascular collapse occurred in all and was followed by successful cardioversion. No patient had electrolyte or QT abnormalities. One patient had slight right ventricular enlargement on M-mode echocardiography, and another had a left ventricular pressure gradient at rest of 30 mm Hg with a normal two-dimensional echocardiogram. Holter electrocardiographic monitoring revealed incessant ventricular tachycardia in one patient and nonsustained ventricular tachycardia in three others. Exercise testing revealed nonsustained ventricular tachycardia in one patient.

Ventricular fibrillation was induced at the time of programmed electrical stimulation in four of the six patients. Documented recurrence of ventricular fibrillation or ventricular flutter occurred in three patients, but in only one patient receiving antiarrhythmic drugs. Four patients were treated with amiodarone and one received an automatic implantable cardioverter-defibrillator. All patients are alive after a mean follow-up period of 78 months after the first documentation of their arrhythmia and 37 months after programmed electrical stimulation.

Ventricular fibrillation can occur in the apparently structurally normal human heart. Antiarrhythmic treatment can provide effective control of this malignant arrhythmia.

(J Am Coll Cardiol 1989;13:911–6)

Sudden cardiac death is usually secondary to ventricular fibrillation or rapid ventricular tachycardia in patients with coronary artery disease (1). Nonischemic sudden cardiac death is usually associated with aortic valve disease, cardiomyopathy or the long QT syndrome (1–3). In patients without overt heart disease, ventricular tachycardia has been widely reported (4–9). However, the occurrence of ventricular fibrillation in patients without clinical evidence of heart disease is unusual (8–18). The purpose of this study is to present the clinical course, electrophysiologic features and long-term follow-up of six such patients seen at our institution during the last 6 years.

Methods

Study patients. All six patients were referred to our institution for evaluation and treatment of at least one documented episode of ventricular fibrillation (five patients) or ventricular flutter (one patient) requiring emergency cardiopulmonary resuscitation. Patients underwent a history and physical examination, chest radiography, 12 lead electrocardiogram (ECG) and M-mode and two-dimensional echocardiography. All patients had cardiac catheterization, right and left ventricular angiography and coronary arteriography. Patients 2 and 4 were also given ergonovine maleate during arteriography, although coronary spasm for the cause of the arrhythmia was not suspected clinically in any of the six patients. Continuous ECG monitoring was performed during hospitalization, and patients exercised on a treadmill or bicycle ergometer.

Electrophysiologic study. Programmed electrical stimulation was performed in all patients with use of two to three quadripolar electrode catheters introduced percutaneously from the femoral vein. Four patients had a baseline study after discontinuation of all antiarrhythmic drugs for at least five half-lives, the other two patients were studied while they were receiving antiarrhythmic drug treatment (Table 1). In one patient, programmed electrical stimulation was repeated during treatment with oral amiodarone. Programmed electrical stimulation was performed in patients in the postabsorptive nonsedated state after informed consent was obtained.

From the Department of Cardiology, University of Limburg, University Hospital, Maastricht, The Netherlands. Dr. Lemery is a Research Fellow of the Canadian Heart Foundation, Ottawa, Ontario, Canada.

Manuscript received June 13, 1988; revised manuscript received August 24, 1988, accepted November 3, 1988.

Address for reprints: Robert Lemery, MD, Montreal Heart Institute, 5000 East Belanger Street, Montreal, Quebec H1T 1C8, Canada.

0735-1097/89/$3.50

Table 1. Electrophysiologic Features and Course of Events in Six Patients

Pt	History of CV Collapse	Treatment*		Lown Class		EPS					F-Up (mo)	
		At Collapse	Started	AM	Ex T	Ventricular Arrhythmia	Init (ms)	Term	VERP 600/500 (ms)	Rec	1st Doc	EPS
1	1. 12 yr pre-EPS 10 min collapse	None	None	—	—	—				Yes		
	2. 9 yr pre-EPS collapse; V fl, Rx CV	None		—	—	—				Yes		
			Q × 1 yr	0	0	—						
	3. 1 mo pre-EPS collapse × 3; V fl, Rx CV	None		0	0	V fl (6 beats)	SR_4	Spont	230/220			
						VF	$SR_{3,4}$	CV				
			Q	0	0					No	168	60
2	1. Collapse at home; adm in AF V rate 70 to 120 beats/min; NSVT; VT/VF, Rx CV	None		0	0	NSPVT	$600_{2,3}$	Spont	—			
						VF	600_3	CV				
			Amio 200/day	0	0	NSPVT	$600_{1,2,3}$	Spont		No	56	56
3	1. Visit to clinic for palpitation NSVT doc; Adm VT/VF, Rx CV	None			—	Incess VT (RBBB, RAD)			—			
			IV lido IV diso IV procain IV sotalol	Inc VT								
			Amio 200/day	2	2	—				No	50	50
4	1. Collapse while shoveling; CPR started; doc VF, CV × 3	None		4b	0	VF	SR_4	CV	210/190			
			Amio 200/day	—	0	—				No	30	30
5	1. 10 yr pre-EPS collapse at home, CV × 1	None	Before EPS	4b	—	—				Yes		
	2. 8 yr and 3 yr PT-EPS pre-syncope		aprin, alpren	4b								
	3. 1 mo pre-EPS, collapse while taking a bath, CRP; doc VF, Rx CV, lido IV, bretylium IV; VF next day, Rx CV	None	mex, amio									
			Amio 800+ Aprin 100	4b	4a	NI	SR_3		270/230			
			Amio 400+ Aprin 75	—	4b	—				No	136	14
6	1. 20 mo pre-EPS; collapse; adm VF × 2, Rx CV	None		4b	—	—				Yes		
			Dil 200+ Met 100	—	II							
	2. Collapse at home; adm	Dil 100+ Met 50		4b	0	NSPVT	600_3	Spont	210/190			
						VF	600_3	CV				
	3. In CCU awaiting AICD; recurrent VT/VF, CV × 2	Dil 200+ Met 100		4b	—	—				Yes		
			AICD Dil 200 Prop 320	—	—	—				No	30	9

*All drug dosages are in milligrams per day. adm = admission; AF = atrial fibrillation; AICD = automatic implantable cardioverter-defibrillator; alpren = alprenolol; AM = ambulatory monitoring; amio = amiodarone; aprin = aprindine; CCU = coronary care unit; CPR = cardiopulmonary resuscitation; CV = cardioversion; dil = Dilantin; diso = disopyramide; doc = documented; ESP = electrophysiologic study; ExT = exercise testing; 1st doc = first documentation of the ventricular arrhythmia; F-Up = duration of follow-up; Init = initiation; Inc VT = incessant ventricular tachycardia; IV = intravenous; lido = lidocaine; Lown = Lown arrhythmia class; met = metoprolol; mex = mexiletine; NS = nonsustained; NSPVT = nonsustained polymorphic ventricular tachycardia; procain = procainamide; prop = propranolol; Pt = patient; Q = quinidine; RAD = right axis deviation; RBBB = right bundle branch block; Rec = recurrence of ventricular flutter or ventricular fibrillation; Rx = treatment; Spont = spontaneous; SR = sinus rhythm; Term = termination; V = ventricular; VERP = ventricular effective refractory period; VF = ventricular fibrillation; V fl = ventricular flutter; VT = ventricular tachycardia; 1-2-3-4 = number of ventricular beats given during programmed electrical stimulation (the presence of more than one number refers to reproducible induction of the ventricular arrhythmia); 600/500 = ventricular pacing at 600 or 500 ms.

Intracavitary electrograms and ECG leads I, II, III, V_1, V_4 and V_6 were recorded on a multichannel recorder at a paper speed of 100 mm/s. Programmed electrical stimulation was performed according to our previously described protocol (19) with delivery of rectangular stimuli of 1 to 2 ms duration at twice diastolic threshold. Ventricular stimulation was performed from the right ventricular apex in all patients. Single, double and triple ventricular extrastimuli were given in three patients: in two patients, a fourth ventricular extrastimulus was also given.

Follow-up. Long-term follow-up was obtained in all patients by visit to the outpatient clinic. We assessed the length of follow-up from the initial documentation of ventricular fibrillation or ventricular flutter and from the time of programmed electrical stimulation.

Results

Clinical and laboratory features. All patients were men; their mean age was 34 years (range 26 to 43). A history of palpitation was present in two patients. Patient 4 was the only patient who collapsed during exercise (while shoveling dirt). This patient also had an uncle who died suddenly at the age of 28 years. No patient had a history of chest pain, shortness of breath or alcohol or drug abuse. A history of smoking was present in three patients. Patient 2 worked with insecticides, but no known toxic exposure had occurred.

Physical examination was normal in three patients, and two others had a grade 2/6 systolic ejection murmur. Patient 4 had a systolic click and a grade 2/6 systolic ejection murmur, but had no mitral valve prolapse during M-mode and two-dimensional echocardiography (performed at rest, during Valsalva maneuver and while inhaling amyl nitrate) or during left ventricular angiography.

Chest radiographs were normal in all patients. The ECG showed complete and incomplete right bundle branch block in Patient 1 and Patient 6, respectively. The other four patients had a normal 12 lead ECG. The QTc interval was <0.45 s in all patients.

M-mode and two-dimensional echocardiographic results were normal in all but Patient 6, who had two echocardiographic studies that revealed a slightly increased end-diastolic right ventricular dimension of 34 mm (normal <30 mm). This patient had normal right and left ventriculograms, and no gross cardiac abnormalities noted at the time of implantation of an automatic cardioverter-defibrillator.

Cardiac catheterization revealed a slightly elevated right ventricular end-diastolic pressure of 10 mm Hg in Patient 5, and normal right ventricular pressure in all other patients. The left ventricular end-diastolic pressure at rest was slightly elevated (15 mm Hg) in Patient 2. Patient 3 had a left ventricular end-diastolic pressure of 28 mm Hg, but incessant ventricular tachycardia was present at the time of the study. Patient 4 had a left ventricular pressure gradient at rest of 30 mm Hg, which increased to 35 mm Hg with isoproterenol (this patient had the systolic click and murmur described earlier). The remaining three patients had a normal left ventricular pressure. All patients had a left ventricular ejection fraction ≥60%. Angiographic features were normal in all but Patient 4, who exhibited hypertrophy of the papillary muscles.

Electrophysiologic features (Table 1). In all patients with documented ventricular fibrillation and in the patient with ventricular flutter the arrhythmia occurred in the absence of antiarrhythmic drug therapy. In three patients, it occurred shortly after admission to the hospital; the other three patients were resuscitated from an out-of-hospital cardiac arrest. Patient 3 had incessant ventricular tachycardia at the time of admission, and responded promptly to oral amiodarone given over the first few days of admission. Nonsustained ventricular tachycardia was present during continuous monitoring in three patients, and Patient 6 also had isolated R on T ectopic ventricular extrasystoles (coupling interval <300 ms). Exercise testing revealed nonsustained ventricular tachycardia in one patient.

Inducible ventricular fibrillation. During programmed electrical stimulation, ventricular fibrillation was induced in two patients with use of three premature ventricular stimuli, in one patient with both three and four ventricular extrastimuli and in another patient with four ventricular extrastimuli. Patient 3 had incessant ventricular tachycardia at the time of programmed electrical stimulation, and overdrive pacing only partially suppressed the ventricular tachycardia; endocardial mapping showed earliest endocardial activation during ventricular tachycardia at the anterior portion of the left side of the ventricular septum. In this patient, the ventricular effective refractory period could not be obtained, but in four other patients, it was obtained during right ventricular pacing at 100 and 120 beats/min and ranged from 190 to 270 ms.

Inducible ventricular tachycardia. None of the patients had sustained monomorphic ventricular tachycardia induced during programmed electrical stimulation. Nonsustained polymorphic ventricular tachycardia was induced in two patients, and nonsustained ventricular flutter was induced in the patient who presented with sustained ventricular flutter.

Antiarrhythmic drug studies. Three patients underwent programmed electrical stimulation while receiving antiarrhythmic drugs. Two patients had a favorable study: with use of up to three premature ventricular stimuli, Patient 4 (amiodarone, 800 mg/day and aprinidine, 100 mg/day) had no inducible tachycardia, whereas Patient 2 (amiodarone, 200 mg/day) had inducible nonsustained polymorphic ventricular tachycardia. In Patient 6, who collapsed while taking Dilantin (100 mg/day) and metoprolol (50 mg/day), ventricular fibrillation was induced with three premature ventricular stimuli.

Follow-up. Documented ventricular flutter recurred in Patient 1 after discontinuation of quinidine therapy. Patient 5 had recurrence of ventricular fibrillation after recent discon-

JACC Vol. 13, No. 4
March 15, 1989:911–6

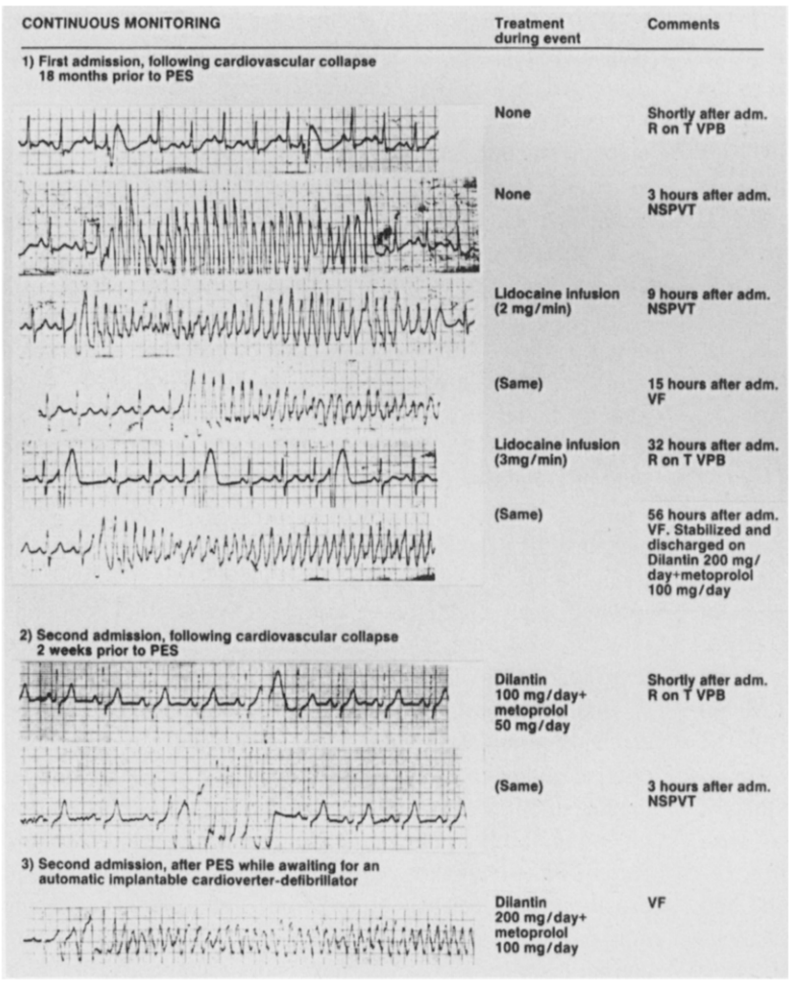

Figure 1. Course of events in Patient 6 as shown on the tracings obtained during continuous monitoring at the time of both admissions (adm). The patient had numerous episodes of R on T ventricular premature beats (VPB), with a short coupling interval (<300 ms), nonsustained polymorphic ventricular tachycardia (NSPVT) (and possibly torsade de pointes) and a total of three in-hospital documented episodes of ventricular fibrillation (VF), all successfully converted to sinus rhythm by defibrillation. HR = heart rate; PES = programmed electrical stimulation; SR = sinus rhythm.

tinuation of antiarrhythmic drug therapy (drug levels at the time of admission for ventricular fibrillation were aprindine, 0.08 μg/ml, and amiodarone, 0.63 μg/ml). The course of events in Patient 6 is depicted in Figure 1, which shows recurrent episodes of polymorphic ventricular tachycardia or ventricular fibrillation during continuous ECG monitoring.

Long-term follow-up after the first documented episode of ventricular fibrillation or ventricular flutter ranged from 30 to 168 months (mean 78) and after programmed electrical stimulation from 12 to 60 months (mean 37). The patient with ventricular flutter was treated with quinidine. Three patients with ventricular fibrillation have been treated with amiodarone alone, and another is receiving amiodarone and a beta-adrenergic blocking agent. Patient 6 has been treated with dilantin, propranolol and an automatic implantable cardioverter-defibrillator; At last follow-up, 18 months after implantation of the device, no shocks had been delivered.

Discussion

This report describes six men without overt heart disease, who were successfully resuscitated from sudden cardiac death. Ventricular fibrillation was documented in five patients and ventricular flutter occurred in the other. A distinctive causative metabolic, electrical or anatomopathologic abnormality could not be found in any of these patients. Although there have been several reports (4–9) of ventricular tachycardia occurring in patients without structural heart disease, there have been only isolated case reports of documented ventricular fibrillation or flutter (8–18) in such patients.

Cause of sudden death. Twenty percent of all cases of sudden cardiac death occur in the absence of coronary artery disease (1). Although numerous causes may be found, the majority of cases are secondary to hypertrophic cardiomyopathy, aortic valve disease and the long QT syndrome, either hereditary or caused by metabolic disturbances or antiarrhythmic drugs (1–3,20–22). Instances of cardiac arrest have also been reported in association with mitral valve prolapse (23,24), the Wolff-Parkinson-White syndrome (25), arrhythmogenic right ventricular dysplasia (26), coronary artery spasm (27–29) and drug exposure. All of our patients had subtle abnormalities during noninvasive or invasive studies, but none of the findings offer a precise explanation of the cause of sudden cardiac death. An underlying myocarditis cannot be excluded in our patients. Reports of

abnormal cardiac histologic findings in patients with malignant ventricular arrhythmias of unknown origin have been described (9,30,31). In one report (9), the only patient with ventricular fibrillation was also the only one to have normal cardiac histologic findings.

Mechanism of ventricular fibrillation. Sustained monomorphic ventricular tachycardia was not seen clinically and could not be induced in any of the patients; this finding suggests that the mechanism of ventricular fibrillation is not reentry (32). Several factors suggest that, in this group of patients, ventricular fibrillation is a primary event: 1) the absence of a sustained ventricular tachycardia before ventricular fibrillation, in contrast to the usual association (33); 2) the clinical observation of only short runs of ventricular tachycardia preceding ventricular fibrillation (Patients 3 and 6); and 3) the inducibility of ventricular fibrillation with three or four extrastimuli during programmed electrical stimulation in four of the six patients (34). Josephson et al. (35) suggested that these factors may produce inhomogeneity in ventricular activation and recovery, as well as shorten ventricular refractoriness, and thus provide the unstable substrate needed for the development of ventricular fibrillation.

Factors contributing to sudden cardiac death. Possible explanations for the occurrence of ventricular fibrillation may be found in three of our patients. Although patients with incessant ventricular tachycardia usually do not develop ventricular fibrillation (7), repetitive runs of ventricular tachycardia (Patient 3), in association with tachycardia-induced left ventricular dysfunction (end-diastolic pressure 28 mm Hg), may favor the development of a malignant rhythm. Patient 6 had ventricular extrasystoles with a short coupling interval (Fig. 1). This has been shown (18,36), although very rarely, to be a precursor to ventricular fibrillation. Finally, Patient 4 has left ventricular hypertrophy, and although hypertrophic cardiomyopathy cannot be firmly established in this patient (37,38), diastolic dysfunction associated with dispersion of refractoriness (39) may favor the occurrence of a malignant arrhythmia.

Role of electrophysiologic studies. The role of programmed electrical stimulation in these patients consists mainly of excluding other abnormalities of atrioventricular conduction and to assess the ability to induce a sustained ventricular tachycardia that could degenerate into ventricular fibrillation. Ventricular fibrillation was induced in four of the six patients; although this may represent a nonspecific response to an aggressive stimulation protocol (36), it is more likely that, in this subgroup of patients with clinical ventricular fibrillation, the inducible arrhythmia reflects underlying electrical abnormalities (34). Three patients underwent programmed electrical stimulation while receiving oral antiarrhythmic drugs. Patient 5 had no inducible arrhythmia, Patient 2 had nonsustained polymorphic ventricular tachycardia and Patient 6 had inducible ventricular fibrillation.

Ventricular fibrillation recurred during antiarrhythmic therapy in only Patient 6 (who was receiving dilantin and a beta-blocking agent); all other patients with ventricular fibrillation were treated with amiodarone. The absence of recurrence of ventricular flutter or ventricular fibrillation in five of the six medically treated patients and the increasing availability of the automatic implantable cardioverter-defibrillator (40) should improve the prognosis of this unique group of patients who have been successfully resuscitated from sudden cardiac death.

We thank Adri van den Dool, RN for assistance in the preparation of this manuscript.

References

1. Lown B. Cardiovascular collapse and sudden cardiac death. In: Braunwald E, ed. Heart Disease: A Textbook of Cardiovascular Medicine. Philadelphia: WB Saunders, 1984:774–806.
2. Maron BG, Roberts WC, McAllister HA, Rosing DR, Epstein SE. Sudden death in young athletes. Circulation 1980;62:218–29.
3. Moss AJ, Schwartz PJ, Crampton RS, Locati E, Carleen E. The long QT syndrome: a prospective international study. Circulation 1985;71:17–21.
4. Froment R, Gallavardin L, Cahen P. Paroxysmal ventricular tachycardia: a clinical classification. Br Heart J 1953;15:172–8.
5. Rowland TW, Schweiger MJ. Repetitive paroxysmal ventricular tachycardia and sudden death in a child. Am J Cardiol 1984;53:1729.
6. Deal BJ, Miller SM, Scagliotti D, Prechel D, Gallastegui JL, Hairman RJ. Ventricular tachycardia in a young population without overt heart disease. Circulation 1986;6:1111–8.
7. Lemery R, Brugada P, Della Bella P, et al. Non-ischemic ventricular tachycardia: clinical course and long term follow-up in patients without clinically overt heart disease. Circulation 1989 (in press).
8. Brodsky MA, Sato DA, Allen BJ, Chesnie BM, Henry WL. Solitary beta-blocker therapy for idiopathic life-threatening ventricular tachyarrhythmias. Chest 1986;89:790–4.
9. Sugrue DD, Holmes DR, Gersh BJ, et al. Cardiac histologic findings in patients with life-threatening ventricular arrhythmias of unknown origin. J Am Coll Cardiol 1984;5:952–7.
10. Ledwich JR, Fay JE. Idiopathic recurrent ventricular fibrillation. Am J Cardiol 1969;24:255–8.
11. Altman KA. Ventricular fibrillation. JAMA 1964;190:781–3.
12. Bechgaard P. Paroxysmal ventricular fibrillation with recovery. Acta Med Scand 1948;132:9–19.
13. Moe T. Morgagni-Adams-Stokes attacks caused by transient recurrent ventricular fibrillation in a patient without apparent organic heart disease. Am Heart J 1949;37:811–9.
14. Storstein O. Adams-Stokes attacks caused by ventricular fibrillation in a man with an otherwise normal heart. Acta Med Scand 1949;133:437–41.
15. Stern TN. Paroxysmal ventricular fibrillation in the absence of other disease. Ann Intern Med 1957;47:552–61.
16. Pistolese M, Richichi G, Catalano V, Staibano M, Berti C. Fibrillazione ventricolare idiopatica ricorrente. Giorn Ital Cardiol 1973;3:433–40.
17. Josephson ME, Spielman SR, Greenspan AM, Horowitz LN. Mechanism of ventricular fibrillation in man. Am J Cardiol 1979;44:623–31.
18. Belhassen B, Shapira I, Shoshani D, Parades A, Miller H, Laniado S. Idiopathic ventricular fibrillation: inducibility and beneficial effects of class I antiarrhythmic agents. Circulation 1987;75:809–16.

19. Brugada P, Green M, Abdollah H, Wellens HJJ. Significance of ventricular arrhythmias initiated by programmed ventricular stimulation: the importance of the type of arrhythmia induced and the number of premature stimuli required. Circulation 1984;69:87–92.

20. Curry P, Stubbs W, Fitchett D, Krikler D. Ventricular arrhythmias and hypokalaemia. Lancet 1976;2:231–3.

21. Koster RW, Wellens HJJ. Quinidine-induced ventricular flutter and fibrillation without digitalis therapy. Am J Cardiol 1976;38:519–23.

22. Velebit V, Podrid P, Lown B, Cohen BH, Graboys TB. Aggravation and provocation of ventricular arrhythmias by antiarrhythmic drugs. Circulation 1982;65:886–94.

23. Wei JY, Bulkley BH, Schaeffer AH, Greene HL, Reid PR. Mitral-valve prolapse syndrome and recurrent ventricular tachyarrhythmias. Ann Intern Med 1978;89:6–9.

24. Pocock WA, Bosman CK, Chesler E, Barlow JB, Edwards JE. Sudden death in primary mitral valve prolapse. Am Heart J 1984;107:378–82.

25. Klein CG, Bachore IM, Sellers TD, Pritchett ELC, Smith WM, Gallagher JJ. Ventricular fibrillation in the Wolff-Parkinson-White syndrome. N Engl J Med 1979;301:1080–5.

26. Marcus FI, Fontaine GH, Guiraudon G, et al. Right ventricular dysplasia: a report of 24 adult cases. Circulation 1982;65:384–98.

27. Levi GF, Proto C. Ventricular fibrillation in the course of Prinzmetal's angina pectoris. Br Heart J 1973;35:601–3.

28. Miller DD, Waters DD, Szlachcic J, Théroux P. Clinical characteristics associated with sudden death in patients with variant angina. Circulation 1982;66:588–92.

29. Crozier I, Stewart D, Ikram H. Sudden death due to painless spasm in near normal coronary arteries. Aust NZ J Med 1986;16:64–5.

30. Strain GE, Irose RM, Factor SM, Fisher GD. Results of endomyocardial biopsy in patients with spontaneous ventricular tachycardia but without apparent structural heart disease. Circulation 1983;68:1171–81.

31. Vignola PA, Aonoma K, Swaye PS, et al. Lymphocytic myocarditis presenting as unexplained ventricular arrhythmias: diagnosis with endomyocardial biopsy and response to immunosuppression. J Am Coll Cardiol 1984;4:812–9.

32. Josephson ME, Horowitz LN, Farshidi A, Kastor JA. Recurrent sustained ventricular tachycardia. 1. Mechanisms. Circulation 1978;57:431–9.

33. Josephson ME, Horowitz LN, Spielman SR, Greenspan AM. Electrophysiologic and hemodynamic studies in patients resuscitated from cardiac arrest. Am J Cardiol 1980;46:948–55.

34. Brugada P, Wellens HJJ. Programmed electrical stimulation of the heart: general principles. In: Josephson ME, Wellens HJJ, eds. Tachycardias: Mechanisms, Diagnosis, Treatment. Philadelphia: Lea & Febiger, 1984: 61–89.

35. Josephson ME, Spielman SR, Greenspan AM, Horowitz LN. Mechanism of ventricular fibrillation in man: observations based on electrode catheter recordings. Am J Cardiol 1979;44:623–31.

36. Coumel P, Leclercq JF, Lucet V. Possible mechanisms of the arrhythmias in the long QT syndrome. Eur Heart J 1985;6(suppl D):D-115–29.

37. Chahine RA, Raizner AE, Ishimori T, Montero AC. Echocardiographic, haemodynamic and angiographic correlations in hypertrophic cardiomyopathy. Br Heart J 1977;39:945–53.

38. Grose R, Maskin C, Spindola-Franco H, Yipintsoi T. Production of left ventricular cavitary obliteration in normal man. Circulation 1981;64:448–55.

39. Franz MR, Bargheer K, Swerdlow CD, Rafflenbeul W. Increased dispersion of action potential duration and local repolarization time in human left ventricular hypertrophy (abstr). Circulation 1986;74(suppl II):II-483.

40. Marchlinski FE, Flores BT, Buxton AE, et al. The automatic implantable cardioverter-defibrillator: efficacy, complications, and device failures. Ann Intern Med 1986;104:481–8.