

Mode of initiation and clinical significance of malignant rapid ventricular arrhythmias

An observational study

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Abstract

The purpose of this study was to explore the modes of initiation and clinical significance of malignant rapid ventricular arrhythmias (MRVAs).

The surface 12-lead electrocardiogram (ECG) or sustained electrocardiogram graph was analyzed in 79 patients. All patients had at least 1 MRVA after being admitted to the hospital.

According to the length of coupling interval of the initial premature ventricular contraction of MRVA, the modes of initiation of MRVA were divided into the following types: those initiated by premature ventricular contraction with short coupling intervals in patients with normal Q-T interval, and for which short-long-short sequences before MRVA precipitation were not observed; those initiated following short-long-short sequences, which were divided into 2 types according to the length of Q-T interval: a normal Q-T interval and a long Q-T interval. On the basis of the different modes of onset, treatments of MRVA were different.

MRVAs have different modes of onset depending on the patients' underlying condition. Prompt recognition of the mode of onset is necessary to facilitate appropriate management. These findings could have important pathophysiologic and clinical implications.

Abbreviations: ECG = electrocardiogram, ICD = implantable cardioverter defibrillator, MRVAs = malignant rapid ventricular arrhythmias, MVT = monomorphic ventricular tachycardia, PVT = polymorphic ventricular tachycardia, VF = ventricular fibrillation.

Keywords: electrocardiography, malignant rapid ventricular arrhythmia, premature ventricular contraction, ventricular fibrillation

1. Introduction

Malignant rapid ventricular arrhythmias (MRVAs) are not uncommon.^[1] MRVA is the primary cause of sudden cardiac death and cardiac syncope.^[2,3] The clinical study of MRVA was always difficult because of its unpredictable occurrence and the short time periods that this disorder is symptomatic.^[4] The use of the Holter monitor, electrocardiogram, and surface electrocardiogram (ECG) recordings enabled clinicians to analyze the mode of initiation of MRVAs.^[5,6] Only a few investigations have described the clinical and electrophysiologic correlations of the onset of MRVAs.^[5,7–11] The aim of the present study was to investigate the electrocardiographic characteristics of the initial premature ventricular contraction of MRVAs and to evaluate the clinical significance and the efficacy of therapeutic measures.

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2. Methods

2.1. Study population

From January 2010 to June 2016 in our hospitals, 79 patients with cardiogenic syncope or sudden death caused by MRVA were treated. The patients were 51 men and 28 women, aged 22 to 82 years (mean, 52.7 ± 19.6 years). All patients had at least 1 MRVA after being admitted to the hospital. Twenty-seven patients were diagnosed with coronary artery disease [9 of these with acute anterior myocardial infarction (including 1 with newly emerging complete right bundle branch block), 2 with acute inferoposterior myocardial infarction, 7 with a previous history of myocardial infarction, 6 with ischemic cardiomyopathy, and 3 with unstable angina], 11 patients with dilated cardiomyopathy, 8 patients with severe brain injury and secondary huge abnormal J wave, 8 patients with idiopathic abnormal J wave, 4 patients with Brugada syndrome, 3 patients with degenerative heart disease, 3 patients with hypertrophic cardiomyopathy, 3 patients with hyperthyroidism, 1 patient with hypertensive heart disease, 1 patient with multifocal cerebral hemorrhage and a huge abnormal J wave, 1 patient with cerebral infarction, 1 patient with chronic renal failure, 1 patient with rheumatic heart disease, 1 patient with polycythemia vera and severe hypokalemia, 1 patient with chlorpromazine poisoning, 1 patient with organophosphorus pesticide poisoning, 1 patient with aconitine poisoning and chronic alcoholism, and 3 patients with unknown conditions (Table 1).

We reviewed the records of the surface ECG, sustained electrocardiogram, and Holter recordings of all 79 patients with MRVA and analyzed the mode, causes, type of onset, and clinical and electrocardiographic characteristics of MRVA, and investigated the efficacy of therapeutic measures.

Table 1
Baseline characteristics of the study population.

Group	ILL (n=30)	NQT-DLL (n=8)	LQT-DLL (n=41)
CHD	10	3	14
DCM	4	2	5
SBI			8
IAJW	6	2	
Brs	3	1	
DHD			3
HCM	3		
HT	1		2
HHD			1
CH			1
CI			1
CRF	1		
RHD			1
PV			1
CP			1
OPP			1
AP			1
UR	2		1

AP=aconitine poisoning and chronic alcoholism, Brs=Brugada syndrome, CH=cerebral hemorrhage, CHD=coronary artery disease, CI=cerebral infarction, CP=chlorpromazine poisoning, CRF=chronic renal failure, DCM=dilated cardiomyopathy, DHD=degenerative heart disease, HCM=hypertrophic cardiomyopathy, HHD=hypertensive heart disease, HT=hyperthyroidism, IAJW=idiopathic abnormal J wave, ILL=Independent of Long Interval group, LQT-DLL=Dependent of Long Interval with Long QT Interval subgroup, NQT-DLL=Dependent of Long Interval with Normal QT Interval subgroup, OPP=organophosphorus pesticide poisoning, PV=polycythemia vera and severe hypokalemia, RHD=rheumatic heart disease, SBI=severe brain injury and secondary huge abnormal J wave, UR=unknown reasons.

2.2. Ethics approval

Ethical approval was obtained from the Ethics Committee of the Hangzhou Xixi Hospital, Hangzhou Cancer Hospital, and the Second Affiliated Hospital of Wenzhou Medical University, and all patients gave informed consent before participation in the study. The study methods were conducted in accordance with approved national and international guidelines.

2.3. Statistical analysis

Results are expressed as mean value \pm standard deviation. SPSS 13.0 software (SPSS Inc., Chicago, IL, USA) was used for statistical analysis. Group *t* test, analysis of variance (ANOVA), and *q* test were used. A value of $P < .05$ was considered significant.

3. Results

3.1. Clinical characteristics of MRVAs

Among the patients in our study, there were 40 cases of cardiac syncope and 39 cases of sudden death; MRVAs were recorded in all patients, as follows: 43 cases of ventricular fibrillation (VF), 17 cases of polymorphic ventricular tachycardia (PVT), 17 cases of torsade de pointes, and 15 cases of monomorphic ventricular tachycardia (MVT). Some of these patients had 2 or more types of these arrhythmias simultaneously or successively.

3.2. Mode of initiation of MRVAs

According to the Q-T interval of the basic rhythm, the coupling interval of premature ventricular contraction initiating the MRVA, and relying or not relying on the long interval ahead of premature ventricular contraction, the modes of onset of

MRVA were divided into the following types: MRVAs in 30 of 79 patients were initiated by the premature ventricular contraction with normal Q-T interval at basic rhythm and were independent on the long interval ahead of the premature ventricular contraction (independent of long interval group). The primary electrocardiographic features were that the Q-T interval of the basic rhythm was normal, the U wave was not obvious, the coupling interval of the premature ventricular contraction initiating the MRVA was usually short, and premature ventricular contraction usually fell on the peak or the descending limb of the preceding T wave (but the coupling interval in a small number of patients was not short, and premature ventricular contraction fell on the end of the T wave, and even appeared R on P). Short-long-short sequences before MRVA (VF, PVT, or MVT) precipitation were not observed (Figs. 1 and 2). MRVAs in 49 of 79 patients were initiated depending on the long interval ahead of the premature ventricular contraction (dependent of Long Interval Group). Short-long-short sequences before MRVA (VF, PVT, or MVT) precipitation were always observed. On the basis of the length of Q-T interval of the basic rhythm, the patients were divided into 2 subgroups: with normal QT interval (dependent of long interval with Normal Q-T Interval Subgroup, 8 cases) (Fig. 3). Except for the difference that the occurrence of MRVAs (VF, PVT, or MVT) was always dependent on the long interval ahead of them, the other features were nearly similar to the Independent of Long Interval group, including the premature ventricular contraction initiating the MRVAs with short coupling interval and falling on the peak or the descending limb of the preceding T wave with long Q-T (U) interval (Dependent of Long Interval with Long Q-T Interval subgroup, 41 cases) (Fig. 4). The electrocardiographic characteristics were that basic heart rate was slow, Q-T interval was significantly prolonged, and U wave (T/U wave integration, illegible) or giant J wave was remarkable. The coupling interval of the premature ventricular contraction initiating MRVAs was often long but with R on T phenomenon. The occurrence of MRVAs (VF, PVT, or MVT) was always dependent on the long interval ahead of them.

3.3. The electrocardiographic characteristics of MRVAs

The electrocardiographic characteristics, including the Q-T interval and the coupling interval, of the premature ventricular contraction initiating MRVAs in the 3 subgroups are summarized in Table 2 and Figs. 1 and 4. The Q-T interval in the Dependent of Long Interval with Long Q-T Interval subgroup was significantly longer than that in the other groups. The coupling interval of the premature ventricular contraction initiating MRVAs in the Dependent of Long Interval with Long Q-T Interval subgroup was significantly longer than that in the other groups. The most common type of MRVA in the Dependent of Long Interval with Long QT Interval subgroup was Torsade de pointes. The most common types of MRVA in the other groups were VF and PVT.

3.4. The cause and induction for MRVA

In our study group of 79 patients with MRVA, 27 patients had coronary heart disease and 14 patients had dilated or hypertrophic cardiomyopathy (Table 1). Coronary heart disease and cardiomyopathy are the most common causes of MRVA. Severe brain injury or cerebral hemorrhage with secondary huge abnormal J wave is a common cause. Brugada syndrome,^[8] hyperthyroidism with severe hypokalemia, and idiopathic abnormal J wave were not rare causes. Cerebral infarction,

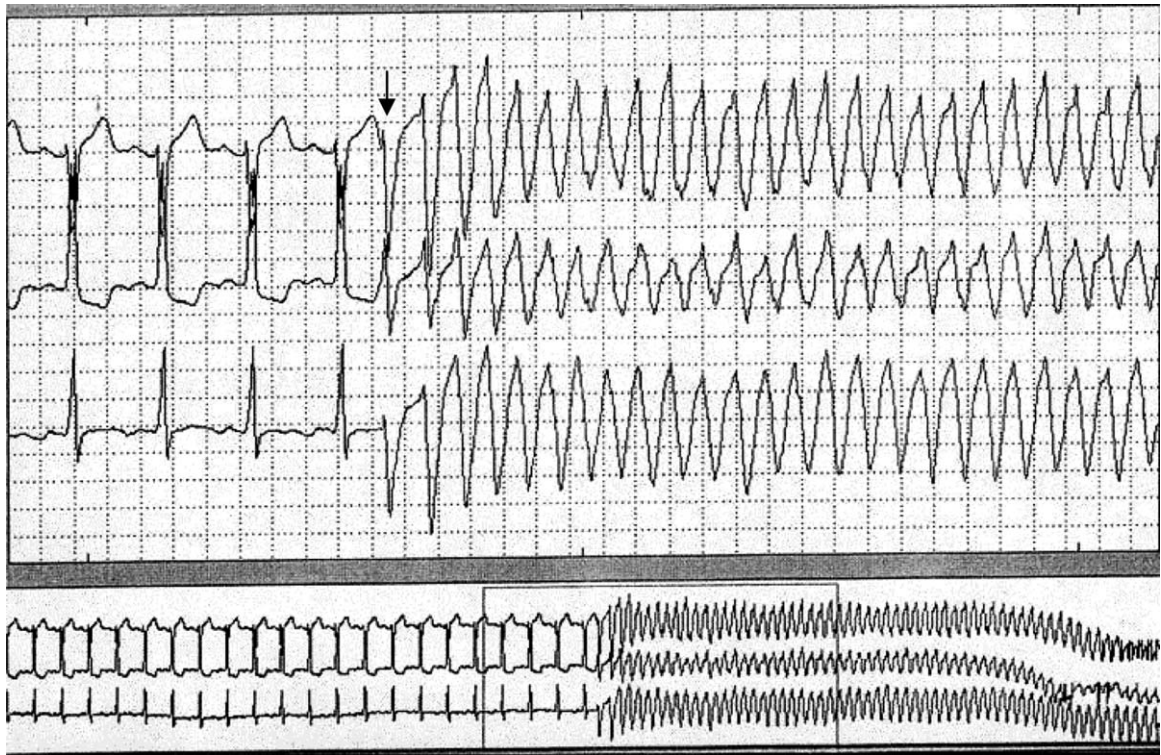


Figure 1. Polymorphic ventricular tachycardia was initiated by premature ventricular contraction (↓) with short coupling interval (300ms), which subsequently degenerated into ventricular fibrillation in a patient with chronic renal failure. The Q-T interval is normally 320ms. Short-long-short sequences before polymorphic ventricular tachycardia precipitation were not observed.

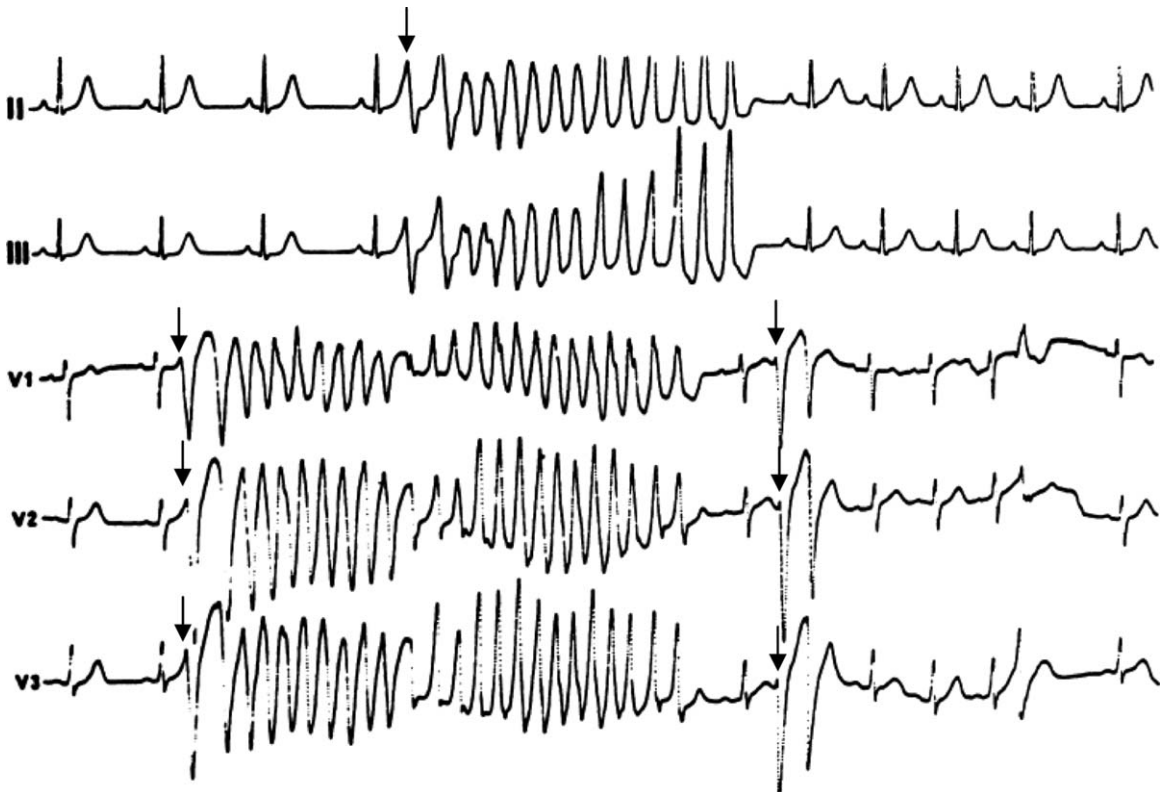


Figure 2. Polymorphic ventricular tachycardia was initiated by premature ventricular contraction (↓) with very short coupling interval (240ms) in a patient with no evidence of ischemic or structural cardiac disease. Each premature ventricular contraction fell on the ascending limb of the preceding sinus T wave. The Q-T interval is normal. Short-long-short sequences before polymorphic ventricular tachycardia precipitation were not observed.

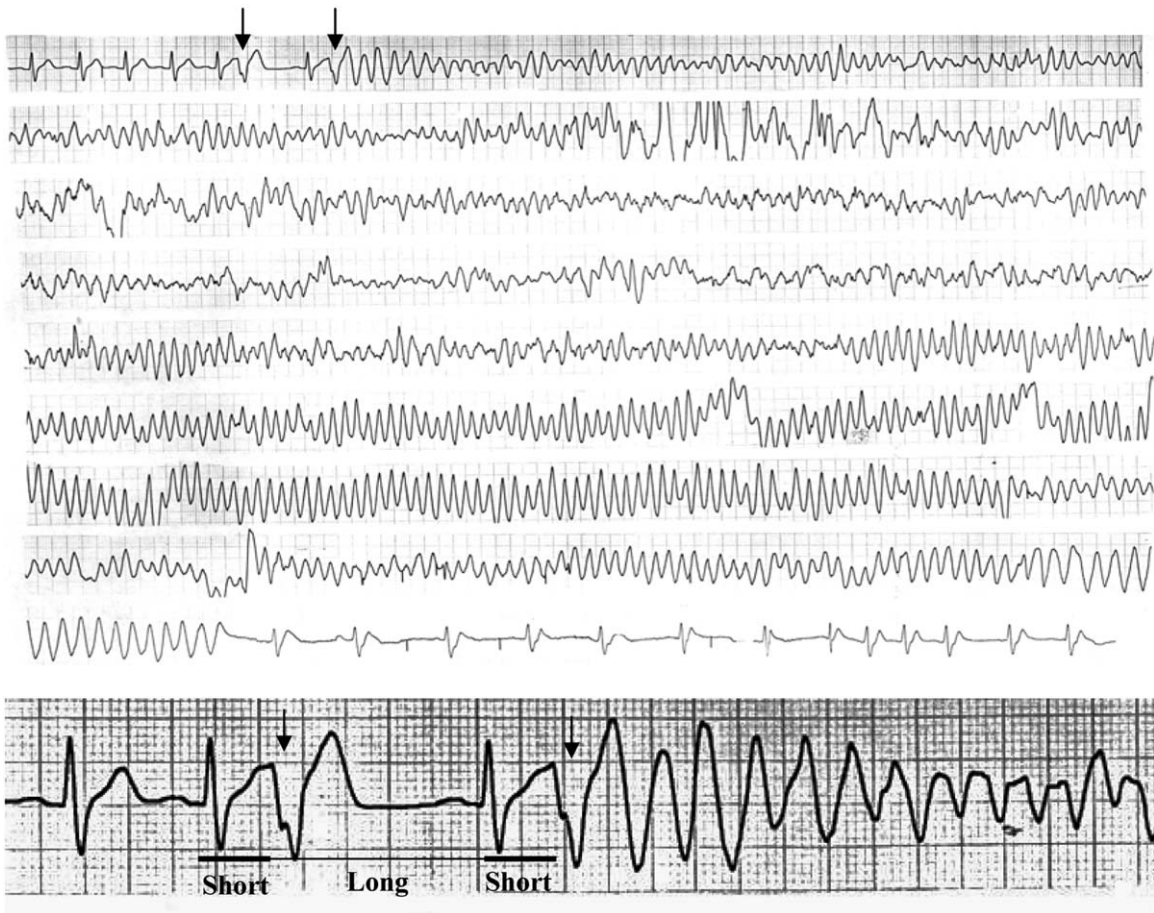


Figure 3. Polymorphic ventricular tachycardia was initiated by premature ventricular contraction (↓) with short coupling interval (300ms), which subsequently degenerated into ventricular fibrillation in a patient with Brugada syndrome. Each premature ventricular contraction fell on the peak of the preceding sinus T wave. Short-long-short sequences (—) before polymorphic ventricular tachycardia precipitation were always observed. The Q-T interval is normal.

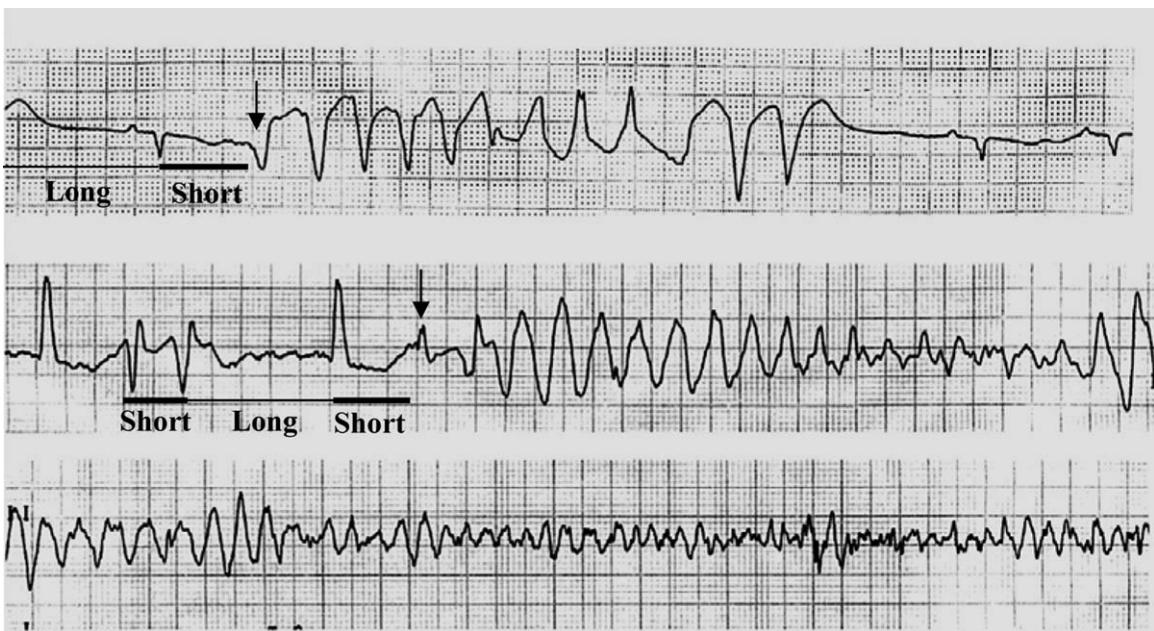


Figure 4. Torsade de pointes was initiated by premature ventricular contraction (↓) with long coupling interval (600ms), which subsequently degenerated into ventricular fibrillation in a patient with coronary heart disease, heart failure, and hypokalemia. Short-long-short sequences (—) before polymorphic ventricular tachycardia precipitation were always observed. The Q-T interval was significantly prolonged (620ms).

Table 2**ECG characteristics of the study population.**

Group	N	Q-T interval	Coupling interval	SLL sequences	MRVA
ILL	30	378 ± 23*	329 ± 14*	No	VF, MVT, PVT
NQT-DLL	8	385 ± 25*	339 ± 13*	Yes	VF, MVT, PVT
LQT-DLL	41	566 ± 64	532 ± 33	Yes	TdP, VF

ILL = Independent of Long Interval group, LQT-DLL = Dependent of Long Interval with Long QT Interval subgroup, MRVA = malignant rapid ventricular arrhythmias, MVT = monomorphic ventricular tachycardia, NQT-DLL = Dependent of Long Interval with Normal QT Interval subgroup, PVT = polymorphic ventricular tachycardia, SLL = sequences, short-long-short sequences, TdP = Torsade de pointes, VF = ventricular fibrillation.

* $P < .05$ vs LQT-DLL.

chronic renal failure, rheumatic heart disease, polycythemia vera with severe hypokalemia, chlorpromazine with organophosphorus pesticide poisoning, aconitine poisoning, and chronic alcoholism were rare causes. Hypokalemia was the primary inducement for patients in the Dependent of Long Interval with Long Q-T Interval subgroup.

4. Discussion

In the present study, we found that the mode of initiation of MRVA was different: those initiated by premature ventricular contraction with short coupling intervals in patients with normal Q-T interval, and for which short-long-short sequences before MRVA precipitation were not observed; those initiated following short-long-short sequences with normal Q-T interval; and those initiated following short-long-short sequences with long Q-T interval. On the basis of the different modes of onset, treatment of MRVA was different.

Sudden cardiac death is the most common cause of death in the United States.^[2,3,12] MRVA, especially VF, is the primary direct cause of sudden cardiac death.^[13] The incidence estimates are approximately 250,000 deaths per year in the US alone.^[12-14] Only a very small proportion of patients who are cardiopulmonarily resuscitated from an out-of-hospital cardiac arrest successfully survive to hospital discharge.^[12,14] Prevention of sudden cardiac death remains a major health care issue. Most of these deaths are related to identifiable causes such as coronary heart disease and cardiomyopathy.^[1,12-14] In agreement with previous studies, we also found that coronary heart disease such as acute myocardial infarction, and cardiomyopathy such as dilated cardiomyopathy and hypertrophic cardiomyopathy, were the primary causes of MRVAs in the present study. Therefore, effective therapies to prevent sudden cardiac death including drugs and devices should be used in at-risk patients.

The prevention of the occurrence of MRVA should adopt the following measures: first, early and timely identification of the high-risk ECG features,^[1] such as abnormal J wave,^[15] premature ventricular contraction with R on T phenomenon,^[10] or long Q-T interval.^[16] Second, underlying heart diseases should be actively treated.^[1] Beta-blockers can reduce sudden death risk in patients with myocardial infarction and heart failure.^[17,18] Angiotensin-converting enzyme inhibitors can counteract ventricular remodeling and reduce sudden cardiac death risk.^[19,20] Third, electrolyte imbalances such as hypokalemia and hypomagnesemia should be corrected early, because they are the most common cause of sudden death or cardiac syncope.^[1,12] Fourth, implantable cardioverter defibrillator (ICD) was the most effective measure to prevent sudden death or cardiac syncope,^[1,2,12] and should be implanted in patients who have survived a life-threatening ventricular arrhythmia, or in patients with heart failure with a left ventricular ejection fraction of 35% or less,^[21] Brugada syndrome,^[22] idiopathic abnormal J wave with a history

of sudden cardiac arrest or cardiac syncope, or a family history of sudden death or cardiac syncope.^[15] Fifth, pacing therapy to maintain fast ventricular rate can prevent RMVA following short-long-short sequences with long Q-T interval in basic rhythm.^[16] Anti-tachycardia pacing treatment may prevent or terminate RMVA caused by premature ventricular contraction independent long interval with normal Q-T interval. In addition, recent research found that the Purkinje network has an important role in the triggering and maintenance of MRVA in patients with and without structural heart disease.^[23-25] Ablation of these triggers may prevent recurrence of future MRVA.^[23-25]

In the present study, we found that MRVA have different modes of onset depending on the patients' underlying condition. MRVAs were initiated by premature ventricular contraction with short coupling intervals and normal Q-T interval in approximately 38.0% of patients, and short-long-short sequences before MRVA precipitation were not observed. MRVAs were initiated following short-long-short sequences with normal Q-T interval in 10.1% of patients. And, MRVAs were initiated following short-long-short sequences with long Q-T interval in 51.9% of patients. Myocardial ischemia, heart failure, and hypokalemia were the most common inducements of MRVAs. On the basis of the different modes of onset, acute treatments of MRVAs were different. First, no matter whether the onset of MRVA was independent or dependent on long interval, patients presenting with normal Q-T interval in basic rhythm should be treated according to the patient's hemodynamic condition. Patients presenting with hemodynamic instability should undergo direct current cardioversion.^[1] If patients are hemodynamically stable, class III (amiodarone) or I (lidocaine, procainamide) antiarrhythmic drugs are suggested for use by current guidelines.^[1] In addition, beta-blockers may be also considered. Amiodarone should be considered first for patients with severe heart failure or acute myocardial infarction.^[1] Second, Torsade de pointes was the most common ventricular arrhythmia following short-long-short sequences with long Q-T interval in basic rhythm, and the arrhythmia repeatedly occurred.^[26] Therefore, direct current cardioversion is not considered a first treatment for the arrhythmia. However, electrical cardioversion should be used for patients with severe hemodynamic instability. The key to prevention of recurrent Torsade de pointes is to rapidly increase heart rate and eliminate the long interval, such as with atrial (ventricular) pacing, isoproterenol infusion, or intravenous atropine to maintain the heart rate at 100 to 120 bpm.^[27] Isoproterenol infusion was banned in patients with coronary artery disease, acute myocardial infarction, or ischemic cardiomyopathy. The arrhythmia was often induced by hypokalemia and/or hypomagnesemia. Therefore, except for increasing heart rate, an infusion of potassium or magnesium should be given. If patients with long Q-T interval in basic rhythm have VT or PVT, acute treatments are similar to those for Torsade de pointes, and class IA, IC, and III antiarrhythmic drugs should not be used

because of the effect of prolonging the Q-T interval.^[28] Calcium channel blocking agents, such as verapamil, have demonstrated efficacy in short-coupled Torsades de pointes.^[29,30] VF or PVT occurring in patients with secondary huge abnormal J wave and long Q-T interval is very serious, and it is difficult to maintain sinus rhythm. The key for the patients with secondary huge abnormal J wave is to treat the primary disease.

In conclusion, MRVAs have distinctive modes of onset depending on the patients' underlying condition. Prompt recognition of the modes of onset of MRVA is necessary to facilitate appropriate management. These findings could have important pathophysiologic and clinical implications.

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References

- Priori SG, Blomström-Lundqvist C, Mazzanti A, et al. 2015 ESC Guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death: the Task Force for the Management of Patients with Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death of the European Society of Cardiology (ESC). Endorsed by: Association for European Paediatric and Congenital Cardiology (AEPIC). *Eur Heart J* 2015;36:2793–867.
- Al-Khatib SM, Yancy CW, Solis P, et al. 2016 AHA/ACC Clinical Performance and Quality Measures for Prevention of Sudden Cardiac Death: a report of the American College of Cardiology/American Heart Association Task Force on Performance Measures. *J Am Coll Cardiol* 2017;69:712–44.
- Zipes DP, Wellens HJJ. Sudden cardiac death. *Circulation* 1998; 98:2334–51.
- Sánchez-Muñoz JJ, García-Alberola A, Martínez-Sánchez J, et al. Same morphology of ventricular premature complexes triggering repeated ventricular fibrillation. *J Interv Card Electrophysiol* 2010;28:95–100.
- Lemmer ME, Majidi M, Krucoff MW, et al. RR-interval irregularity precedes ventricular fibrillation in ST elevation acute myocardial infarction. *Heart Rhythm* 2010;7:65–71.
- Zhang H, Ye H, Huang W. A meshfree method for simulating myocardial electrical activity. *Comput Math Methods Med* 2012; 2012:936243.
- Anderson KP, Shusterman V, Aysin B, et al. Distinctive RR dynamics preceding two modes of onset of spontaneous sustained ventricular tachycardia. (ESVEM) Investigators. *Electrophysiologic Study Versus Electrocardiographic Monitoring*. *J Cardiovasc Electrophysiol* 1999; 10:897–904.
- Roelke M, Garan H, McGovern BA, et al. Analysis of the initiation of spontaneous monomorphic ventricular tachycardia by stored intracardiac electrograms. *J Am Coll Cardiol* 1994;23:117–22.
- Taylor E, Berger R, Hummel JD, et al. Analysis of the pattern of initiation of sustained ventricular arrhythmias in patients with implantable defibrillators. *J Cardiovasc Electrophysiol* 2000;11:719–26.
- Chiladakis JA, Karapanos G, Davlouros P, et al. Significance of R-on-T phenomenon in early ventricular tachyarrhythmia susceptibility after acute myocardial infarction in the thrombolytic era. *Am J Cardiol* 2000;85:289–93.
- Nishizaki M, Arita M, Sakurada H, et al. Polymorphic ventricular tachycardia in patients with vasospastic angina-clinical and electrocardiographic characteristics and long-term outcome. *Jpn Circ J* 2001;65: 519–25.
- Yousuf O, Chrispin J, Tomaselli GF, et al. Clinical management and prevention of sudden cardiac death. *Circ Res* 2015;116:2020–40.
- Stecker EC, Reinier K, Marijon E, et al. Public health burden of sudden cardiac death in the United States. *Circ Arrhythm Electrophysiol* 2014;7:212–7.
- Nichol G, Thomas E, Callaway CW, et al. Regional variation in out-of-hospital cardiac arrest incidence and outcome. *JAMA* 2008;300: 1423–31.
- Yan GX, Antzelevitch C. Cellular basis for the electrocardiographic J wave. *Circulation* 1996;93:372–9.
- Mizusawa Y, Horie M, Wilde AA. Genetic and clinical advances in congenital long QT syndrome. *Circ J* 2014;78:2827–33.
- Dargie HJ. Effect of carvedilol on outcome after myocardial infarction in patients with left-ventricular dysfunction: the CAPRICORN randomized trial. *Lancet* 2001;357:1385–90.
- MERIT-HF Study Group. Effect of metoprolol CR/XL in chronic heart failure: Metoprolol CR/XL Randomised Intervention Trial in Congestive Heart Failure (MERIT-HF). *Lancet* 1999;353:2001–7.
- Køber L, Torp-Pedersen C, Carlsen JE, et al. A clinical trial of the angiotensin-converting-enzyme inhibitor trandolapril in patients with left ventricular dysfunction after myocardial infarction. *Trandolapril Cardiac Evaluation (TRACE) Study Group*. *N Engl J Med* 1995;333:1670–6.
- Pfeffer MA, Braunwald E, Moyé LA, et al. Effect of captopril on mortality and morbidity in patients with left ventricular dysfunction after myocardial infarction. Results of the survival and ventricular enlargement trial. The SAVE Investigators. *N Engl J Med* 1992;327:669–77.
- Ponikowski P, Voors AA, Anker SD, et al. 2016 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure: the Task Force for the diagnosis and treatment of acute and chronic heart failure of the European Society of Cardiology (ESC). Developed with the special contribution of the Heart Failure Association (HFA) of the ESC. *Eur Heart J* 2016;37:2129–200.
- Brugada J, Brugada P. Further characterization of the syndrome of right bundle branch block, ST segment elevation, and sudden cardiac death. *J Cardiovasc Electrophysiol* 1997;8:325–31.
- Haissaguerre M, Shoda M, Jais P, et al. Mapping and ablation of idiopathic ventricular fibrillation. *Circulation* 2002;106:962–7.
- Bansch D, Oyang F, Antz M, et al. Successful catheter ablation of electrical storm after myocardial infarction. *Circulation* 2003;108: 3011–6.
- Marrouche NF, Verma A, Wazni O, et al. Mode of initiation and ablation of ventricular fibrillation storms in patients with ischemic cardiomyopathy. *J Am Coll Cardiol* 2004;43:1715–20.
- Khan IA, Gowda RM. Novel therapeutics for treatment of long-QT syndrome and torsade de pointes. *Int J Cardiol* 2004;95:1–6.
- Barra S, Agarwal S, Begley D, et al. Post-acute management of the acquired long QT syndrome. *Postgrad Med J* 2014;90:348–58.
- Lazzara R. Antiarrhythmic drugs and torsade de pointes. *Eur Heart J* 1993;14(Suppl H):88–92.
- Beach LY, Goldschlager N, Moss JD, et al. Idiopathic ventricular fibrillation in a 29-year-old man. *Circulation* 2017;136:112–4.
- Leenhardt A, Glaser E, Burguera M, et al. Short-coupled variant of torsade de pointes. A new electrocardiographic entity in the spectrum of idiopathic ventricular tachyarrhythmias. *Circulation* 1994;89:206–15.