Effect of Short-term Cardiac Memory on Ventricular Electrical Restitution and QT Intervals in Humans

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Cardiac memory (CM) can alter the configuration of action potentials and the transmural repolarization gradient in ventricular tissue. This study evaluated the effects of CM on ventricular arrhythmogenicity. A total of 20 patients (12 females, 8 males; mean age, 46 ± 13 years) were enrolled. The following indicators were measured to evaluate ventricular arrhythmogenicity: (1) the action potential duration at 90% repolarization (APD_{90}) recorded from the right ventricular apex (RVA); (2) the maximal slope of the action potential duration restitution curve (APDR) constructed by programmed extra stimuli from RVA; and (3) the maximal corrected QT interval (QTc) and QT interval dispersion (QTd). The short-term CM was induced by constant pacing from the RVA at a pacing cycle length (PCL) of 400 ms for 20 minutes. After induction of CM, the mean APD_{90} were significantly shortened at both PCLs of 600 ms and 400 ms (252.9 ± 6.4 ms *vs.* 235.6 ± 6.4 ms and 231.2 ± 6.4 ms *vs.* 214.4 ± 7.3 ms, respectively; $p = 0.001$). No significant change regarding the maximal slopes of APDR were found at both PCLs of 600 ms and 400 ms (1.05 ± 0.09 *vs.* 0.96 ± 0.11 and 0.85 ± 0.08 *vs.* 0.84 ± 0.09, respectively). QTc (417.3 ± 9.1 ms *vs.* 454.7 ± 8.3 ms; $p = 0.001$), but not QTd (63.4 ± 5.4 ms *vs.* 65.7 ± 6.1 ms), was significantly shortened. Short-term CM significantly decreased ventricular APD_{90} and QTc, but did not significantly change the maximal slope of APDR or QTd. These results suggest that CM might not have a significant effect on ventricular arrhythmogenicity.

Key Words: cardiac memory, QT interval, ventricular arrhythmias, ventricular electrical restitution

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Cardiac memory (CM) is characterized by persistent changes in the T wave on electrocardiogram (ECG), which follows the resumption of sinus rhythm after a period of an altered ventricular activation sequence [1,2]. The direction of the change in the T wave vector is the same as that of the vector of the inciting and abnormally activated QRS complex. Changes in the T wave can be induced by both short and long periods of pacing from the right ventricle and can also occur in a variety of clinical settings that follow abnormal ventricular activation, such as left bundle branch block, ventricular pre-excitation, post-tachycardia syndromes and extrasystoles [3–8]. The mechanisms of CM have been widely studied in animal models and in humans. In canines, 4-aminopyridine, which blocks the transient outward current (I_{to}), can abolish the T wave change after a short period of right ventricular

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pacing, which suggests that the kinetics of ion channels may play an important role in CM [7]. In addition, the roles of the L-type calcium current (I_{CaL}), delayed rectifier potassium currents (I_{Kr}, I_{Ks}) and inward rectifier current (I_{K1}) in the modulation of CM expression have been well established in animal studies [9,10]. In our previous human study, the expression of short-term CM was found to be suppressed by verapamil and lidocaine but not by procainamide and nitroglycerin [11]. Previous animal studies have demonstrated that CM could alter the configuration of action potentials and the transmural repolarization gradient in ventricular tissue [9,10,12]. Recently, Jeyaraj et al reported that segmental dispersion of repolarization caused by mechanoelectrical feedback played a significant role in the genesis of T wave memory [13]. All of these reports raise the question of whether the expression of CM would increase ventricular arrhythmogenicity. Because dynamic changes of QT intervals, QT interval dispersion (QTd) and electrical restitution curve have been reported to be correlated with the propensity for ventricular arrhythmias [14–16], we investigated the effect of CM on changes in the QT interval and the ventricular action potential duration restitution curve.

Methods

The study protocol was approved by the institutional review board of our hospital. Informed consent was obtained from all patients.

Patients

The study group consisted of 20 patients, 12 females and eight males (mean age, 46 ± 13 years), with paroxysmal supraventricular tachycardia who were referred for electrophysiologic study and catheter ablation. None of the patients had taken amiodarone, and the other antiarrhythmic drugs were discontinued at least five half-lives before the electrophysiologic study. The electrophysiologic study and catheter ablation were performed in unsedated and postabsorptive states. Thirteen patients had common slow-fast atrioventricular nodal reentrant tachycardia and seven patients had atrioventricular reciprocating tachycardia and used a concealed accessory pathway for retrograde conduction. None of the patients had overt pre-excitation. Before the electrophysiologic study and catheter ablation, all patients underwent routine physical examinations, echocardiography, a 12-lead ECG examination and an exercise treadmill test; no significant organic heart disease was found.

Monophasic action potential duration recording and measurement

After catheter ablation, a 7F monophasic action potential (MAP) recording catheter (Boston Scientific EP Technologies, San Jose, CA, USA) was inserted through the right femoral vein to the right ventricular apex (RVA) for MAP recording and pacing. The catheter position was adjusted under fluoroscopy until a stable catheter position and an acceptable MAP signal was obtained. The MAP electrogram was filtered from 0.05 Hz to 500 Hz. Twelve-lead surface ECG and MAP electrogram were recorded and stored digitally (Bard LabSystem EP Laboratory, Lowell, MA, USA) for retrospective analysis. The monophasic action potential duration was measured at 90% repolarization ($MAPD_{90}$) by a computer caliper on the screen at a speed of 100 mm/s.

Construction of the monophasic action potential duration restitution curve

The $MAPD_{90}$ restitution curve of RVA was determined by a standard S1–S2 protocol at pacing cycle lengths (PCLs) of 600 ms and 400 ms, respectively, as described previously [16]. In brief, a single pulse (S2) was delivered after every eighth basic pulse (S1) by the MAP catheter at the RVA and started with an S1–S2 coupling interval of 500 ms and 380 ms at PCLs of 600 ms and 400 ms, respectively. The S1–S2 interval was decreased in 60-ms steps while the S1–S2 interval was longer than 400 ms. Subsequently, the S1–S2 interval was decreased in 20-ms steps while the S1–S2 interval was longer than 300 ms. When the S1–S2 interval was shorter than 300 ms, the S1–S2 interval was decreased in 10-ms steps until the S2 capture failed. The S1–S2 interval was then increased by 20 ms and decreased in 2-ms steps until the ventricular effective refractory period (ERP) was reached. The $MAPD_{90}$ at PCLs of 600 ms and 400 ms from the RVA for each patient was calculated from the mean value of the last three beats of S1. The MAP duration restitution (MAPDR) curve was constructed by plotting the $MAPD_{90}$ of S2 versus the preceding diastolic interval (DI) at PCLs of 600 ms and 400 ms, respectively. The DI was defined as the S1–S2 interval

minus the $MAPD_{90}$ at the eighth S1. The MAPDR curve obtained by S1–S2 was fit using the monoexponential equation: $y(APD_{90}) = y_0 + A_1 \times (1-e^{-DI/t})$, where A_1 is the free-fitting variable, with each A_1 and t at the correspondent DI, the slope was calculated using the equation: $Slope = (A_1/t) \times [Exp(-DI/t)]$. The slope of the shortest DI was defined as the maximal slope of the MAPDR curve.

Measurement of corrected QT interval and QTd

The QT interval and R–R interval were measured on a computer screen using a digital caliper at a screen rate of 100 mm/s. The QT interval was the interval from the initial Q wave to the end of the T wave. The end of the T wave was: (1) a return to T-P baseline or (2) the nadir of the curve between T and U wave if the U wave was present. When the end of T wave could not be identified, the lead was excluded. The mean of the QT intervals and RR intervals were obtained by measuring the QT intervals and RR intervals of three consecutive sinus beats in each lead. The QTd was the difference between the maximal and minimal mean QT interval calculated from 12-lead ECG. The corrected QT interval (QTc) was calculated by the equation of: maximal mean QT interval/√mean R–R interval.

Induced short-term cardiac memory

The method for inducing short-term CM has been described in our previous study [11]. In brief, sustained pacing was introduced by applying rectangular pulses of 2-ms duration at 2 times the diastolic threshold from RVA by the MAP catheter. The pacing cycle length was 400 ms and ventricular capture for each pacing was confirmed. The pacing duration was 20 minutes.

Study protocol

After 30 minutes of catheter ablation, a baseline 12-lead ECG was recorded to measure the QTc interval and the QTd. The baseline MAPDR curves were constructed from the MAP recordings obtained from the RVA by the S1–S2 pacing protocol at PCLs of 600 ms and 400 ms, respectively. After baseline MAPDR curves were achieved, a short-term CM was induced by constant RVA pacing for 20 minutes. The induction of short-term CM was confirmed by the occurrence of characteristic changes in T wave morphology in each of the 12-lead ECGs after stopping RVA pacing [11]. Immediately after RVA pacing was stopped and short-term CM was presented, the QTc and the QTd were measured and the MAPDR curves were reconstructed by the same S1–S2 pacing protocol as described for the baseline state. To evaluate the effect of short-term CM, all of the MAPDR curve construction protocols were completed within 15 minutes after the induction of short-term CM [11]. The QTc, QTd and $MAPD_{90}$ at ventricular pacing and the maximal slope of MAPDR curves were compared between baseline and after the induction of short-term CM.

Statistical analysis

All data were analyzed using SPSS version 11.0 (SPSS Inc., Chicago, IL, USA). Results are presented as mean ± standard error of the mean. Comparisons of RR interval, QTc, QTd and $MAPD_{90}$ at ventricular pacing and the maximal slope of MAPDR curves before and after the induction of short-term CM were made using Wilcoxon's test. A p value of less than 0.05 was considered statistically significant.

RESULTS

No significant ventricular arrhythmias could be induced during constructing MAPDR curves by extrastimuli protocol (S1–S2) pacing from the RVA at both baseline and after induction of short-term CM. The expression of CM induced by short-term pacing from the RVA is shown in Figure 1. After 20 minutes of pacing from the RVA, the changes in polarity of T waves corresponded to the polarity of the QRS complex induced by pacing from the RVA in individual leads.

$MAPD_{90}$ at ventricular pacing and MAPDR curve

At baseline, before CM was induced, the mean $MAPD_{90}$ at PCLs of 600 ms and 400 ms from the RVA were 252.9 ± 6.4 ms and 231.2 ± 6.4 ms, respectively. After induction of CM, the mean $MAPD_{90}$ at PCLs of 600 ms and 400 ms from the RVA was significantly shortened to 235.6 ± 6.4 ms and 214.4 ± 7.3 ms, respectively ($p = 0.001$). Figure 2 shows a representative example of $MAPD_{90}$ recordings during basic PCLs of 600 ms and 400 ms with serial ventricular extrastimuli at both baseline and after the induction of CM.

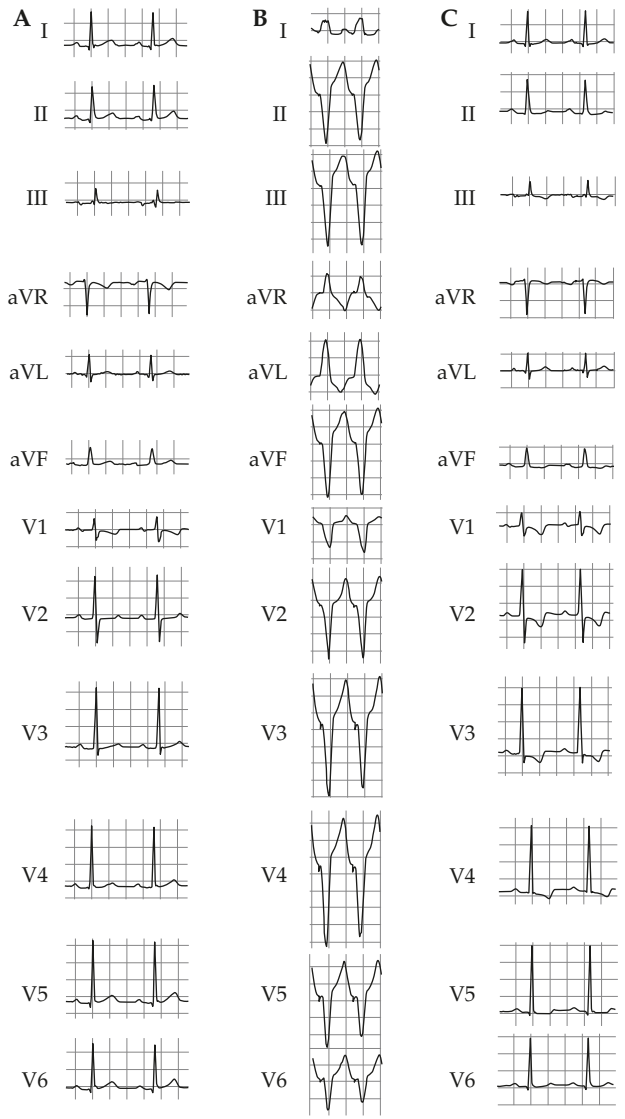

Figure 1. *Twelve-lead ECG: (A) at baseline; (B) during pacing from the right ventricular apex (RVA); (C) after RVA pacing. After 20 minutes of pacing from the RVA, the cardiac memory was expressed as significant changes of T waves on leads II, III, aVF, and V1 to V5 (C) compared with baseline ECG (A).*

When the coupling interval of S1–S2 and consequently the DI was progressively shortened, the $MAPD_{90}$ of S2 was also gradually decreased. The MAPDR curves at PCLs of 600 ms and 400 ms were constructed for each patient. Representative MAPDR curves from one subject constructed at both baseline and after the induction of CM are shown in Figure 3. At baseline, the maximal slopes of the MAPDR curve were more than 1 in 12/20 and 10/20 patients at PCLs of 600 ms and 400 ms, respectively. The mean values of the maximal slope of the MAPDR curves of each patient were 0.96 ± 0.11 and 0.84 ± 0.09 at PCLs of 600 ms and 400 ms, respectively. After the induction of CM, the maximal slopes of MAPDR curve were more than 1 in 13/20 and 11/20 patients at PCLs of 600 ms and 400 ms, respectively. The mean values of the maximal slope of the MAPDR curves of each patient were 1.05 ± 0.09 and 0.85 ± 0.08 at PCLs of 600 ms and 400 ms, respectively. Comparing the mean value of the maximal slope of the MAPDR curves between baseline and after the induction of short-term CM, there were no significant changes at PCLs of 600 ms ($p = 0.64$) and 400 ms ($p = 0.98$), respectively.

QTc and QTd

After the induction of CM, the mean QTc interval (417.3 ± 9.1 ms *vs.* 454.7 ± 8.3 ms; $p = 0.001$) was significantly shortened. However, there were no significant changes in the QTd (63.4 ± 5.4 ms *vs.* 65.7 ± 6.1 ms; $p = 0.5$) or the mean RR intervals (824.1 ± 31.7 ms *vs.* 776.8 ± 34.1 ms; $p = 0.06$) (Figure 4).

Discussion

After the induction of short-term CM, the $MAPD_{90}$ at RVA pacing and the QTc were significantly shortened; however, the QTd and the maximal slopes of the MAPDR curves at PCLs of 600 ms and 400 ms did not change significantly.

The electrical restitution curve was first defined by Bass, who described the time course of recovery of MAPD as a function of the DI between a steady state and an extrastimulus response [17]. The maximal slope of the MAPDR curve constructed by MAP recordings was reported to be related to the propensity of arrhythmogenesis. Steeper slopes of the MAPDR curves have been found in the ventricular endocardium of patients with cardiomyopathy at high risk of ventricular arrhythmias and in atrial endocardium of patients with atrial fibrillation [18,19]. The electrical restitution curve may reflect the sum of various ion channel recovery kinetics and the respective intracellular calcium recycling and recovery of contractile force [20].

Three types of myocardial cells, the epicardial, M and endocardial cells, with distinct electrophysiologic properties were found within the ventricular wall [21]. The M cell has the ability of its action potential to prolong more than that of the epicardium or endocardium with slowing of rate. Previous studies have

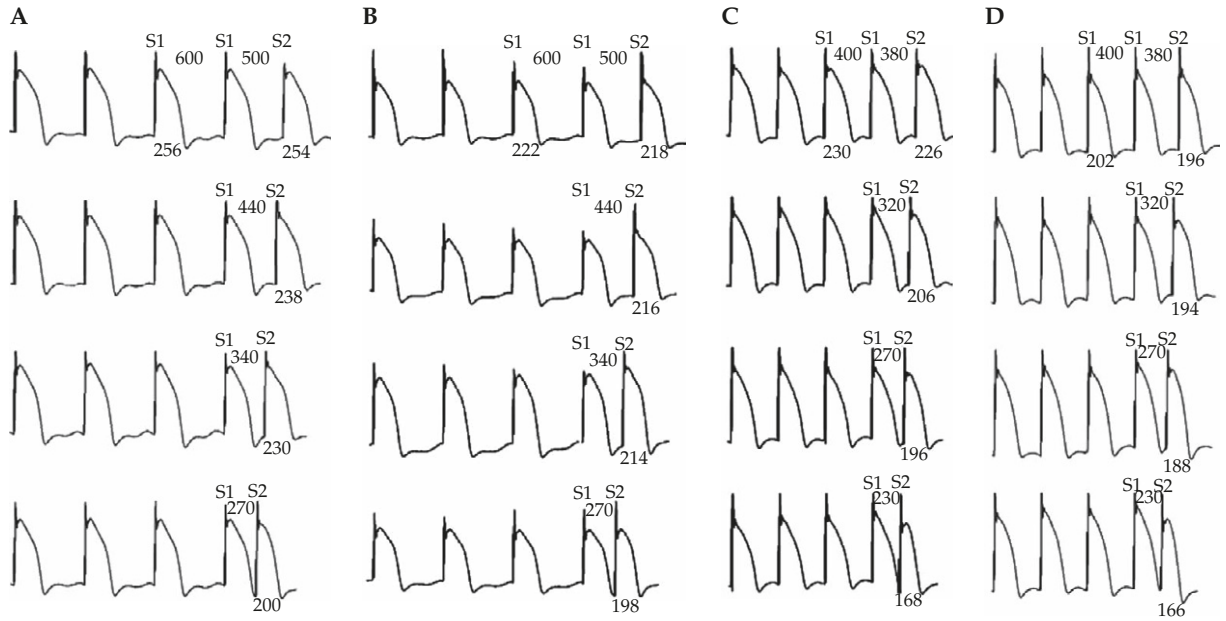

Figure 2. *An example of monophasic action potentials recorded from the right ventricular apex by the S1–S2 protocol at pacing cycle lengths (PCLs) of 600 ms and 400 ms at baseline and after the induction of short-term cardiac memory (CM). (A) At baseline, the monophasic action potential duration at 90% repolarization ($MAPD_{90}$) was 256 ms at the basic PCL (S1–S1) of 600 ms. While the coupling interval of S1–S2 was progressively shortened from 500 ms to 270 ms, the $MAPD_{90}$ of S2 (from 254 ms to 200 ms) was also gradually shortened. (B) After induction of short-term CM, the $MAPD_{90}$ was 222 ms at the basic PCL of 600 ms. While the coupling interval of S1–S2 was progressively shortened from 500 ms to 270 ms, the $MAPD_{90}$ of S2 (from 218 ms to 198 ms) was also gradually shortened. At the basic PCL of 400 ms, the $MAPD_{90}$ was 230 ms and 202 ms at baseline (C) and after induction of short-term CM (D), respectively. While the coupling interval of S1–S2 was progressively shortened from 380 ms to 230 ms, the $MAPD_{90}$ of S2 was also gradually shortened at both baseline and after induction of short-term CM (from 226 ms to 168 ms and from 196 ms to 166 ms, respectively).*

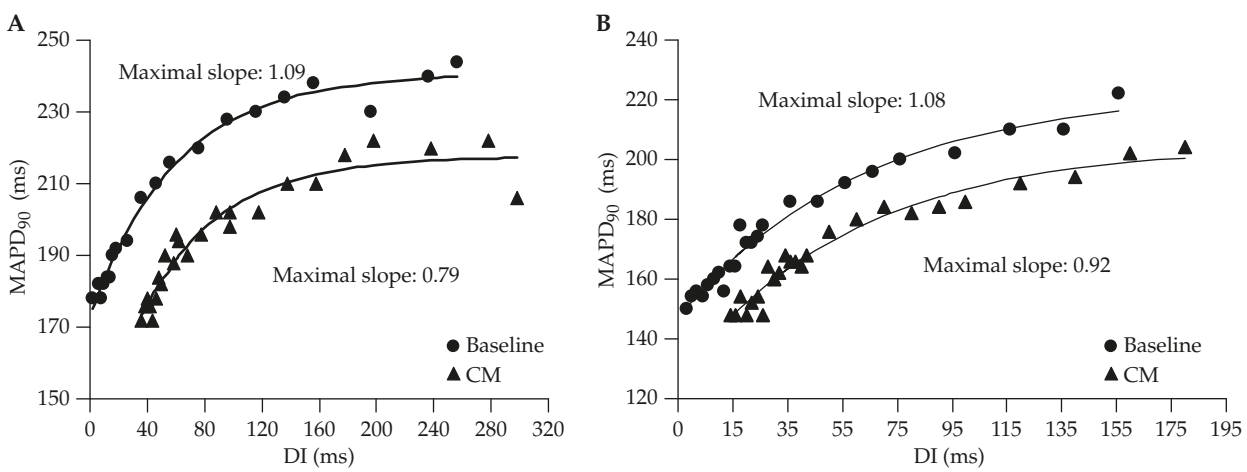

Figure 3. *Representative examples of a monophasic action potential duration restitution (MAPDR) curve at baseline and after the induction of short-term CM at PCLs of: (A) 600 ms; (B) 400 ms. Compared between baseline (circle) and after the induction of CM (triangle), the maximal slopes of MAPDR curves were not significantly changed at the PCLs of 600 ms and 400 ms (1.09 vs. 0.79 and 1.08 vs. 0.92, respectively). $MAPD_{90}$ = monophasic action potential duration at 90% repolarization; DI = diastolic interval.*

defined the transmural voltage gradients responsible for the ECG patterns observed in normal conditions and in patients with a variety of arrhythmogenic cardiomyopathies [22]. The transmural differences in repolarization of the three representative myocardial cell types have been shown to be responsible for the inscription of the J wave and the T wave of ECG [22,23]. The repolarization of the M cell is consistent

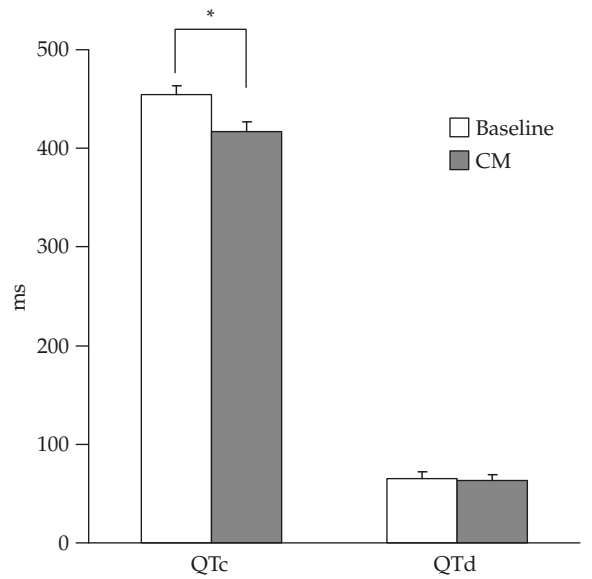

Figure 4. *The maximal mean corrected QT interval (QTc) and mean QT interval dispersion (QTd) at baseline and after the induction of cardiac memory (CM). The mean value of QTc was significantly decreased by induction of CM while the mean value of QTd was not significantly changed. *p < 0.05. Bars represent mean ± standard error of the mean.*

with the end of the T wave and the action potential duration of the M cell determines the QT interval. The prolongation of the QT interval may reflect prolongation of cardiac repolarization. The QTd, which is the interlead difference between the maximal and minimal QT interval calculated in the surface ECG, may represent the degree of repolarization heterogeneity in the ventricular myocardium. Prolongation of the QT interval and QTd could increase the risk of ventricular arrhythmias and cardiac mortality in population-based studies and in patients with cardiovascular disease [15]. In this study, after the induction of short-term CM, the QTc was significantly shortened and the QTd did not change significantly. This finding suggests that short-term CM may not prolong the ventricular repolarization time and may not significantly increase the degree in ventricular repolarization heterogeneity.

The mechanisms of CM are complex. Results from animal studies have suggested that changes in the characteristics of multiple ion channels and angiotensin II synthesis induced by stretching of the myocardial tissue might be involved in the initiation of short-term CM [24]. The effects of CM on ventricular arrhythmogenicity are controversial. Janse et al reported that short-term CM induced by left ventricular epicardial pacing was the result of altering apicobasal repolarization gradients and shortened ventricular repolarization time and action potential duration of ventricular tissue [10]. Goyal et al studied short-term CM in humans and reported that it could shorten the $MAPD_{90}$ of ventricular tissue [25]. However, animal studies from isolated tissue showed that CM could prolong the action potential duration by modulating the expression of ion channels [9]. In our previous study, we demonstrated that, in humans, the QT interval was prolonged after long-term CM and the prolonged QT interval could be shortened by interaction with short-term CM [26]. The controversial results of the effects of CM on action potential duration may result from the different study materials and different pacing protocols, including pacing sites and duration to induce CM. The occurrence of CM in a clinical setting is not uncommon. However, only a few reports on the effect of CM on ventricular arrhythmogenicity are available. Kurita et al reported a patient with implantation of pacemaker who developed torsade de pointes shortly after the loss of pacing [27]. They proposed that the mechanisms responsible for torsade de pointes may involve an increased dispersion of ventricular repolarization, which occurred shortly after the loss of pacing, and the pacing induced CM. In this study, the QTc, the QTd and the slope of the MAPDR curve were used as indicators to evaluate the effects of short-term CM on ventricular arrhythmogenicity. Consistent with the results of previous studies, the effects of short-term CM on the ventricular repolarization properties included shortening of the $MAPD_{90}$ and the QTc. As described above, the slope of the MAPDR curve was considered to be a powerful determinant of the functional behavior of reentrant spiral and scroll waves [16,28]. The steeper slope may promote rotor instability and easily induced ventricular arrhythmias. In this study, the maximal slopes of the MAPDR curve were not significantly changed before and after the induction of short-term CM. In addition, the QTd did not show any significant change. All these results suggest that short-term CM may not significantly affect the reentrant stability and ventricular repolarization homogeneity. This finding may partially explain why ventricular arrhythmias related to CM are rarely reported in clinical practice.

Some limitations in this study should be addressed. First, the MAPDR cure was plotted only from the RVA. Changes in the slope of the MAPDR curve constructed

from other sites of the ventricle might not be equal to that calculated from the RVA. Second, the maximal slope of the MAPDR curve, QTc and QTd were used to evaluate the effects of short-term CM on ventricular arrhythmogenicity in this study. We did not directly use programmed ventricular stimulation to evaluate the effect of short-term CM on ventricular arrhythmogenicity. However, no significant episodes of ventricular arrhythmia were observed during the construction of the MAPDR curve by S1–S2 pacing from the RVA at baseline or after the induction of short-term CM. Third, we only evaluated effects of the short-term CM. Further studies are needed to evaluate whether the effects of long-term CM on ventricular arrhythmogenicity are similar. Fourth, none of our patients had clinically significant organic heart disease, and the results from this study may not apply to patients with organic heart disease. Finally, the number of subjects in this study is small. The lack of a significant effect of short-term CM on QTd and the maximal slope of MAPDR curve may be due to the small number of subjects. Further studies with a larger number of subjects may be needed.

Short-term CM could decrease $MAPD_{90}$ at ventricular pacing and the QTc interval, but did not significantly change the maximal slope of the ventricular MAPDR curve or the QTd. These results suggest that short-term CM may not have a significant effect on ventricular arrhythmogenicity.

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短時間心臟記憶對人心室電氣恢復曲線及 QT 長度的影響

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心臟記憶現象會改變心室組織動作電位形狀及心室組織間的電位差。心臟記憶是否因改變心室組織動作電位形狀，進而引起心室性心律不整，目前並無詳細研究報告。本研究共包含 20 位陣發性上心室頻脈病人，於接受心電氣生理燒灼術後，測量 QT 長度，計算 QT 差異 (QTd)，並且分別在 600 ms 及 400 ms 基礎刺激下，繪出心室電氣恢復曲線，並計算其曲線之最大斜率。以 400 ms 間隔，連續於右心室尖刺激 20 分鐘，誘發短暫心臟記憶後，馬上再度測量 QT 長度，計算 QTd 及心室電氣恢復曲線之最大斜率。比較心臟記憶誘發前後，QTc 長度，QTd 及心室電氣恢復曲線之最大斜率。結果發現心臟記憶誘發後，心室電氣恢復曲線之最大斜率及 QTd 並無明顯變化，然而，QTc 長度明顯變短。由以上結果推論，心臟記憶對誘發心室性心律不整並無明顯影響。

關鍵詞：心臟記憶，QT 長度，心室性心律不整，心室電氣恢復曲線
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