Time-dependent effect of cardiac resynchronization therapy on ventricular repolarization and ventricular arrhythmias


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Aims
Cardiac resynchronization therapy (CRT) improves the clinical status of patients with congestive heart failure, although left ventricular epicardial pacing may increase transmural dispersion of repolarization (TDR). The aim of this study was to investigate the time-dependent effect of CRT on ventricular repolarization and ventricular arrhythmia at mid-term follow-up.

Methods and results
The study group consisted of 84 patients treated with CRT. Twelve-lead electrocardiogram was digitally recorded and Tpeak-to-Tend interval (Tp–e) was measured at baseline, 1 week, 1 month, and 3, 6, and 12 months after device implantation. We determined the time-dependent changes in Tp–e, ventricular tachycardia and ventricular fibrillation (VT/VF) during 12 months of follow-up, in both CRT responders and non-responders. Seventeen of 84 patients (20%) had VT/VF during first year. Six of those 17 patients (35%) experienced VT/VF within 1 month of implantation and diminished over time. Tp–e decreased significantly at 6 and 12 months after implantation compared with 1 week (108 ± 14 ms at 1 week vs. 97 ± 21 ms at 6 months (P = 0.03) and 95 ± 19 ms at 12 months (P = 0.01)). Responders demonstrated a greater time-dependent reduction of Tp–e at 6 and 12 months of CRT and had a lower rate of VT/VF compared with non-responders (log-rank test, P = 0.004).

Conclusion
Transmural dispersion of repolarization and the number of patients with VT/VF decreased over time after CRT. Patients with reverse remodelling demonstrated a lower rate of VT/VF and a greater time-dependent reduction of TDR.

Keywords
Cardiac resynchronization therapy • Transmural dispersion of repolarization • Tp–e interval • Ventricular arrhythmia • Responder

Introduction
Cardiac resynchronization therapy (CRT) has emerged as an effective treatment strategy for patients with advanced drug refractory heart failure (HF), left ventricular (LV) systolic dysfunction, and ventricular dyssynchrony.1,2 Cardiac resynchronization therapy has been shown to reduce hospital stay, morbidity, and mortality risk especially in patients implanted with a CRT-defibrillator (CRT-D).3,4 In the extension phase of CARE-HF (Cardiac Resynchronization-Heart Failure) trial, a reduction in sudden cardiac death as well as death due to worsening HF was observed.5 However, CRT alone had no effect on the frequency of ventricular arrhythmias (VAs) in patients who underwent upgrade from an implantable cardioverter-defibrillator (ICD) to CRT-D.6 Some studies have shown that CRT can potentially promote ventricular tachyarrhythmias. Biventricular pacing was shown to increase transmural dispersion of repolarization (TDR), which may be associated with prolongation of the Tpeak-to-Tend interval (Tp–e).7,8 However, patients demonstrating reverse remodelling in response to CRT show a reduction in VAs after device implantation.9–13 Limited data are available on the

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effects of CRT on dispersion of ventricular repolarization and VAs during mid-term follow-up. Therefore, the aim of this study was to investigate the time-dependent effect of CRT on ventricular repolarization and arrhythmias at mid-term follow-up.

Methods

Patients

We included 88 consecutive patients who had a CRT-D implanted at our centre from September 2006 to August 2011. Indications for CRT were severe HF [New York Heart Association (NYHA) functional class III or IV] despite optimal medical therapy, left ventricular ejection fraction (LVEF) ≤ 35%, and QRS duration ≥ 130 ms. All patients were receiving stable medical therapy including beta-blockers, angiotensin-converting enzyme inhibitors or angiotensin receptor blockers, diuretics, spironolactone, and amiodarone. We excluded four patients who met the following criteria: (i) a recent myocardial infarction or coronary revascularization (<3 months) or scheduled revascularization, (ii) inadequate data on arrhythmic events and device-stored electrograms during follow-up. As a result, 84 patients treated with a CRT-D device were enrolled in this study. All patients gave written informed consent.

Device implantation

All devices were triple-chamber defibrillators capable of providing CRT and detecting and treating ventricular tachyarrhythmias. The detection rate cutoff of ventricular tachycardia (VT) was at least 350 ms (171 b.p.m.) to avoid under-reporting of arrhythmic episodes by the implanted device. The right atrial lead was positioned in the right atrial appendage, and the RV lead in the RV apex. The LV lead was implanted transvenously via the coronary sinus tributaries and placed to stimulate the lateral or posterolateral LV wall. Twenty-seven patients with complete atrioventricular block underwent an upgrade from a pacemaker or ICD to a CRT-D. All leads were connected to a triple-chamber CRT-D device (InSync Marquis CRT-D, Concerto, Consulta CRT-D, Protecta XT CRT-D, Medtronic Inc.; Atlas+ HF, Promote RF, Unify; RENEWAL 4 HE, Boston Scientific). Cardiac resynchronization therapy-defibrillator implantation was successful in all patients and all patients underwent CRT optimization of the atrioventricular and interventricular interval and interventricular interval using pulsed-wave Doppler echocardiography at 1 week after device implantation.

All patients were seen at our outpatient centre for routine follow-up visits at 1, 3, 6, and 12 months after CRT device implantation. Clinical evaluation, 12-lead ECG, and device interrogation were performed at each follow-up visit. All patients underwent echocardiography after 3, 6, and 12 months of CRT.

Electrocardiography assessment

Twelve-lead ECGs were digitally recorded at baseline, 1 week, 1 month, 3 months, 6 months, and 12 months after CRT device implantation. Each lead was magnified and analysed by two independent electrophysiologists blinded to all other patient data. Tpeak-to-Tend interval was measured in each lead and averaged for all 12 leads. In all leads, Tp–e was measured from the peak of the T-wave to the end of the T-wave, defined as the point at which a tangent to the maximal downslope of the descending limb of the T-wave crossed the isoelectric baseline. In the case of negative or biphasic T-waves, the QT peak was measured to the nadir of the T-wave. If a U-wave was present, only the T-wave was considered for measurement. The time-dependent change of Tp–e was evaluated, using Tp–e at 1 week after CRT as a reference.

Evaluation of appropriate implantable cardioverter-defibrillator therapy

To assess the occurrence of VA, we recorded the number of patients with VA episodes and the number of VA episodes in each patient that occurred during 1 year of CRT. Ventricular arrhythmia episodes in this study were all spontaneous ventricular tachyarrhythmias detected by the implanted device and subsequently validated by two electrophysiologists, who were blinded on patient follow-up data. Ventricular arrhythmia episodes were classified as sustained VT [requiring antitachycardia pacing or shock therapy, excluding ventricular fibrillation (VF)] or VF.

Furthermore, we investigated the relationship between the effect of CRT on ventricular repolarization and the occurrence of VT/VF episodes. Moreover, a positive response to CRT was defined as a relative change in LVESV ≥ 15% after 6 months of CRT. Patients were classified into responders or non-responders. Ventricular tachycardia/VF episodes and time-dependent changes in Tp–e were evaluated in both of these groups.

Statistical analysis

Continuous data were expressed as mean ± standard deviation. Categorical data were expressed as numbers or percentages. Continuous variables were compared between groups using a Mann–Whitney U test. Categorical variables were compared using Fisher’s exact test or χ² analysis, as appropriate. Time-dependent change in Tp–e was compared using one-way analysis of variance (ANOVA), followed by the Bonferroni test for multiple comparisons when the ANOVA showed a significant difference. The time to first VT/VF episodes was plotted according to the Kaplan–Meier method, and event-free survival curves were compared between groups by a log-rank test. Univariate and multivariate Cox proportional hazards models were performed to detect predictors of VT/VF. Variables with P < 0.1 on univariate analysis were retained in the multivariate model. All tests were two-tailed, and a P < 0.05 was considered significant for all tests. All analyses were performed using the SPSS 20.0 software package (SPSS Inc.).
Results

Patients
The baseline clinical characteristics of the patients are described in Table 1. Eighteen patients received CRT-D for secondary prevention of sudden cardiac death. Four patients were cardiac arrest survivor due to VF and 14 patients had a history of VT. All patients were on maximum tolerated medical therapy for congestive HF and the therapy remained stable during follow-up.

At 6 months after CRT, 49 patients (59%) were classified as responders based on reverse LV remodelling. The baseline characteristics were similar between the responder group and the non-responder group. However, in the responder group, there were more patients with left bundle branch block (LBBB) and fewer patients with right bundle branch block (RBBB) compared with those in the non-responder group. The distribution of the LV lead placement was as follows: lateral wall, 50 patients; posterolateral wall, 21 patients; anterolateral wall, 12 patients; posterior wall, 1 patient. The LV lead position was in the lateral or posterolateral wall in 40 responders (82%) and 31 non-responders (89%, P = 0.43). No significant changes in VT and VF detection zone were observed between responders and non-responders (VT: 159 ± 13 vs. 155 ± 11 b.p.m., P = 0.19; VF: 192 ± 8 vs. 200 ± 18 b.p.m., P = 0.16).

At follow-up, responders showed a significant increase in LVEF [from 25.2 ± 8.0 to 37.9 ± 10.4% at 3 months, to 40.1 ± 11.3% at 6 months, and 44.0 ± 11.0% at 1 year follow-up (P < 0.001), respectively] and a decrease in LVESV [from 132 ± 58 to 88 ± 57 mL at 3 months, 81 ± 53 mL at 6 months, 74 ± 48 mL at 1 year follow-up (P < 0.001), respectively]. However, non-responders showed no significant changes in LVEF and LVESV during follow-up (LVEF: from 27.9 ± 9.9 to 30.6 ± 9.6% at 3 months, to 29.7 ± 10.8% at 6 months, to 31.2 ± 11.2% at 1 year follow-up, ANOVA P = 0.13; LVESV: from 131 ± 63 to 138 ± 73 mL at 3 months, to 137 ± 68 mL at 6 months, to 137 ± 72 mL at 1 year follow-up, ANOVA P = 0.46).

Distribution of patients with ventricular arrhythmias during follow-up
A total of 176 appropriate VA episodes occurred within 1 year after CRT device implantation. These episodes consisted of 6 VF and 170 VT. Antitachycardia pacing therapies were successful in terminating

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Total (n = 84)</th>
<th>Responder (n = 49)</th>
<th>Non-responder (n = 35)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male gender</td>
<td>61 (73)</td>
<td>33 (67)</td>
<td>28 (80)</td>
<td>0.20</td>
</tr>
<tr>
<td>Age (years)</td>
<td>67.5 ± 11.5</td>
<td>66.3 ± 11.6</td>
<td>69.3 ± 10.9</td>
<td>0.17</td>
</tr>
<tr>
<td>Ischaemic cardiomyopathy</td>
<td>25 (30)</td>
<td>12 (25)</td>
<td>13 (37)</td>
<td>0.21</td>
</tr>
<tr>
<td>Secondary prevention</td>
<td>18 (21)</td>
<td>8 (16)</td>
<td>10 (29)</td>
<td>0.18</td>
</tr>
<tr>
<td>NYHA class</td>
<td>3.1 ± 0.6</td>
<td>3.0 ± 0.6</td>
<td>3.1 ± 0.7</td>
<td>0.55</td>
</tr>
<tr>
<td>AF</td>
<td>16 (20)</td>
<td>7 (14)</td>
<td>9 (26)</td>
<td>0.19</td>
</tr>
<tr>
<td>QRS duration (ms)</td>
<td>170 ± 30</td>
<td>172 ± 31</td>
<td>166 ± 30</td>
<td>0.30</td>
</tr>
<tr>
<td>Conduction disturbance</td>
<td></td>
<td></td>
<td></td>
<td>0.008*</td>
</tr>
<tr>
<td>LBBB</td>
<td>43 (51)</td>
<td>32 (65)</td>
<td>11 (31)</td>
<td></td>
</tr>
<tr>
<td>RBBB</td>
<td>8 (10)</td>
<td>1 (2)</td>
<td>7 (20)</td>
<td></td>
</tr>
<tr>
<td>IVCD</td>
<td>6 (7)</td>
<td>2 (4)</td>
<td>4 (11)</td>
<td></td>
</tr>
<tr>
<td>Right ventricular pacing</td>
<td>27 (32)</td>
<td>15 (31)</td>
<td>12 (34)</td>
<td></td>
</tr>
<tr>
<td>LVEF (%)</td>
<td>26.3 ± 8.8</td>
<td>25.2 ± 8.0</td>
<td>27.9 ± 9.9</td>
<td>0.33</td>
</tr>
<tr>
<td>LVEDV (ml)</td>
<td>175 ± 68</td>
<td>173 ± 64</td>
<td>178 ± 75</td>
<td>0.89</td>
</tr>
<tr>
<td>LVESV (ml)</td>
<td>132 ± 60</td>
<td>132 ± 58</td>
<td>131 ± 63</td>
<td>0.90</td>
</tr>
<tr>
<td>IVMD (ms)</td>
<td>44.8 ± 22.7</td>
<td>52.4 ± 21.6</td>
<td>25.8 ± 14.3</td>
<td>0.001*</td>
</tr>
<tr>
<td>β-blocker</td>
<td>72 (86)</td>
<td>44 (90)</td>
<td>28 (81)</td>
<td>0.21</td>
</tr>
<tr>
<td>ACE inhibitors or ARB</td>
<td>71 (85)</td>
<td>42 (86)</td>
<td>29 (83)</td>
<td>0.72</td>
</tr>
<tr>
<td>Diuretics</td>
<td>64 (76)</td>
<td>35 (71)</td>
<td>29 (83)</td>
<td>0.23</td>
</tr>
<tr>
<td>Spironolactone</td>
<td>52 (62)</td>
<td>28 (57)</td>
<td>24 (72)</td>
<td>0.29</td>
</tr>
<tr>
<td>Amiodarone</td>
<td>18 (21)</td>
<td>7 (14)</td>
<td>11 (31)</td>
<td>0.08</td>
</tr>
<tr>
<td>VT detection zone (b.p.m)</td>
<td>157 ± 13</td>
<td>159 ± 13</td>
<td>155 ± 11</td>
<td>0.19</td>
</tr>
<tr>
<td>VF detection zone (b.p.m)</td>
<td>195 ± 14</td>
<td>192 ± 8</td>
<td>200 ± 18</td>
<td>0.16</td>
</tr>
</tbody>
</table>

NYHA, New York Heart Association; AF, atrial fibrillation; LBBB, left bundle branch block; RBBB, right bundle branch block; IVCD, non-specific intraventricular conduction disturbances; LVEF, left ventricular ejection fraction; LVEDV, left ventricular end-diastolic volume; LVESV, left ventricular end-systolic volume; IVMD, interventricular mechanical delay; MR, mitral regurgitation; ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blockers.

Values are presented as n (%) or mean ± SD.

*P < 0.05, responder vs. non-responder.
153 VT episodes, and shock therapies were necessary for 17 VT episodes and 6 VF episodes.

Seventeen of 84 patients (20%) had at least one VA episode during a year (VF episodes, 4 patients; VT episodes, 13 patients). There was no patient who experienced both VT and VF episodes during follow-up. Moreover, 6 of those 17 patients (35%) experienced VT or VF episodes within the first month of CRT. Beyond the first month, there was a decrease in the number of patients with VA episodes over time (Figure 1). Ventricular tachycardia/VF episodes were observed in 13 primary prevention patients (20%) and 4 secondary prevention patients (22%, P = 0.81).

**Relationship between ventricular arrhythmias and Tpeak-to-Tend interval**

Tpeak-to-Tend interval decreased over time after CRT device implantation, although there was a slight increase in Tp–e at 1 week after device implantation. The increase in Tp–e coincided with a period of high rate of VT/VF episodes. Tpeak-to-Tend interval decreased significantly at 6 and 12 months after CRT implantation compared with 1 week [108 ± 14 ms at 1 week vs. 97 ± 21 ms at 6 months (P = 0.03) and 95 ± 19 ms at 12 months (P = 0.01), respectively]. No significant difference between patients with and without VT/VF episodes was observed in Tp–e before CRT (106 ± 15 vs. 108 ± 14 ms, P = 0.39), but Tp–e after 1 week after CRT was greater in patients with VT/VF episodes than in patients without VT/VF episodes (105 ± 20 vs. 119 ± 16 ms, P = 0.02). On multivariate Cox regression analysis, Tp–e at 1 week after CRT and IVMD before CRT were independently associated with the risk of VT/VF [hazard ratio (HR): 1.058; P = 0.002 and HR: 0.947; P = 0.002, respectively; Table 2].

**Comparison of responders and non-responders**

During 1 year of follow-up, 6 of 49 (12%) responders experienced VT/VF episodes, compared with 11 of 35 (31%) non-responders (P = 0.03). Kaplan–Meier event-free survival analysis demonstrated that the responder group had a significantly lower rate of VT/VF episodes, compared with the non-responder group (log-rank test, P = 0.004; Figure 2).

There was a trend for Tp–e to decrease over time in both groups, and Tp–e in the responder group was lower than that in the non-responder group at all time points (Figure 3). Tpeak-to-Tend interval in responders was significantly lower than that in non-responders at 3-month, 6-month, and 12-month follow-up [108 ± 14 vs. 94 ± 16 ms at 3 months (P = 0.02), 105 ± 15 vs. 92 ± 16 ms at 6 months (P = 0.03), 102 ± 17 vs. 90 ± 16 ms at 12 months (P = 0.005)]. In the responder group, there was a significant reduction of Tp–e at 6 and 12 months after device implantation compared with 1 week [105 ± 13 ms at 1 week vs. 92 ± 16 ms at 6 months (P = 0.04), 90 ± 16 ms at 12 months (P = 0.02), respectively]. However, no significant temporal change was observed in the non-responder group.

**Discussion**

To our knowledge, this is the first study to assess the time-dependent effect of CRT on Tp–e and VAs. The major findings of the present study were: (i) the largest number of patients experienced VT/VF episodes within 1 month after CRT device implantation, and there was a decrease in number of patients with VT/VF episodes over time; (ii) CRT was associated with a time-dependent reduction of Tp–e at 6 and 12 months after device implantation; and (iii) the responder group demonstrated a greater time-dependent reduction of Tp–e at 6 and 12 months of CRT and a lower rate of VT/VF episodes compared with the non-responder group.

The baseline characteristics were similar between responders and non-responders in our study. However, there were more patients with LBBB, fewer patients with RBBB, and the IVMD was longer in the responder group. This finding supports previous results showing larger improvements after CRT in patients with LBBB and LV dyssynchrony. 15,16
Effect of cardiac resynchronization therapy on the occurrence of ventricular tachycardia/ventricular fibrillation episodes

In our study, the largest number of patients experienced VT/VF episodes within 1 month after CRT device implantation. In patients receiving an upgrade from an ICD to a CRT-D, the frequency of VA requiring appropriate ICD therapy did not significantly change before and after CRT. Furthermore, in our study, there were no differences in VT/VF between patients with primary and secondary prevention.

Several mechanisms have been proposed for the proarrhythmic effects of CRT based on experimental studies. Reversal of the normal activation sequence by epicardial pacing is one of the major underlying mechanisms and has been shown to cause prolongation of the QT interval and TDR, creating a substrate and trigger for reentrant arrhythmias. In addition, some studies have shown that CRT can potentially promote ventricular tachyarrhythmias within the first hours or days after the initiation of biventricular pacing.
In contrast, recent studies reported that patients with a positive response to CRT exhibited a significant reduction in VT/VF episodes, and non-responders had a significant increase in VT/VF episodes. Furthermore, patients with LBBB treated with CRT-D, experience fewer and later VT/VF episodes compared with matched controls without LBBB treated with an ICD. In our study, the number of patients with VT/VF episodes decreased over time, and the responder group had a significantly lower rate of VT/VF episodes, compared with the non-responder group. Gold et al. showed that the antiarrhythmic effect of CRT could be explained by induction of favourable LV reverse remodelling and decreased myocardial wall tension and electrical stabilization of myocyte membranes.

Effect of cardiac resynchronization therapy on ventricular repolarization
Reversal of the direction of the LV wall activation during biventricular pacing is accompanied by a prominent increase in Tp–e as a non-invasive index of TDR. Previous studies have reported both an increase and a decrease in Tp–e after CRT device implantation. Lellouche et al. demonstrated that CRT decreased Tp–e in responders, and this group had a lower rate of appropriate ICD therapy. In our study, there was a decrease in the number of patients with VT/VF episodes as well as a reduction in Tp–e over time after device implantation, but a significant reduction of Tp–e was observed only in responders. In addition, Tp–e at 1 week after implantation was associated with an increased risk of VT/VF. The early increase of Tp–e might be associated with high rate of VT/VF episodes in the early phase after CRT. Previous studies demonstrated that Tp–e measured within 24 h after CRT-D implantation was the only independent predictor of ICD therapy after 1-year follow-up. Unfortunately, the results of Tp–e in our study and previous studies do not solve the issue of whether CRT pacing alone without ICD therapy is sufficient to shorten the Tp–e and reduce the risk of VA. However, we found that the time-dependent reduction of Tp–e and the decrease in the number of patients with VT/VF episodes over time was associated with a positive response to CRT.

In this study, a significant improvement in echocardiographic parameters was observed in the responder group beyond 3 months of CRT. Responders showed a significant decrease of Tp–e significantly at 6 months and 12 months after CRT, but non-responder showed no significant changes over time. In addition, Tp–e in responders was significantly lower than that in non-responders at 3-month, 6-month, and 12-month follow-up. Therefore, the change of Tp–e might be dependent on the mechanical reverse remodelling. Several experimental studies have shown the effect of CRT on dysynchronous HF. First, CRT generates homogenous stress kinases and reduced apoptosis. Secondly, CRT corrected the alterations in gene expression induced by electromechanical dysynchrony. Thirdly, CRT restores ion channel function and abnormal Ca$^{2+}$ homeostasis and attenuates the regional heterogeneity of action potential duration. These various effects of CRT might lead to a reduction of VA and an improvement of systolic function. The mechanism of the time-dependent reduction in Tp–e and VA episodes in patients with reverse remodelling requires further investigation.

Limitations
This study has several important limitations. Since it was a retrospective cohort study, device programming and use of antiarrhythmic drug therapy could not be controlled. However, device programming and use of antiarrhythmic drug therapy was similar between the two subgroups and remained constant during the study period. We compared Tp–e at each time with Tp–e at 1 week after CRT, because the slight increase in Tp–e at 1 week after CRT may be induced by ventricular stimulation itself. This study was further limited by the small sample size and the effect of CRT on VT/VF and Tp–e might have been overestimated. Further studies are needed to investigate the underlying mechanisms responsible for the time-dependent change in Tp–e in patients with CRT.

Conclusion
To our knowledge, this is the first study to assess the time-dependent effect of CRT on Tp–e and VAs. The largest number of patients experienced VT/VF episodes within 1 month after CRT device implantation and diminished over time. Similarly, the Tp–e decreased over time after CRT device implantation. Responders demonstrated a lower rate of VT/VF and greater time-dependent reduction of Tp–e than non-responders.

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Conflict of interest: none declared.

References
Ice vs. fire: cryoballoon ablation for the prevention of inappropriate implantable cardioverter-defibrillator shocks in a 14-year-old girl with Brugada syndrome

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We report the case of a 14-year-old girl with Brugada syndrome (BS) (Figure) admitted to our department because of repeated inappropriate implantable cardioverter-defibrillator (ICD) shocks elicited by episodes of paroxysmal rapid atrial fibrillation (AF).

Three years earlier she received a single-chamber ICD implantation because of aborted sudden cardiac death due to ventricular fibrillation and 2 years after she developed paroxysmal AF. Pharmacological therapy with sotalol and bisoprolol failed to maintain sinus rhythm and to avoid inappropriate ICD interventions. Due to the inefficacy of sotalol and the inability to use a class Ic drug for the prophylaxis of paroxysmal AF, we performed a successful pulmonary vein isolation (PVI) by means of cryoballoon (CB) ablation.

Although CB ablation of paroxysmal AF has been proven as an effective alternative to radiofrequency in achieving PVI in adults, no data have been published about CB ablation for AF in children. To our knowledge, this is the first report of successful CB isolation of PVI's in a child with BS.

The full-length version of this report can be viewed at: http://www.escardio.org/communities/EHRA/publications/ep-case-reports/Documents/cryoballoon-ablation.pdf

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