**Background:** The aim of this study was to carry out 3-dimensional speckle tracking imaging (3DSTI) of the right ventricle (RV) and evaluate RV regional wall deformation.

**Methods and Results:** 3DSTI of the RV was performed in 35 normal subjects, 8 patients with arrhythmogenic right ventricular cardiomyopathy, and 8 patients with pulmonary arterial hypertension. Peak systolic area change ratio and regional contraction timing relative to global systolic time (time to peak strain/time to end-systole × 100) were measured in each segment. Good-quality images were acquired of the inflow segment in 87%, apex in 87%, outflow in 57%, and septum in 94% of the 35 normal subjects. In normal subjects, peak systolic area change ratio of the inflow anterior wall was –41±14%; inflow inferior wall, –35±9%; apical anterior wall, –41±10%; apical inferior wall, –31±11%; outflow, –31±9%; and septum, –36±11%. Contraction timing of the apical anterior wall and septum wall were earlier than those of other segments. In patients with RV dysfunction, 3DSTI indicated low peak systolic area change ratio in the damaged area.

**Conclusions:** RV 3DSTI indicated segmental heterogeneity in magnitude and timing of RV contraction. 3DSTI may be a promising modality for providing precise quantitative information on complex RV wall motion. (Circ J 2013; 77: 1760–1768)

**Key Words:** Echocardiography; Right ventricular function; Right ventricular regional deformation; 3-D speckle tracking imaging

In recent years, many studies have confirmed the prognostic value of right ventricular (RV) function in cardiovascular disease.1–5 RV ejection fraction (RVEF) <35% has been reported to be a strong and independent predictor of mortality in left heart failure.6 Impaired RV function in advanced heart failure was also reported to be independently associated with worse ability to perform exercise.6,7 RV function and volume, however, are difficult to assess because of the fundamentally complex geometry of the RV chamber.8 Several methods have been suggested to assess RV systolic function. Two-dimensional (D) assessment of RVEF with Simpson’s method and the area-length method had moderate correlation with other modalities,9 and RV fractional area change correlated well with RVEF calculated from cardiac magnetic resonance imaging (MRI) and was superior to other 2-D methods to assess RV function.10 The American Society of Echocardiography (ASE) published recommendations for RV assessment as part of its guidelines on chamber quantification,11 but the lack of standard methods for assessing RV volumes was also recognized and discussed in this ASE document. Now, RV volume and RVEF can be calculated on dynamic 3-D echocardiography, and its accuracy has been validated on cardiac MRI.12 Although EF can be accurately measured with 3-D echocardiography compared to cardiac MRI, volumes tend to be much larger on MRI. Although there is some difference between the 2 modalities, volume assessment with 3-D images may be more desirable for RV functional analysis than with 2-D images because of the complex geometry of the RV chamber. To evaluate RV regional wall motion, RV regional longitudinal strain on tissue Doppler imaging13 and 2-D speckle tracking imaging (2DSTI) were reported.14 Those assessments, however, were limited to the inflow lateral wall, and other segments remained to be assessed. Meanwhile, for the left ventricle (LV), 2-D and 3-D speckle tracking were reported to be able to provide quantitative information on regional wall motion.15,16 Accordingly, in this study, we analyzed the feasibility and reproducibility of...
Assessment of RV Regional Deformation

Methods

Subjects
For the evaluation of normal RV 3DSTI, 45 normal subjects were enrolled. They were healthy volunteers who had normal LV size and systolic function without regional wall motion abnormalities, and normal diastolic function considering their age. They also had normal RV size and function as assessed on 2-D echocardiography. Exclusion criteria were frequent premature arrhythmias, atrial fibrillation, and poor 3-D image quality for assessment. Among consecutive normal subjects, 10 (22%) had insufficient image quality for 3DSTI analysis and were excluded from further analysis.

Ten patients with arrhythmogenic RV cardiomyopathy (ARVC) with ventricular tachycardia were also assessed. The diagnosis was based on Task Force criteria. These patients had already been diagnosed as having a damaged RV on right ventriculography. Nine patients with pulmonary arterial hypertension (PAH) were also included in this study. These patients had been diagnosed as having PAH on right heart catheterization. Their estimated RV systolic pressures on echocardiography were >40mmHg, even though they received treatment with an oral or i.v. pulmonary vasodilator. Two ARVC and 1 PAH patient were excluded because of insufficient quality of 3-D imaging.

Imaging Protocol

2-D Echocardiography
Conventional echocardiography was performed for all subjects. RV wall motion was assessed by eye with various cross-sectional approaches that are recommended by the ASE.11 RV end-diastolic diameter was obtained from the apical 4-chamber view. The percentage of RV fractional area change (FAC), defined as (end-diastolic area – end-systolic area)/end-diastolic area × 100, was also calculated from the apical 4-chamber view. Tricuspid annular plane systolic excursion (TAPSE), a method to measure the distance of systolic excursion of the RV annular segment along its longitudinal plane, was also evaluated in the apical 4-chamber view.

LV end-diastolic diameter (LVDd) and end-systolic diameter (LVDs) were acquired from the parasternal short-axis view. LV end-diastolic volume and end-systolic volume were acquired by modified Simpson’s rule using the apical 4-chamber and apical 2-chamber views. Left atrial dimension was the anteroposterior linear dimension obtained from the parasternal long-axis view.

RV Full-Volume 3-D Data Acquisition
Subsequently, the electrocardiography-gated RV full-volume 3-D dataset consisting of 6 subvolumes was acquired. For image acquisition, patients were encouraged to stop breathing during expiration. The transducer was positioned at the modified apical position (position more lateral than that in the standard apical view) for full coverage of the RV. This modified apical view was also
recommended for the purpose of precise evaluation of RV wall motion on 2-D echo in the ASE guidelines. All echocardiographic images were acquired with an ARTIDA ultrasound system with PST-2SSX transducer (Toshiba Medical Systems, Tochigi, Japan). The full-volume 3-D datasets were digitally stored and then transferred to an off-line workstation (Ultra Extend; Toshiba Medical Systems).

**3DSTI Analysis**  As shown in Figure 1, the 3-D raw data were first shown in 5 views (2 long-axis views and 3 short-axis views) on the monitor. Then, the axis through the inflow tract to the apex was adjusted manually in the long-axis views (Figure 1A). Next, endocardial tracings were done, and wall thickness was adjusted at end-diastole in one of the long-axis views. These same steps were followed in the other long-axis views. After the endocardial tracings in both long-axis views were finished, the borders of the endocardium were automatically divided into 16 segments (Figures 1C-1C-3), especially those of the septum and outflow wall. Finally, 3-D speckle tracking was automatically done by the software, and endocardial motion of the RV could be visualized (Figure 1). The tracking images were assessed by the observer, and when poor tracking was recognized visually, re-tracking was performed for more precise analysis. Although the RV endocardium was automatically divided into 16 segments by the software, we used 6 segments (inflow anterior wall; inflow inferior wall; apical anterior wall; apical inferior wall; outflow wall; and septum wall) for analysis. We measured the peak systolic area change ratio, which indicates endocardial surface % area change and is a unique index of 3-D assessment, longitudinal strain, and circumferential strain of each segment. To assess the timing of contraction of each segment, we calculated the regional contraction timing relative to global systolic time (time to peak strain/time to end-systole from the R-wave peak on the electrocardiogram) ×100.

**Figure 2.** Strain curves calculated from right ventricle (RV) 3-dimensional speckle tracking imaging: area change ratio-time curves for each segment (same patient as in Figure 1). Vertical green dotted line, end-systole. Circles, peak systolic area change ratio in each segment; arrow, time to peak strain.

**Results**

**Conventional Echocardiographic Measurements and RVEF**

Baseline characteristics and standard echocardiographic data in the normal, ARVC, and PAH groups are listed in Table 1. In the normal group, LV size and function were normal, and RV size and function were normal and LVd was smaller than in normal subjects. In contrast, RV size in the ARVC and PAH patients was significantly larger and LVd was smaller than in normal subjects. In addition, LVd and RV volumes were also smaller than in normal subjects and ARVC patients. TAPSE and FAC in ARVC and PAH patients were significantly lower than in normal subjects.

RV volumes and RVEF are also listed in Table 1. Although RVEF in normal subjects was relatively low compared with the ASE guidelines, that in ARVC and PAH patients was significantly lower than in the normal subjects.
Assessment of RV Regional Deformation

Table 1. Subject Characteristics and Basic Echocardiographic Data

<table>
<thead>
<tr>
<th></th>
<th>Normal subjects (n=35)</th>
<th>ARVC patients (n=8)</th>
<th>PAH patients (n=8)</th>
<th>P-value</th>
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<tr>
<td>Age (years)</td>
<td>48±19</td>
<td>46±22</td>
<td>48±22</td>
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<tr>
<td>Sex (F/M)</td>
<td>10/27</td>
<td>1/7</td>
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2-D echo parameters

<table>
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<tr>
<th>Parameter</th>
<th>Normal subjects</th>
<th>ARVC patients</th>
<th>PAH patients</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>RV end-diastolic diameters</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Base-to-apex length</td>
<td>66.8±7.2</td>
<td>77.5±9.7*</td>
<td>74.8±14.6*</td>
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<td>Mid-RV diameter</td>
<td>29.7±4.4</td>
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<td>39.6±6.3*</td>
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<td>Basal-RV diameter</td>
<td>33.5±5.5</td>
<td>39.6±6.9</td>
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<td>LV diameters and volumes</td>
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</tr>
<tr>
<td>LVDd</td>
<td>48.6±4.0</td>
<td>43.8±4.3*</td>
<td>39.8±6.2*</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>LVDs</td>
<td>29.9±4.4</td>
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<td>23.5±5.1†</td>
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<tr>
<td>LVESV</td>
<td>28.5±12.3</td>
<td>28±14.8</td>
<td>17.8±4.0</td>
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<td>LVEF (modified Simpson)</td>
<td>67.7±6.9</td>
<td>61.8±12.2</td>
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<td>LAD</td>
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<td>32.3±6.7</td>
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<td>TAPSE</td>
<td>22.7±3.3</td>
<td>13.5±5.7*</td>
<td>17.6±3.7†</td>
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<tr>
<td>FAC</td>
<td>42.4±8.5</td>
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<td>19.8±8.2*</td>
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<td>RVEDV</td>
<td>79.1±24.3</td>
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<td>112.4±43.3*</td>
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<td>RVESV</td>
<td>45.5±16.7</td>
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<td>85.2±36.5*</td>
<td>&lt;0.0001</td>
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<tr>
<td>RVEF</td>
<td>43.1±7.8</td>
<td>21.9±8.4*</td>
<td>25.1±6.6*</td>
<td>&lt;0.0001</td>
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</tbody>
</table>

Data given as mean±SD or n. *P<0.05 vs. normal subjects, †P<0.05 vs. ARVC patients. ARVC, arrhythmogenic right ventricular cardiomyopathy; FAC, right ventricular fractional area change; LAD, left atrial dimension; LV, left ventricular; LVDd, LV end-diastolic diameter; LVDs, LV end systolic diameter; LVEDVs, LV end-diastolic volume (modified Simpson); LVEF, LV ejection fraction (modified Simpson); LVESV, LV end-systolic volume (modified Simpson); PAH, pulmonary arterial hypertension; RV, right ventricular; RVEDV, RV end-diastolic volume; RVEF, RV ejection fraction; RVESV, RV end-systolic volume; TAPSE, tricuspid annular plane systolic excursion; 3DSTI, 3-D speckle tracking imaging.

Figure 3. (A) Area change ratio data and (B) longitudinal and circumferential strain data for each indicated segment relative to global end-systole. Apical-ant, apical anterior wall; apical-inf, apical inferior wall; inflow-ant, inflow anterior wall; inflow-inf, inflow inferior wall; sep, septum wall. *P<0.05 vs. outflow; †P<0.05 vs. apical inferior wall; ‡P<0.05.
RVSTI in Normal Subjects: Peak Systolic Strain

The mean volume rate was 19.7±1.7 volumes/s for 3-D echocardiography. In 35 normal subjects, good-quality images were acquired of the inflow segment in 30 subjects (87%), apex in 30 (87%), outflow segment in 25 (71%), and septum in 34 (97%). Peak systolic strain of each segment is shown in Figure 3A. The RV wall motion of each segment may have heterogeneity for the area change ratios. The inflow anterior wall and apical anterior wall had a significantly larger area change ratio than did the apical inferior and outflow walls.

Peak systolic longitudinal and circumferential strains are shown in Figure 3B. These parameters also showed segmental heterogeneity. Longitudinal strain of the inflow anterior wall was significantly larger than that of other areas. The peak strain of the inflow inferior wall and apical anterior wall were significantly larger than that of the septum wall. For circumferential strain, the outflow wall had significantly smaller peak strain than other areas, and there were no significant differences between these 2 strains in the apical and outflow walls. In contrast, longitudinal strain predominated in the inflow wall, and circumferential strain predominated in the septum wall.

RVSTI in Normal Subjects: Contraction Timing

Regional contraction timing of area change ratio relative to global systolic time is shown in Figure 4A. Regional contraction timing <100% indicated that the regional timing of contraction was earlier; and >100% indicated that the region had delayed contraction in relation to that of global end-systole. Contraction of the apical anterior wall and septum ceased earlier than that of the outflow wall. Regional timing of longitudinal and circumferential shortening is shown in Figure 4B. For longitudinal strain, contraction timing of the inflow inferior wall occurred later than that of the apical anterior wall and/or septum wall. For circumferential strain, the timing of contraction of the apical anterior wall and septum wall was significantly earlier than that of the other regions. In other words, the characteristics of regional contraction timing in the circumferential direction were almost identical to those of the area change ratio. In the outflow tract, regional circumferential contraction timing was significantly later than that of longitudinal contraction, whereas in the apex, there were no differences in the timing of contraction between the circumferential and longitudinal directions.

RV 3DSTI in Patients With RV Dysfunction

In 8 ARVC and 8 PAH patients, good-quality images were acquired of the inflow segment in 15 (94%), apex in 14 (88%), outflow segment in 12 (75%), and septum in 14 (88%). Figure 5 shows the comparison between normal subjects and RV dysfunction patients for area change ratio, longitudinal strain, and circumferential strain. For circumferential strain, the measurements for normal and RV dysfunction overlapped. In contrast, according to longitudinal strain and area change ratio, some segments in RV dysfunction patients showed apparent impaired strain, which was beyond the 1-SD range of strain distribution of normal RV segments. In particular, area change ratio showed less overlap compared with normal segments. Impaired area change ratio was frequently observed in inflow, outflow, and apical segments in ARVC patients. Similarly, impaired area change ratio was also observed in PAH patients, especially in...
Assessment of RV Regional Deformation

To our knowledge, this is the first report on the application of 3DSTI to the RV to evaluate regional wall motion and timing of contraction quantitatively. In normal subjects, there was segmental heterogeneity in magnitude and timing of RV contraction. Additionally, 3DSTI identified RV regional wall motion abnormality in ARVC patients. In previous studies, RV regional wall motion has been evaluated on M-mode, 2-D imaging, or tissue Doppler-derived strain imaging. Another technique for determining regional deformation is strain imaging derived from 2DSTI.

RVEF Derived From 3DSTI

3DSTI can also be used to determine RV volume and EF. In this study, RVEF of normal subjects was relatively low compared with that in previous studies. RV form cannot be represented correctly, especially for the portion between inflow and outflow by this method, because the system we used was not specialized for the RV. For this reason, RVEDV and function.

Reproducibility

Intra- and inter-observer reproducibility among normal subjects is shown in Table 2.

Discussion

Major Findings

In the present study, we applied 3DSTI to the analysis of RV wall motion to identify RV regional wall motion and global function. To our knowledge, this is the first report on the application of 3DSTI to the RV to evaluate regional wall motion and timing of contraction quantitatively. In normal subjects, there was segmental heterogeneity in magnitude and timing of RV contraction. Additionally, 3DSTI identified RV regional wall motion abnormality in ARVC patients. In previous studies, RV regional wall motion has been evaluated on M-mode, 2-D imaging, or tissue Doppler-derived strain imaging. Another technique for determining regional deformation is strain imaging derived from 2DSTI.

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The bellows-like movement of the RV differs from that of LV deformation, which starts from the apex and expands to the base with torsional deformation. The RV is reported to be made up of 3 separate mechanisms: inward movement of the free wall; contraction of the longitudinal fibers, which shortens the long axis and draws the tricuspid annulus toward the apex; and traction of the free wall at the points of attachment secondary to LV contraction.

Heterogeneity of peak systolic area change ratios was also seen in the present study. This heterogeneity may be caused by the time lag of contraction or the different role of each segment during RV contractile movement. For instance, the inward movement of the anterior wall is the primary mechanism by which blood is ejected. Moreover, the continuity between the muscle fibers of the LV and RV functionally binds the ventricles together and represents the anatomic basis of free ventricular wall traction caused by LV contraction.

Therefore, area change ratio could be used as an indicator of endocardial RV deformation independent of the ventricular axis, and this might be an ideal new 3-D parameter for assessment of RV regional wall motion.

According to contraction timing results, the apical anterior wall and septum wall contracted first, and then other regions followed, which represented a bellows-like movement of the RV. This movement of the RV differs from that of LV deformation, which starts from the apex and expands to the base with torsional deformation. The bellows-like movement of RVESV may be overestimated and RVEF may be underestimated. TAPSE and RVEF correlated well, however, similar to the correlation in previous studies.

For assessing 3-D RV motion, area change ratio, which is the integrated parameter of longitudinal and circumferential strain, was used based on the following anatomic background. The RV wall is mainly composed of 2 muscle layers: the epicardial layer and the endocardial layer. The fibers of the epicardial layer are arranged more circumferentially in a direction that is parallel to the atrioventricular groove, and these fibers turn obliquely toward the cardiac apex. The fibers of the endocardial layer are longitudinally aligned from base to apex.

Because the RV has only 2 muscle layers, shortening of the RV is greater longitudinally and circumferentially than radially. Additionally, according to the present results, each segment of the RV has a distinctive direction of contraction. Therefore, area change ratio could be used as an indicator of endocardial RV deformation independent of the ventricular axis, and this might be an ideal new 3-D parameter for assessment of RV regional wall motion.

Heterogeneity in Magnitude and Timing of RV Contraction

According to contraction timing results, the apical anterior wall and septum wall contracted first, and then other regions followed, which represented a bellows-like movement of the RV. This movement of the RV differs from that of LV deformation, which starts from the apex and expands to the base with torsional deformation. The bellows-like movement of
disease, or pulmonary hypertension. It is more important to quantify RV motion than to evaluate it by eye for patients with these diseases. In this study, we used 3DSTI for ARVC and PAH patients and quantitatively evaluated regional wall motion abnormality, as well as using impaired area change ratio. Doppler or 2DSTI-derived strain imaging, which is simple and has good temporal resolution, has provided some useful results for clinical use. The previous studies noted here, however, used 2-D apical 4-chamber views to measure regional strain, and useful parameters were limited to basal, mid, and apical free wall strain and basal, mid, and apical septum wall strain. Quantification of outflow tract strain in an animal study has been reported, but to assess the entire RV wall, many images should be acquired, and regional wall deformation in each image needs to be analyzed. 3DSTI may contribute to solving this problem. Use of 3DSTI allowed evaluation not only of regional function of the RV wall but also RV wall motion as a whole from 1 image. Additionally, as noted here, RV wall motion had a distinctive direction in each segment. In the evaluation of patients with RV dysfunction, we found that some patients had lower area change ratio than longitudinal strain and also had normal circumferential strain. This phenomenon may occur because circumferential wall motion compensates for longitudinal wall motion. Because of this aspect, assessing only longitudinal strain could lead to misjudgment of wall motion, and area change ratio could provide comprehensive data on RV wall motion and distribution. 3DSTI has the advantage of identifying RV motion precisely without angle dependency by simultaneously evaluating area change ratio, longitudinal strain, and circumferential strain. 3DSTI may allow us to assess in which direction the wall motion would be more impaired in each segment in the clinical setting. Although the ASE guidelines recommend that TAPSE should be used routinely as a simple method of estimating RV function, 3DSTI, which has no angle dependency, will provide correct information on RV deformation. Moreover, we can also evaluate the variability of peak strain and contraction timing between each segment on 3DSTI. The present study suggests that ARVC patients have a wider SD of contraction timing between each segment than normal subjects and PAH patients. These results may indicate the presence of RV dysynchrony in ARVC patients.

Feasibility and Reproducibility

The feasibility of real-time 3-D echocardiographic quantification of the RV has been reported to be approximately 80%, so feasibility in the present study was considered to be comparable. Segmental reproducibility, however, was relatively low compared with previous studies using 2-D echocardiography.

Study Limitations

We performed this study with a relatively small number of subjects in a single center, therefore further analysis with a larger number of normal subjects of various age groups is necessary to determine normal values for area change ratio in each of the RV segments. Another limitation was the lack of validation with gold standards such as conventional 2DSTI or cardiac MRI. This, however, is a preliminary report that shows the application of 3DSTI to analyze RV regional wall motion, and the primary purpose of the present study was not to determine normal ranges but to show the feasibility of 3DSTI in the RV. In regard to technical aspects, 3-D image quality, volume rates, and reproducibility still require improvement. Low reproducibility or high variability may result in a low power of discrimination to determine differences between normal functioning and disease. This point is 1 major limitation of the present study. In addition, a considerable number of normal subjects (22%) were excluded because of insufficient image quality for 3DSTI, and the low feasibility of 3-D imaging is another major limitation of this study. Improvements in image quality directly leading to reliable tracking of the endocardium and a decrease in noise artifacts may overcome this problem in the near future. If these technical problems can be overcome, reproducibility and variability may be improved. Still, we should keep in mind that there are some patients in whom sufficient 3-D images cannot be acquired, especially in the outflow tract. In such patients, application of transesophageal 3-D echocardiography may improve outflow image acquisition. Furthermore, the 3DSTI software used in the present study was not specialized for RV analysis, so the infundibulum could not be represented with this method. Considering the complex geometry of the RV, specialized software for RV 3DSTI analysis should be developed.

Conclusions

In this first application of 3DSTI to the assessment of RV global and regional wall motion, heterogeneity of wall deformation and timing of contraction have been found in normal subjects. 3DSTI could provide precise novel quantitative information on the complex wall motion of the RV and could contribute to the identification of additional pathophysiological information on RV function.

Disclosures

None.

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