Surgical Ventricular Restoration Improves Mechanical Intraventricular Dyssynchrony in Ischemic Cardiomyopathy

Marisa Di Donato, MD; Anna Toso, MD; Vincent Dor, MD; Michel Sabatier, MD; Giuseppe Barletta, MD; Lorenzo Menicanti, MD; Fabio Fantini, MD; and the RESTORE Group*

Background—In ischemic cardiomyopathy, dyssynchrony of left ventricular (LV) mechanical contraction produces adverse hemodynamic consequences. This study tests the capacity of geometric rebuilding by surgical ventricular restoration (SVR) to restore a more synchronous contractile pattern after a mechanical, rather than electrical, intervention.

Methods and Results—A prospective study of the global and regional components of dyssynchrony was conducted in 30 patients (58±8 years of age) undergoing SVR at the Cardiothoracic Center of Monaco. The protocol used simultaneous measurements of ventricular volumes and pressure to construct pressure/volume (P/V) and pressure/length (P/L) loops. Angiograms were done before and after SVR to study a 600-ms cycle during atrial pacing at 100 bpm. Mean QRS duration was similar, at 100±17 ms preoperatively and 114±28 ms postoperatively (NS). Preoperative LV contraction was highly asynchronous, because P/V loops showed abnormal isometric phases with a right shifting. Endocardial time motion was either early or delayed at the end-systolic phase so that P/L loops were markedly abnormal in size, shape, and orientation. Postoperatively, SVR resulted in leftward shifting of P/V loops and increased area; endocardial time motion and P/L loops almost normalized to allow a better contribution of single regions to global ejection. The hemodynamic consequences of SVR were improved ejection fraction (30±13% to 45±12%; P=0.001); reduced end-diastolic and end-systolic volume index (202±76 to 122±48 and 144±69 to 69±40 mL/m²; P=0.001); more rapid peak filling rate (1.75±0.7 to 2.32±0.7 EDV/s; P=0.0001); peak ejection rate (1.7±0.7 to 2.6±0.9 Sv/s; P=0.0002), and mechanical efficiency (0.56±0.15 to 0.65±0.18; P=0.04).

Conclusions—SVR produces a mechanical intraventricular resynchronization that improves LV performance. (Circulation. 2004;109:2536-2543.)

Key Words: ventricles • remodeling • surgery

Myocardial activation-contraction sequence abnormality is frequent in dilated cardiomyopathy and often associated with interventricular conduction delay to produce contraction dyssynchrony that impairs left ventricular (LV) ejection. Biventricular pacing has become a treatment option in ischemic and nonischemic cardiomyopathy with conduction delay. This concept is called electrical resynchronization and may improve LV performance by avoiding right ventricular septal displacement (interventricular dyssynchrony) and limit presystolic mitral regurgitation.1–3 LV dyssynchrony may also involve different regions of the LV mass to produce intraventricular dyssynchrony that is related to survival.4 Post–myocardial infarction (MI) cardiomyopathy is a clear subset, because the scar causes an in vivo model of tissue inhomogeneity in dyskinetic and akinetic muscle, where nonuniform contraction, relaxation, and filling may develop and contribute to deterioration of global systolic and diastolic function. This intraventricular dyssynchrony stems from asynchrony of the component parts of the LV chamber and may be independent of electrical conduction delay. Surgical ventricular restoration (SVR) accomplishes exclusion of scarred and thinned myocardium contiguous to normal, nearly normal, or ischemic tissue.

This study evaluated how chamber rebuilding by SVR may influence function by focusing on how restoring size and shape can affect global ventricular performance. None of the 30 consecutive patients with previous anterior infarction had intraventricular conduction delay. Principal attention was directed toward how component ventricular segments contributed to changes in postoperative global function. Sequential regional contraction and relaxation was evaluated to determine whether, and to what extent, ventricular rebuilding can produce a mechanical resynchronization.

Methods

Study Group

Thirty consecutive patients in sinus rhythm undergoing SVR for a previous anterior MI (mean age, 58±8 years) were prospectively studied during diagnostic heart catheterization using an intraventricular-tipped micromanometer catheter to obtain simultaneous...
pressure-volume measurements. The preoperative QRS was normal (100±17 ms), and heart rate was paced at 100 bpm (atrial pacing) during angiography to avoid cycle length differences between patients. LV angiograms were obtained in right anterior oblique 30-degree projection with power injection of 12 mL/s of nonionic contrast medium and filmed at 50 frames per second.

The procedure was performed before and after surgery (approximately 10 days) after obtaining the informed consent to repeat LV angiography. Extensive regional dyssynergy (dyskinesia or akinesia) was present in all patients, global LV function was depressed (ejection fraction was 30±13%), and congestive heart failure was present in 60% of patients.

**Parameters**

LV angiographic silhouettes were traced frame by frame for an entire cardiac cycle at 600 ms and digitized using a uniform sampling every 0.1 mm (Digitizer MYPAK-A3, model k-510 Mk2). LV volumes were calculated with the Chapman method by applying the Simpson rule, and regional wall motion was analyzed with the Centerline method for 45 chords around the ventricular perimeter, from the aortic (chord 1) to the mitral corner (chord 45). An original proprietary software allowed calculation of endocardial time motion of 15 regional segments (6 anterior, 3 apical, and 6 inferior).

Each segment motion resulted from the average of 3-contiguous-chord motion of the 45 chords identified by the Centerline method. These regions were considered to be component regions contributing to global function. Control endocardial motion values during 1 cardiac cycle (Figure 1) were taken from a subset of 15 subjects with normal values at diagnostic cardiac catheterization and angiography.

**Segmental Pressure/Length Loop**

Segmental pressure/length (P/L) loops were constructed at the 45 chords identified by the Centerline, plotting LV pressure (ordinate) against the endocardial movement (abscissa) throughout the entire cycle (Figure 2). The sequence of values along each chord was smoothed using a 0.26- to 6.6-Hz low-pass filter.

Systolic and diastolic parameters were also analyzed:

- Systolic parameters included end-systolic pressure (ESP) (mm Hg), measured as the pressure value at the minimal volume; end-systolic volume index (ESVI) (mL/m²); ejection fraction (EF); stroke volume index (mL/m²); systolic stroke work (g*m); peak ejection rate (Sv/s); rapid ejection (ms); slow ejection (ms); and pressure/volume (P/V) max (the maximal value of P/V ratio) (mm Hg/mL).

- Diastolic parameters included end-diastolic pressure (mm Hg); minimal diastolic pressure (mm Hg); peak filling rate (PFR) (EDV/s); the constant Tau of pressure decay according to Weiss et al; and diastolic stroke work (g*m).

In addition, we calculated the following:

- **Mechanical efficiency** was the ratio between the effective stroke work and the theoretical maximal work in g*m. Theoretical maximal work = (Pmax − Pmin) × stroke volume × 0.0136.
- **Synchrony index** was the number of segments (N) reaching the maximal motion at the end-systolic phase with respect to all 15 segments. SI = N/15.
- **Uniformity index:** LV contraction was considered uniform when the degree of contribution to global ejection was similar for all the segments within the cardiac cycle. The area of P/V loop represents a measure of external (global) work of the ventricle. The component aspects were evaluated by P/L loop area that tabulates an index of how single ventricular chords contribute to total ventricle work. The optimal contribution to ejection by a chord is obtained when the morphology of the loop is rectangular (preserved isometric phases) and the area (effective work) is not narrowed. We related the effective area for each single P/L loop to a theoretical ideal area (the loop area underlying a theoretical isometric phases) and the area (effective work) is not narrowed. We related the effective area for each single P/L loop to a theoretical ideal area (the loop area underlying a theoretical rectangular loop with similar abscissa shifting).

Figure 2 shows a drawing of different types of P/L loops that are observed in our patients. The normal loop is the white area (dotted lines), and the ideal, theoretical loop is the white rectangle having the same shifting of the dotted line from end diastole to end systole. Portions located outside the hypothetically normal (vertical) isometric phases (gray areas) do not perform any effective work and are considered at zero work. Portions at zero work are present in all loop configurations (A, clockwise [CW]; B, figure-8 configuration; C, early lengthening; and D, early shortening). Conversely, an indirect estimate of effective work of each chord occurred in portions lying inside the ideal theoretical loop. We calculated the ratio between the effective and theoretical area for all of the 45 chords around the LV perimeter. The summation of each

**TABLE 1. Clinical Characteristics of the Study Group**

<table>
<thead>
<tr>
<th>Variable, No. patients (%)</th>
<th>Men/women, n</th>
<th>Age, y</th>
<th>Delay from MI, mo</th>
<th>QRS duration, ms</th>
</tr>
</thead>
<tbody>
<tr>
<td>NYHA class</td>
<td>273</td>
<td>58±8</td>
<td>35±41</td>
<td>100±17</td>
</tr>
<tr>
<td>I–II</td>
<td>13 (43.3)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>III</td>
<td>10 (33.3)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IV</td>
<td>7 (23.3)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Angina</td>
<td>19 (63.3)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CHF</td>
<td>18 (60)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Coronary disease</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 Vessel</td>
<td>9 (30)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2 Vessels</td>
<td>16 (53.3)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3 Vessels</td>
<td>5 (16.7)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Type of asynergy</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dyskinetic</td>
<td>19 (63.3)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Akine tic</td>
<td>11 (36.7)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

CHF indicates congestive heart failure; NYHA, New York Heart Association.

**TABLE 2. Hemodynamic Results**

<table>
<thead>
<tr>
<th>Variable, No. patients (%)</th>
<th>Preoperative</th>
<th>Postoperative</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>QRS duration, ms</td>
<td>100±17</td>
<td>114±28</td>
<td>NS</td>
</tr>
<tr>
<td>Systolic parameters</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ESP, mm Hg</td>
<td>122±15</td>
<td>104±22</td>
<td>0.016</td>
</tr>
<tr>
<td>CI, L/min per m²</td>
<td>2.8±0.6</td>
<td>2.7±0.7</td>
<td>NS</td>
</tr>
<tr>
<td>SVI, mL/beat</td>
<td>56±20</td>
<td>59±13</td>
<td>NS</td>
</tr>
<tr>
<td>EF, %</td>
<td>30±13</td>
<td>45±12</td>
<td>0.001</td>
</tr>
<tr>
<td>EDVI, mL/m²</td>
<td>202±76</td>
<td>122±48</td>
<td>0.001</td>
</tr>
<tr>
<td>ESVI, mL/m²</td>
<td>144±69</td>
<td>69±40</td>
<td>0.001</td>
</tr>
<tr>
<td>PNmax, mm Hg/mL</td>
<td>1.06±0.7</td>
<td>1.6±0.8</td>
<td>0.001</td>
</tr>
<tr>
<td>PER, Sv/s</td>
<td>1.7±0.7</td>
<td>2.6±0.9</td>
<td>0.0002</td>
</tr>
<tr>
<td>ME</td>
<td>0.56±0.15</td>
<td>0.65±0.18</td>
<td>0.04</td>
</tr>
<tr>
<td>Diastolic parameters</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PFR, EDV/s</td>
<td>1.75±0.7</td>
<td>2.32±0.7</td>
<td>0.0001</td>
</tr>
<tr>
<td>Pmax, mm Hg</td>
<td>12±9</td>
<td>5±4</td>
<td>0.003</td>
</tr>
<tr>
<td>r, ms</td>
<td>56±19</td>
<td>44±10</td>
<td>0.01</td>
</tr>
<tr>
<td>EDP, mm Hg</td>
<td>20±12</td>
<td>17±8</td>
<td>NS</td>
</tr>
<tr>
<td>CWP, mm Hg</td>
<td>14±7</td>
<td>10±4</td>
<td>0.0001</td>
</tr>
<tr>
<td>Mean PAP, mm Hg</td>
<td>21±8</td>
<td>13±6</td>
<td>0.001</td>
</tr>
<tr>
<td>Sphericity index</td>
<td>0.68±0.05</td>
<td>0.74±0.05</td>
<td>0.01</td>
</tr>
<tr>
<td>DSW, g/m per min</td>
<td>19±12</td>
<td>13±8</td>
<td>0.016</td>
</tr>
</tbody>
</table>

CI indicates cardiac index; ESP, end systolic pressure; PNmax, pressure volume maximal ratio; SVI, stroke volume index; EDVI, end-diastolic volume index; ESVI, end-systolic volume index; PER, peak ejection rate; ME, mechanical efficiency; PFR, peak filling rate; P, pressure; r, constant of pressure decay; EDP, end-diastolic pressure; CWP, capillary-wedge pressure; PAP, pulmonary artery pressure; sphericity index, short-to-long-axis ratio; and DSW, diastolic stroke work.
value was divided by 45 to obtain an average uniformity index, whereby a value of 1 represents the optimal and zero the worst contribution.

**Surgical Procedure**

The surgical technique of ventricular reconstruction (SVR) for post-MI ischemic cardiomyopathy has been reported in previous studies.10–12 Briefly, the procedure is conducted on heart arrested with antegrade crystalloid or cold blood cardioplegia. Coronary revascularization is performed first with internal mammary and venous grafts. Then the left ventricle is opened at the center of the depressed area, and a circular suture (Fontan Stitch) is performed at the transitional zone between scarred/fibrotic and normal tissue and tied over a sizing device inflated at 50 to 60 mL/m². An oval synthetic patch then closes the ventricular opening or a direct suture is used with 3-cm opening. The mitral valve is evaluated by transesophageal echo, and mitral repair is done (by either atrial or ventricular approach) if mitral regurgitation is quantified as grade 2 or 35 mm mitral annulus size.

**Statistical Analysis**

Values are reported as mean±SD. Two-tailed Student’s t test for paired data was used to test preoperative and postoperative differences. P<0.05 was considered significant.

**Results**

Clinical characteristics of the study group are reported in Table 1. All patients had patch repair and coronary bypass surgery with internal mammary artery on left anterior descending; 1 patient had associate mitral repair for grade 3+ mitral regurgitation.

**Global Systolic and Diastolic Function**

Preoperative and postoperative hemodynamic parameters are reported in Table 2. Figure 3 shows the improvement of regional wall motion after surgery in the 2 cases. A significant improvement in systolic function was evidenced by an increase in EF and pressure/volume maximal ratio (P/V max), a decrease in ESVI, and an increase in peak ejection rate. The 2 components of ejection, ie, the rapid and the slow component, were considered, and the rapid ejection (rapid ejection from 225±33 to 178±39 ms; P=0.0001) phase increased markedly. Diastolic function also improved, because surgical restoration provided a significant increase in PFR and decrease of constant Tau of pressure decay in minimal pressure and diastolic work.

**Global P/V Loops**

Mechanical efficiency, as calculated by P/V loops, significantly improved (from 0.56±0.15 to 0.65±0.18; P<0.04),
and a marked left shifting of the curve, with an improvement of isometric phases, was observed after surgery (Figure 4).

**Endocardial-Time Motion**

Figure 1 shows endocardial time motion (continuous line) of the component parts of anterior, apical, and inferior segments in cases 1 (left) and 2 (right). Gray vertical bars represent the standard deviations of endocardial time motion in healthy subjects. Endocardial time motion in healthy subjects is synchronous and symmetric within the cardiac cycle. In healthy subjects, all segments reach their maximal motion at the end-systolic phase (vertical line) (synchrony), and the endocardial motion has the same direction in all regions (symmetry). In contrast, our preoperative patients showed high asymmetry, ie, opposite direction motion (dyskinesia), absence of motion (akinesia), or reduced motion (hypokinesia). More importantly, the temporal sequence of contraction is markedly abnormal in that only few segments reach their maximal motion at the end-systolic phase (dyssynchrony). After SVR, a greater number of segments resynchronize, reaching maximal motion at end-systolic phase. Overall, the mean SI improves from 0.23±0.12 to 0.60±0.13 (P<0.0001).

**Pressure/Length Loops**

Preoperatively, most P/L loops around the LV perimeter have abnormal shape and orientation. The most common abnormalities were early shortening and early relaxation or figure-8 configuration (Figure 5). These abnormalities were more frequent in adjacent segments but occurred in remote regions (chords 38 and 40 in case 1 and 35 and 37 in case 2). Preoperatively, most of the loops contained a narrowed area, and several are right oriented (CW or counterclockwise [CCW]), thereby located to the right of the theoretical isometric contraction phase (vertical line in Figure 2). After surgery, almost all chords normalize, showing normal isometric phases and increased area at all regions (scarred and remote).

Table 3 shows that overall, only 18% of P/L loops were normal before surgery. SVR significantly improves the number of normal P/L loops and reduces P/L loop abnormalities with a consistent and significant decrease of left-oriented (CW and CCW) and right-oriented CCW loops. Right-oriented CW loops were seen at the scarred regions and did not change significantly after surgery. The mean uniformity index increased significantly after SVR (from 0.28±0.17 to 0.47±0.17; P<0.0001), showing how single-chord contributions improved global ejection.

**Discussion**

This study shows that postinfarction cardiomyopathy develops a marked intraventricular dyssynchrony and nonuniform contraction. This abnormality was independent of electrical conduction delay, because no patient had left-bundle branch block and preoperative mean duration of QRS was <120 ms. The global hemodynamic consequences of ventricular restoration showed that SVR improved systolic and diastolic function. EF and P/Vmax rose, end-systolic pressure and ESVI were reduced, PFR increased, Tau and diastolic stroke work decreased, and mechanical efficiency improved.

Simultaneously, SVR reduced intraventricular dyssynchrony as the components of asynergy changed. Most P/L loops became normal in shape, size, and orientation, whereas only 18% were normal preoperatively. The more uniform contribution of each single segment to global ejection contributed to an improvement in uniformity index. Mechanical efficiency improved as the synchrony index increased, showing that more segments reached maximal motion at the end-systolic phase or mechanical resynchronization.

**Mechanical Performance in Ischemic Cardiomyopathy**

The heart is a muscular pump composed of contractile elements, cardiac myocytes, which are central to the contractile function and viability of myocardium. The extracellular matrix (cardiac interstitium and collagen) connects the cells in a complex array of fibers forming the 3D architecture of ventricular wall while coordinating the delivery of forces generated by myocytes. These forces are important determinants of diastolic and systolic stiffness and serve to resist deformation and maintain shape and wall thickness, thereby preventing ventricular bulge and rupture.13 The increase in chamber volume after myocardial infarction compensates for the decreased performance of the ischemic or necrotic regions, thus increasing wall tension. In post-MI patients, like ours, mechanical events such as early and late shortening, early and late relaxation, or lengthening reflect interacting areas of differing performance of myocardium, which are considered irreversible. Conversely, during ischemia, similar abnormalities of P/L loops are linked to afterload increase and become reversible when blood supply is restored.14,15

Systolic lengthening occurs during the isovolumic phase and is caused by stretching of ischemic/scarred adjacent segments by the normally contracting segments, as readily seen in aneurysms. Energy is expended as normal segments contract to produce pressure, because tensile strength is required. The ischemic/scarred segment is unable to generate sufficient tension and thus passively bulges. The consequence is dissipation and wasting of some energy produced by...
normal segments, which stretch the ischemic/scarred tissue and therefore does not contribute to ejection. These changes explain the impaired pressure volume loops shown in Figure 4, which define global function that was ultimately changed by restoration.

Effects of SVR on Mechanical Performance
Preoperatively, the endocardial time motion defined mechanical synchrony in controls and became asynchronous with maximal motion at different phases within the 600-ms cycles with regard to healthy subjects (intraventricular mechanical dyssynchrony). Regional differences improved after restoration, because SVR made almost all regions develop more uniform endocardial motion that reached its maximum extent at end-systolic phase. Such uniformity contributed ejection without wasting energy, thereby defining mechanical resynchronization.

Analysis of P/L loops provided a more sensitive means of examining regional components of synchrony and uniformity, because they defined contraction of given segments toward global LV performance. The area enclosed by each loop over the entire course of the cardiac cycle reflects the net mechanical work of the myocardial segment. Although LV pressure reflects the time course of contraction of LV mass, local segment length provides focus and reflects the time course of the contraction of the ischemic/scarred segment and how it interacts with adjacent normal myocardium.

Before surgery, most P/L loops showed abnormalities in morphology, size, and orientation. The most common observed abnormalities were early shortening and early relaxation, with markedly reduced effective work (loop D in Figure 2). Early shortening occurs because of the unloading effect of dyskinetic myocardium in series, which acts as an elastic slack element during the isovolumic phase of contrac-
tion.17 Right-oriented CW loops (loop A in Figure 2) were also observed at the anteroapical regions preoperatively. This type of loop abnormality means the loss of all contractile properties. Paradoxical systolic expansion means absence of force development and stretching by adjacent normal fibers. Early shortening and early lengthening are experimentally reproducible by connecting weak means absence of force development and stretching by adjacent normal fibers. Early shortening and early lengthening are experimentally reproducible by connecting weak and strong myocardium in series.18 Thus, within 1 cardiac cycle, regional P/L loops move in an opposite direction and asynchronously, giving each region a different contribution to global ejection.

Postoperatively, an almost complete reversal of P/L loops abnormalities occurred in regions remote from anterior scar. Most loops in the inferior regions reverted to normal orientation and shape, with steeper isometric phases and increased area, reflecting an increased effective work and more synchronous ejection. These findings are consistent with our previous report describing that preoperative P/L loop abnormalities were independent of myocardial ischemia and thought to be caused by high wall tension imposed by the large anterior scar.19

The present study confirms that relieving the abnormal tension by excluding the scar and reducing the volume allows SVR to improve overall mechanical performance by increasing effective work of each single loop. Normalization of most regional P/L loops became transcribed into marked improvement in isovolumic phases of the global P/V loops and increased mechanical efficiency. The reduction of dys synchrony during the relaxation phase likely explains the improvement in global LV diastolic function, as evidenced by the improvement in Tau and PFR.

Acute hemodynamic improvement induced by SVR depends on a complex interplay of changes in LV geometry and shape, loading conditions, and stress distribution within the LV wall. In fact, this surgical intervention includes revascularization, reduction of mitral regurgitation, and exclusion of the scar, and all these procedures may be beneficial to pump function improvement by relieving ischemia and reducing volume overload and remodeling. Consequently, the effects of SVR are complex, and we can only hypothesize that the observed mechanical resynchronization contributed to the postoperative improvement in function. Ventricular restoration by SVR is a fixed geometric event that changes many hemodynamic determinants of cardiac function, so that we cannot precisely quantify the effective role of mechanical resynchronization. These induced anatomic changes are not reversible and cannot be turned on and off like electrical resynchronization induced by biventricular pacing, so we cannot compare these mechanical effects with those obtained after electrical stimulation.

Limitation of the Study
LV volumes in patients with marked abnormal shape attributable to postinfarction cardiomyopathy were calculated from single-plane RAO LV angiography. We recognize that an incorrect estimate of volumes occurred if our measurements were compared with biplane measurements.20 However, this underestimation of volume measurements does not disprove the results, because this was a case-control study whereby each value was made preoperatively and postoperatively in each patient. Moreover, the study focuses on the changes induced by SVR on the asynchrony and asymmetry of wall motion, which are dimensionless numbers and independent of volume measurements. The endocardial P/L loops also allowed focus on how regional measurements altered global performance evaluated by P/V loops that are also dimensionless numbers.

This study describes a mechanical intraventricular dyssynchrony that is central to LV dysfunction in ischemic cardiomyopathy in hearts with normal conduction. This dyssynchrony is therefore not dependent on conduction delay. SVR improves LV performance through a mechanical resynchronization of LV contraction caused by improved synergic distribution of regional stresses during the isovolumic contraction and relaxation phases. This resynchronization reduces wasted energy and improves systolic and diastolic function.

Appendix

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