Impaired Maximal Rate of Left Ventricular Relaxation in Patients with Coronary Artery Disease and Left Ventricular Dysfunction

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SUMMARY  It has been suggested that the rate of left ventricular (LV) relaxation is related to the inotropic state, end-systolic fiber length and peak LV pressure, but little information is available regarding the rate of LV relaxation in patients with coronary artery disease (CAD) and LV dysfunction. To assess the rate of LV relaxation, we obtained high-fidelity LV pressure measurements with manometer-tip catheters in 39 patients. The signal was analyzed by a digital computer to yield the maximal rate of pressure rise (pos dP/dt) and the maximal rate of pressure fall (neg dP/dt). Selective coronary arteriography and biplane LV angiography with determination of LV volumes, ejection fraction (EF) and percent abnormally contracting segments (ACS), when present, were performed in all patients. In 10 patients with normal LV function (EF > 0.50, no asymmetry) mean neg dP/dt (2074 ± 121 mm Hg/sec) was significantly (p < 0.01) greater than in 29 patients with CAD and LV dysfunction (1695 ± 66 mm Hg/sec). In nine patients with LV dysfunction and EF < 0.35, mean neg dP/dt was reduced to 1405 ± 107 mm Hg/sec, significantly (p < 0.01) lower than in patients with normal LV function. Neg dP/dt correlated well with pos dP/dt (r = 0.75), with EF (r = 0.74), and with ACS (r = -0.74), and less well with LV end-systolic volume (r = -0.67). There was very poor correlation between neg dP/dt and peak LV pressure (r = 0.30).

These data suggest that the rate of LV relaxation, as assessed by neg dP/dt, is impaired in patients with CAD and LV dysfunction, and the extent of impairment is related to the severity of the dysfunction as determined hemodynamically by pos dP/dt, and angiographically by EF and ACS. In these patients the maximal rate of LV relaxation is inversely related to LV end-systolic volume, and is not related to peak LV pressure.

RELAXATION OF CARDIAC MUSCLE is an energy-dependent process that can be altered independently of contraction and can be modified by pharmacologic agents, and disease states, ischemia, and changes in the inotropic state of the myocardium. Clinical and animal studies have shown that changes during isovolumic left ventricular (LV) relaxation can be a sensitive and early indicator of myocardial dysfunction, and the study of the relaxation phase may be important in the hemodynamic evaluation of LV performance. In patients with coronary artery disease (CAD), LV function can be significantly impaired with marked alterations in the isovolumic and ejection phases of contraction, but the changes in isovolumic relaxation in these patients have not been well defined.

In this study we assess LV relaxation in patients with LV dysfunction secondary to CAD, and determine relationships between the maximal rate of isovolumic relaxation and hemodynamic and angiographic parameters of LV performance.
Methods

Hemodynamic Evaluation

After giving informed consent, 39 patients underwent diagnostic right- and left-heart catheterization in the postabsorptive state, approximately 60 minutes after premedication with Phenergan 50 mg and atropine 0.6 mg, both by intramuscular injection. All other medications had been discontinued 12 hours before study. High-fidelity LV pressures were recorded using a Millar Instruments Model PC-370 #7F micromanometer-tip catheter (frequency response 0–20 kHz, natural frequency 35 kHz), inserted via a right brachial arteriography. The manometer-tip catheter was calibrated before its insertion using the Millar Instruments Model TCB-100 transducer control unit. The high-fidelity LV pressure signals were amplified and recorded by an Electronics for Medicine DR-8 recorder and simultaneously digitized, on request, in 15-second increments by an IBM 1800 computer at a sampling rate of 1,000 samples/sec, so that 15,000 data points along the LV pressure waveform, comprising 15–25 consecutive cardiac cycles, were available for on-line, beat-by-beat computer analysis. The high-fidelity LV pressure signals were analyzed by the computer to yield the median value of the following indices of LV performance: 1) LV end-diastolic pressure (LVEDP); 2) peak LV pressure; 3) maximal positive dP/dt (positive dP/dt), the maximal rate of LV pressure rise during isovolumic contraction, and 4) maximal negative dP/dt (negative dP/dt), the maximal rate of LV pressure fall during isovolumic relaxation. The computer calculated dP/dt using a quadratic digital filter which has a linear response up to 40 Hz and decreases by 3 dB beyond 60 Hz.

When hemodynamic measurements were performed, all patients were stable, with no clinical or electrocardiographic evidence of acute ischemia.

Angiographic Evaluation

After baseline hemodynamic measurements, all patients, underwent selective coronary angiography and biplane left ventriculography. In 29 patients, coronary angiography was performed before left ventriculography, and in 10 patients it was performed afterwards. Biplane left ventriculograms were obtained in the anteroposterior and lateral projection in all patients, after injecting 60 ml of sodium and meglumine diatrizoate (Renografin-76) through a #8F Cook pigtail ventriculography catheter. LV volumes were determined using the area-length method of Dodge et al. End-diastolic and end-systolic frames were selected by visual inspection, and films exposed during premature contractions or the following cycle were excluded from analysis. The angiographic ejection fraction (EF) was calculated as follows:

\[
EF = \frac{\text{end-diastolic volume} - \text{end-systolic volume}}{\text{end-diastolic volume}}
\]

Consecutive end-diastolic and end-systolic frames were superimposed using fixed external x-ray beam markers as reference system. The percent abnormally contracting segment (%ACS) was calculated by the method of Feild et al. The value of the %ACS in the two projections was averaged and used as an estimate of the amount of akinetic or dyskinetic myocardium present.

Assessment of LV Function

LV function was defined as normal when the following conditions were present: 1) LVEDP of 12 mm Hg or less; 2) normal LV volumes (the following values have been established as normal for our laboratory; end-diastolic volume 77 ± 15 (SD) ml/m², end-systolic volume 31 ± 9 (SD) ml/m²); 3) EF of 0.50 or greater, and 4) normal regional wall motion. If any of these conditions were absent, LV function was considered to be abnormal.

Patients

Based on the hemodynamic and angiographic assessment of LV function, the 39 patients studied were divided into two groups (table 1). The group with normal LV function was composed of 10 patients (two females and eight males) with a mean age of 42 ± 3 years. None of these patients had a history or elec-

| TABLE 1. Summary of Clinical and Catheterization Data                                   |
|-----------------------------------------------|-------------------------------|-------------|
| Normal LV function                          | Abnormal LV function          | p           |
| Patients (no.)                              | 10                            | 29          |
| Age (years)                                 | 42 ± 3                        | 49 ± 1      |
| Sex                                          | 2 F, 8 M                      | 1 F, 28 M   |
| Previous MI (no. patients)                  | 0                             | 23          |
| Inferior                                    | 0                             | 1           |
| Anterolateral                               | 0                             | 11          |
| Digoxin (no. patients)                      | 0                             | 7           |
| Propranolol (no. patients)                  | 1                             | 7           |
| Catheterization data:                       |                               |             |
| HR (beats/min)                              | 81 ± 3                        | 73 ± 2      |
| Peak LV pressure (mm Hg)                    | 112 ± 3                       | 124 ± 3     |
| LVEDP (mm Hg)                               | 9 ± 3                         | 14 ± 1      |
| EF                                          | 0.59 ± 0.02                   | 0.39 ± 0.02 |
| ACS (no. patients)                          | 0                             | 24          |
| CAD (no. patients)                          | 2                             | 29          |
| 1V                                          | 1                             | 9           |
| 2V                                          | 1                             | 12          |
| 3V                                          | 0                             | 8           |

Values represent mean ± SEM.
Abbreviations: LV = left ventricular; MI = myocardial infarction; HR = heart rate; LVEDP = left ventricular end-diastolic pressure; EF = ejection fraction; ACS = abnormally contracting segment; CAD = coronary artery disease; V = coronary vessel with ≥ 75% luminal stenosis; NS = not significant.
trocardiographic evidence of previous myocardial infarction. One patient had taken propranolol (20 mg/day), but this drug was discontinued 12 hours before catheterization. Mean heart rate was 81 ± 3 beats/min, peak LV pressure 112 ± 3 mm Hg, LVEDP 9 ± 3 mm Hg and EF 0.59 ± 0.02. None of these patients had abnormally contracting segments; two had CAD (≥75% stenosis of a major coronary artery); one had complete occlusion of the right coronary artery and the other had patent saphenous vein grafts to completely occluded right and left anterior descending arteries. In both of these patients, collateral circulation to the regions distal to the occlusions appeared adequate, and they had no wall motion abnormalities.

The group with abnormal LV function comprised 29 patients (one female and 28 males) with a mean age of 49 ± 1 years. Twenty-three patients had a history and electrocardiographic evidence of a transmural myocardial infarction (Q waves of at least 0.03 second duration or QS complexes in two or more leads of the standard 12-lead ECG) at least 4 weeks before study. In 12 patients the infarction was inferior, and in 11 patients it involved the anterolateral wall. Seven patients were on maintenance digoxin (0.25 mg/day) and seven were on propranolol therapy (20-160 mg/day). These drugs were discontinued 12 hours before study. At cardiac catheterization the mean heart rate was 73 ± 2 beats/min, peak LV pressure 124 ± 3 mm Hg, LVEDP 14 ± 1 mm Hg and EF 0.39 ± 0.02. Abnormally contracting segments were present in 24 patients. All 29 patients in this group had CAD; nine had disease involving only one major coronary artery, 12 had disease of two major coronary arteries, and in eight the three major vessels were involved. Two patients in this group had undergone coronary artery bypass surgery; at the time of the present study one of these patients had a completely occluded proximal left anterior descending coronary artery and a patent saphenous vein graft supplying the distal

**Figure 1.** Percent abnormally contracting segment. End-diastolic (dashed lines) and end-systolic (solid lines) silhouettes in the anteroposterior (AP) and lateral projections were superimposed using fixed x-ray beam markers (solid dots) as reference system. The region of the end-diastolic circumference which is akinetic or dyskinetic (portion between brackets) was expressed as a percentage of the total end-diastolic circumference. This percentage was averaged for the AP and lateral projections and expressed as percent abnormally contracting segment. Hypokinesis is not detected by this method.

**Figure 2.** Negative dP/dt in patients with normal and abnormal left ventricular (LV) function. The 10 patients with normal LV function had a significantly greater negative dP/dt than the 29 patients with abnormal LV function and the nine patients with EF <0.35. EF = ejection fraction.

\[
\text{PERCENT ABNORMALLY CONTRACTING SEGMENT} = \frac{\text{AKINETIC or DYSKINETIC LENGTH}}{\text{TOTAL END DIASTOLIC CIRCUMFERENCE}} \times 100
\]

\[\text{NEG} \text{dP/dt (mmHg/sec)}\]

\[\text{N} = 10\]

\[\text{NORMAL LV FUNCTION}\]

\[\text{1695 ± 66}\]

\[\text{ALL PTS.}\]

\[\text{EF} < 0.35\]

\[\text{N} = 29\]

\[\text{N} = 9\]

\[\text{ABNORMAL LV FUNCTION}\]

\[\text{1405 ± 107}\]

\[\text{MEAN ± SEM}\]

\[\ast \quad p < 0.01\]

\[\text{vs NORMAL}\]
vessel; the other patient had three-vessel disease with three saphenous vein grafts, two that were patent and one that was occluded.

Statistics

Linear correlations were determined using the method of least-squares analysis, and differences between groups were assessed by the nonpaired t test or the chi-square test. Unless stated otherwise, data are presented as mean values ± SEM.

Results

Negative dP/dt for the two groups of patients is shown in figure 2. In the 10 patients with normal LV function, negative dP/dt was 2074 ± 121 mm Hg/sec, and in the 29 patients with abnormal LV function it was significantly reduced (p < 0.01) to 1695 ± 66 mm Hg/sec. In the group with abnormal LV function, nine patients had markedly impaired LV performance and an EF < 0.35; in these nine patients, negative dP/dt was significantly reduced (p < 0.01) to 1405 ± 107 mm Hg/second. Figures 3 and 4 illustrate the relation of negative dP/dt to positive dP/dt and to EF in all 39 patients. The correlations of negative dP/dt with positive dP/dt and with EF were very good (r = 0.75 and 0.74, respectively). Patients with a reduction in positive dP/dt and EF also had a diminished negative dP/dt.

It is interesting to observe the ratio of peak negative
FIGURE 5. Correlation of percent abnormally contracting segment (%ACS) with negative dP/dt in the 24 patients with abnormal left ventricular function and areas of akinesis or dyskinesis.

FIGURE 6. Correlation of end-systolic volume (ESV) and negative dP/dt. In the 10 patients with normal left ventricular (LV) function (A) no significant relation between these two parameters was observed. In the 29 patients with abnormal LV function (B) an inverse correlation ($\tau = -0.67$) was present.

TABLE 2. Ratio of Neg dP/dt to Pos dP/dt

<table>
<thead>
<tr>
<th></th>
<th>Normal LV function (n = 10)</th>
<th>Abnormal LV function (n = 29)</th>
<th>Patients with EF &lt; 0.35 (n = 9)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neg dP/dt</td>
<td>1.37</td>
<td>1.24</td>
<td>1.14*</td>
</tr>
<tr>
<td>Pos dP/dt</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*p < 0.05 vs normal LV function.

Abbreviations: Neg dP/dt = maximal negative dP/dt; Pos dP/dt = maximal positive dP/dt; LV = left ventricular; EF = ejection fraction.

dP/dt to peak positive dP/dt (table 2). As LV function deteriorates, this ratio progressively diminishes. In the 10 patients with normal LV function the ratio was 1.37, and in the nine patients with markedly impaired LV function and EF < 0.35, this ratio was significantly ($p < 0.05$) reduced to 1.14. In the 29 patients with abnormal LV function the ratio was 1.24, and although it was smaller than in the normals, the difference was not statistically significant.

The relation between %ACS and negative dP/dt in the 24 patients with abnormal LV function who had areas of akinesis or dyskinesis is shown in figure 5. Those patients with larger areas of akinesis or dyskinesis, manifested by greater %ACS, also had a more significant reduction in negative dP/dt. There was a strong inverse correlation ($r = -0.74$) between these two parameters. Figure 6 illustrates the relation between end-systolic volume and negative dP/dt in the two groups. In the patients with normal LV function, there was no correlation ($r = -0.03$) between end-systolic volume and negative dP/dt; however, in those with abnormal LV function, there was an inverse correlation ($r = -0.67$) between these two parameters, so that, as end-systolic volume increases, negative dP/dt decreases. The relation of peak LV pressure to negative dP/dt is shown in figure 7. There was no correlation ($r = 0.00$) between peak LV pressure and negative dP/dt in the patients with normal LV function, and very little relationship ($r = 0.30$) between these two parameters in the patients with abnormal LV function.

Discussion

The present study demonstrates that in patients with CAD and LV dysfunction, the maximal rate of LV pressure fall during isovolumic relaxation is reduced. The magnitude of this reduction in relaxation velocity is related to the severity of the dysfunction as assessed hemodynamically by positive dP/dt and angiographically by EF and abnormally contracting segments. Negative dP/dt was significantly reduced in patients with abnormal LV function, with a greater reduction in patients with more severe depression in maximal positive dP/dt and EF. In patients with abnormal LV function and abnormally contracting segments there was also a good inverse correlation with maximal negative dP/dt, and those with larger akinetic or dyskinetic segments also had a greater reduction in maximal rate of pressure fall.

Determinants of Maximal Negative dP/dt

The maximal rate of LV relaxation is related to the active removal of calcium from the contractile sites.
and to the passive elastic and viscous properties of the myocardium. Studies in the isolated papillary muscle have shown that relaxation is loosely coupled to contraction, and the degree of coupling can be altered by inotropic interventions that result in similar changes in contraction and different changes in relaxation, suggesting that the processes can be modified independently. Cohn et al. have suggested that the maximal rate of LV pressure fall is related to the intrinsic contractility and the end-systolic volume of the left ventricle. Their observations in the dog heart suggest that an increase in LV contractility or end-systolic volume results in an increased maximal rate of LV pressure fall during isovolumic diastole. Weisfeldt et al. observed that the peak aortic pressure was an important hemodynamic determinant of the maximal rate of pressure fall during isovolumic diastole, and their observations appear to indicate that LV wall stress is a major determinant of peak relaxation rate. Recently, Weiss et al., in an isolated canine LV preparation, and Karliner et al., in the conscious dog heart, have obtained data suggesting that the rate of isovolumic ventricular relaxation, after maximal negative dP/dt, is independent of peak LV systolic pressure or end-systolic volume, and is principally influenced by the rate and extent of fiber shortening during systole.

Mechanisms of Impairment in Maximal Negative dP/dt in Patients with LV Dysfunction

The mechanisms responsible for the reduction in the peak rate of pressure fall in the patients with LV dysfunction are not apparent in our study, but several factors should be considered. It has been shown experimentally that partial or complete occlusion of a coronary artery results in a significant and rapid fall in maximal negative dP/dt, despite the fact that peak aortic pressure and heart rate are held constant, and even before any changes in positive dP/dt are detected. McLaurin et al. showed that in patients with CAD and angina pectoris, atrial pacing produced a significant fall in maximal negative dP/dt, an increase in LVEDP and a decrease in echocardiographic LV internal diameter, changes that were consistent with incomplete relaxation produced by tachycardia-induced ischemia.

These studies suggest that the reduction in the peak rate of pressure fall in our patients with CAD may be at least partially related to myocardial ischemia. Although at the time of hemodynamic measurements none of our patients had chest pain or electrocardiographic changes, chronic myocardial ischemia has to be considered in the presence of hemodynamically significant coronary stenoses. The relaxation of LV myocardium is an energy-dependent process, and a reduction in the maximal rate of LV relaxation would not be unexpected in patients with impaired myocardial perfusion.

However, our data suggest that the extent of LV dysfunction, independent of the severity and extent of coronary disease, is a major factor in the impairment in maximal negative dP/dt. Patients with more depressed maximal positive dP/dt, more reduced EF and with larger abnormally contracting segments had the greatest reduction in maximal negative dP/dt. Although these correlations do not imply a cause-effect relationship, it is likely that abnormal systolic performance and ACSs directly influence the rate of pressure fall during isovolumic diastole.

The abnormally contracting segment is an accepted method to quantitate akinesis or dyskinesis. Whether these segments represent scar tissue or ischemic but viable myocardium is still a controversial issue. If the abnormally contracting segment represents fibrotic scar tissue, a fall in maximal negative dP/dt would be anticipated as a result of the alteration in the viscoelastic properties of the left ventricle. However, if the akinetic or dyskinetic segment represents ischemic but viable and structurally normal myocardium, one would also expect a reduction in the peak rate of pressure fall during diastole, probably explained, as hypothesized by Waters et al., by dysynchronous wall motion in the ischemic zone dur-
ing isovolumic relaxation. These authors observed that the segment of LV wall rendered ischemic by partial occlusion of the left anterior descending artery exhibited less shortening during early systole, bulged during late systole and recoiled during early diastole, so that the ischemic segment would not contribute to the rapid fall of tension during early diastole, and could even subtract from it by recoiling. As seen in our patients, the larger the size of the abnormally contracting segment, the greater the alteration in maximal negative dP/dt anticipated.

There is a proportionately larger decrease in maximal negative dP/dt than in maximal positive dP/dt with abnormal LV function (table 2). This suggests that, as LV function deteriorates, the mechanisms responsible for peak rate of pressure fall during isovolumic diastole are more impaired than those responsible for peak rate of pressure rise during isovolumic contraction. These observations are in agreement with those reported by McLaurin et al.18 with pacing-induced ischemia in patients with CAD.

We found no relation between end-systolic volume and maximal negative dP/dt in the normal patients, and an inverse correlation between these two parameters in the group with abnormal LV function. The influence of end-systolic volume on the rate of LV relaxation is controversial, and the differing conclusions drawn by various authors10, 11, 18 may be in part related to different indices used to assess relaxation. Our observations do not support an independent influence of end-systolic volume in the maximal rate of pressure fall, and the inverse relation in patients with abnormal LV function can be explained by the fact that those patients with larger end-systolic volumes also had a more depressed systolic performance and diminished systolic fiber shortening.

We also found no significant correlation between peak LV pressure and maximal rate of pressure fall. Using the canine right-heart bypass preparation, Weisfeldt et al.18 have shown that peak LV pressure is a major determinant of peak LV pressure fall, and higher levels of peak systolic pressure resulted in substantial increase in negative dP/dt. We performed only baseline hemodynamic measurements in our patients and did not attempt to document changes in maximal negative dP/dt related to changes in peak systolic pressure in the same patient. It has also been shown, however, that maximal negative dP/dt can be altered significantly by other interventions, without any changes in peak LV pressure.8, 11 Our data suggest that in our patients, the possible effect of peak LV pressure on maximal negative dP/dt was outweighed by other factors.

The group of patients with normal LV function had a faster heart rate (81 ± 3 beats/min) (table 1) than those with abnormal function (73 ± 2 beats/min); this might have been related to digoxin and propranolol therapy in some patients in the latter group. This small difference in heart rate probably did not cause any changes in negative dP/dt, and in fact, one would predict that in the presence of coronary disease, slowing the heart rate would result in an increase, rather than a decrease, in negative dP/dt.13

Finally, negative dP/dt represents only one point on the isovolumic pressure curve, and does not necessarily predict the time-course or extent of relaxation, so that caution is needed when interpreting these results in relation to the entire phase of LV relaxation.

In summary, this study demonstrates that the maximal rate of LV relaxation, as assessed by maximal negative dP/dt, is impaired in patients with CAD and LV dysfunction. Our observations indicate that the extent of impairment is related to the severity of the dysfunction as determined hemodynamically by maximal positive dP/dt, and angiographically by EF and abnormally contracting segments. It also appears that in these patients peak relaxation rate is inversely related to end-systolic volume and is not related to peak LV pressure. Systolic performance and abnormal regional wall motion may be responsible for this alteration in LV relaxation.

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References

Quantitative Left Ventricular Wall Motion Analysis: A Comparison of Area, Chord and Radial Methods

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SUMMARY We were interested in devising a relatively simple quantitative technique that could be used on a routine clinical basis for wall motion analysis. Three quantitative methods of left ventricular (LV) regional analysis were compared in the 30° right anterior oblique and 60° left anterior oblique projections. The control group consisted of 17 patients with qualitatively normal LV wall motion; the abnormal group comprised 17 patients with at least one region of severe, qualitative wall motion abnormality. Normal regional values were determined for area, chord and radial methods by applying the techniques to the ventriculograms of the control group. Each technique was then applied to the abnormal group's ventriculograms to determine the percentage of qualitatively abnormal regions not detected by each method. The area method had the lowest failure rate (p < 0.001) and the best separation of measured normal and abnormal regions' ejection changes (p < 0.001), and best reflected symmetric uniform motion of the ventricular silhouette. We conclude that the area method, of the techniques examined, was best for the quantitative analysis of LV wall motion abnormalities.

LEFT VENTRICULAR contractile abnormalities can be an important manifestation of coronary artery disease. These wall motion changes may represent ischemia or infarction of myocardium.1-3 Quantifying the extent of regional wall motion abnormality may aid in determining the myocardial effects of coronary artery disease. It would also simplify analysis of wall motion changes after diagnostic and therapeutic interventions and would permit comparison of different imaging techniques to assess their diagnostic accuracy. Often, wall motion evaluation is done on a subjective basis; however, subjective, qualitative analysis has a substantial error rate. An objective, quantitative system is required for accurate regional evaluation.4 No quantitative technique is universally accepted, many of the more recent methods require sophisticated computer facilities,5 and normal segmental wall motion values have not been established for simultaneous 30° right anterior oblique (RAO) and 60° left anterior oblique (LAO) views. We evaluated three commonly used quantitative methods (chord, radial and area) of wall motion analysis to determine which technique best detects abnormal function. The methods chosen are relatively simple and therefore suitable for routine clinical use. The study was performed by analyzing simultaneous 30° RAO and 60° LAO projections of contrast left ventriculograms by each method.

Methods

Technically adequate cineventriculograms of patients evaluated for possible coronary artery disease were included in this investigation. Ventriculograms were performed using a General Electric 16 mm cinegraphic biplane system at 60 frames/sec with alternate firing of the cameras. The patient was positioned in the 30° RAO to the anterior-posterior tube and 60° LAO to the lateral tube. We performed left ventriculography using 0.8 ml/kg of Renografin-76 to a maximum dose of 60 ml injected in 4 seconds. After ventriculography, each patient had selective coronary angiography by the Judkins technique.
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