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Doppler Echocardiography in Cardiac Tamponade: Exaggerated Respiratory Variation in Transvalvular Blood Flow Velocity Integrals

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Pulsed Doppler echocardiography has been used previously to demonstrate marked changes in transvalvular blood flow velocities during cardiac tamponade in laboratory animals and a small number of patients. To further assess the respiratory changes in transvalvular blood flow during tamponade, pulsed Doppler tracings of flow velocity profiles across all four cardiac valves were recorded during inspiration and expiration in 13 patients during cardiac tamponade, in 6 of the 13 patients after relief of tamponade by pericardiocentesis and in 8 normal control subjects. Flow velocity integrals were calculated for each valve during inspiration and expiration.

In the setting of cardiac tamponade, inspiration caused an 85 ± 46% increase in the flow velocity integral across the pulmonary valve, an 81 ± 34% increase across the tricuspid valve, a 33 ± 13% decrease across the aortic valve and a 35 ± 8% decrease across the mitral valve. These phasic respiratory changes were markedly reduced after relief of tamponade ($p < 0.05$ compared with tamponade) and were observed to only a minimal extent in the normal individuals ($p < 0.01$ compared with tamponade). The exaggerated respiratory variations in transvalvular flow velocity integrals suggest that Doppler evaluation may be a valuable tool in the diagnosis of cardiac tamponade.

Transmitral Doppler indexes of left ventricular filling during cardiac tamponade revealed that inspiration caused a shift to increased filling during late diastole, with a greater contribution of atrial systole to total left ventricular filling. These Doppler indexes did not vary significantly with respiration in the group studied after relief of tamponade or in the control group. The change in these indexes during inspiration in cardiac tamponade is consistent with an inspiratory decrease in left ventricular compliance.

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Pulsus paradoxus, an inspiratory decrease in systolic blood pressure in excess of 10 mm Hg, is a frequent finding in cardiac tamponade. Pandian et al. (1,2) recently described a "flow velocity paradoxus" in cardiac tamponade. Using pulsed Doppler echocardiographic techniques in dogs with experimentally created tamponade and in a small number of patients with cardiac tamponade, they reported an increase in blood flow velocity across the tricuspid and pulmonary valves during inspiration and an inspiratory decrease in blood flow velocity across the mitral and aortic valves. The present study was undertaken to characterize more completely the respiratory changes in transvalvular blood flow in patients with cardiac tamponade.

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Methods

Study patients. Thirteen patients with clinical and echocardiographic signs of cardiac tamponade were evaluated with Doppler echocardiography. The five men and eight women had a mean age of 49 ± 17 years. Tamponade was subsequently confirmed in all patients by intracardiac and intrapericardial pressure measurements obtained during combined Swan-Ganz catheterization and needle pericardiocentesis. Six of the patients underwent a second Doppler examination within 48 h of relief of tamponade. Eight normal men with a mean age of 32 years and no evidence of cardiovascular disease by history, physical examination or two-dimensional echocardiography underwent Doppler examinations to serve as a control group.

Echocardiographic evaluation. Two-dimensional echocardiographic images and pulsed Doppler velocity signals were recorded on an ATL Mark 600 or a Hewlett-Packard (model 770 20A) combined imaging-Doppler echocardio-

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graph. During two-dimensional imaging, the size of the pericardial effusion was subjectively graded, and the presence and timing of right atrial (3) and right ventricular (4) collapse were determined.

Doppler velocity tracings of mitral transvalvular flow were recorded from the apical four chamber view, with the sample volume positioned at the level of the mitral anulus. Tricuspid flow velocity recordings were obtained at the level of the tricuspid anulus using a parasternal short-axis or apical four chamber view. Doppler tracings of aortic valve flow were obtained using the apical five chamber view, with the sample volume placed just beyond the valve leaflets within the proximal aortic root. Doppler tracings of pulmonary blood flow were obtained using the parasternal short-axis view, with the sample volume positioned just beyond the valve leaflets within the proximal pulmonary artery. The transducer position was held as constant as possible, and Doppler tracings were recorded throughout several respiratory cycles. Patients were given no instructions with regard to breathing and were unaware that inspiration and expiration were being monitored by a separate technician who observed the movement of the chest wall and marked the onset of inspiration and expiration by reversing the black-white color pattern on the video image. Still frames for analysis could be printed only in black on white format, but were chosen to represent the portions of the respiratory cycle indicated by the variable white and black backgrounds of the video images.

Doppler flow velocity calculations. Hard copy tracings of the pulsed Doppler velocity signals were printed and digitized with a graphics tablet (GTCO) and microcomputer (IBM PC) using custom-written software (Datastat). Digitization consisted of tracing the velocity curves along their darkest portion (modal velocity). From these curves, velocity signals were obtained every 4 ms, and the flow velocity integrals were calculated. The cardiac cycles analyzed during inspiration were those demonstrating the highest flow velocity across the tricuspid and pulmonary valves and the lowest flow velocity across the mitral and aortic valves. Similarly, the cardiac cycles analyzed during expiration were those demonstrating the lowest flow velocity across the tricuspid and pulmonary valves and the highest flow velocity across the mitral and aortic valves. The percent inspiratory change in flow velocity integral was then calculated as the difference between the flow velocity integral of the inspiratory beat and that of the next expiratory beat according to the formula:

$$\% \text{ Inspiratory change in flow velocity integral (FVI)} = \frac{(\text{Inspiratory FVI} - \text{Expiratory FVI}) \times 100}{\text{Expiratory FVI}}$$

For each of the four valves in each subject, 3 to 10 respiratory cycles were analyzed, and the mean percent inspiratory change in flow velocity integral was calculated for each valve in that subject. The mean percent inspiratory change in flow velocity integral for each valve was then calculated for the group as a whole. The transmitral Doppler velocity signals were subjected to further analysis to determine if the time course of left ventricular filling varied during the respiratory cycle. Peak early diastolic velocity (E velocity) and peak late diastolic velocity at the time of atrial systole (A velocity) were measured and the E/A velocity ratio was calculated. The integrated area under the A velocity triangle was measured, and the A area/mitral flow velocity integral ratio was calculated (Fig. 1). The E/A velocity ratios and the A area/mitral flow velocity integral ratios for the inspiratory and expiratory beats were compared in the three groups.

Statistical evaluation. Mean values and standard deviations were determined for all variables. Values for variables measured before and after relief of tamponade were compared using the Student's paired *t* test, and comparisons between groups were made using the unpaired *t* test. A p value of <0.05 was considered significant.

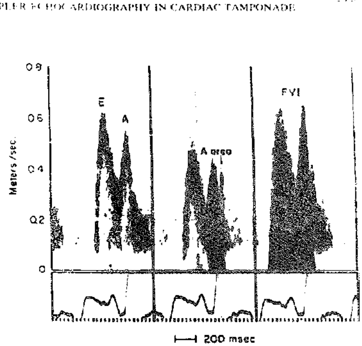


Figure 1. Pulsed Doppler transmitral waveforms, showing the measured Doppler indexes. A = peak late diastolic velocity at the time of atrial systole; A area = area under the A velocity curve; E = peak early diastolic velocity (in patients in whom the E and A areas overlapped, the A area was defined as the portion of the diastolic velocity integral over that expected if the slope of deceleration from the E point was extrapolated to the baseline); FVI = flow velocity integral, which is the area under the entire diastolic velocity curve.

Results

Clinical and hemodynamic characteristics (Table 1). The study patients consisted of 13 patients with a moderate or large pericardial effusion and echocardiographic signs of cardiac tamponade. All underwent right heart catheteriza-

Table 1. Hemodynamic Data for 13 Patients With Cardiac Tamponade

	During Tamponade	After Pericardiocentesis
Heart rate (beats/min)	106 ± 17	98 ± 18
Systolic pressure (mm Hg)	144 ± 45	150 ± 55
Right atrial pressure (mm Hg)	17 ± 7	8 ± 4
Pulmonary capillary wedge pressure (mm Hg)	18 ± 6	12 ± 6
Pericardial pressure (mm Hg)	16 ± 5	0 ± 3
Pulsus paradoxus (mm Hg)	20 ± 13	2 ± 4

tion and pericardiocentesis within several hours of the echocardiographic and Doppler examinations.

All patients had sinus rhythm. The most common cause of the pericardial effusion was malignancy. Other identifiable origins included uremia, radiation therapy and infection. Echocardiography revealed collapse of the right atrium in all patients and right ventricular collapse, persisting for more than 0.05 s after the opening of the mitral valve, in 8 (62%) of the 13 patients. Tamponade physiology was confirmed in all patients by the findings of elevation and equalization of right atrial, pulmonary capillary wedge and intrapericardial pressures. Successful and uncomplicated pericardiocentesis was performed in all patients, with removal of 700 ± 440 ml of pericardial fluid. The mean pulsus paradoxus of 20 mm Hg (range 0 to 35) during tamponade decreased to 2 mm Hg (range 0 to 10) after pericardiocentesis.

Doppler flow velocity integrals (Table 2). Flow velocity tracings adequate for analysis could not be obtained across all four valves in every subject. Tracings obtained during tamponade were suitable for evaluation of the aortic valve in 12 patients, for the mitral valve in 13 patients, for the pulmonary valve in 8 patients and for the tricuspid valve in 12 patients. In the six patients restudied after relief of tamponade, tracings adequate for analysis were obtained for the aortic valve in five patients, the mitral valve in five patients, the pulmonary valve in four patients and the tricuspid valve in six patients. Tracings from all four valves in each of the eight normal subjects were considered adequate for analysis.

The transvalvular Doppler flow velocity integrals during inspiration and expiration in the 13 patients with tamponade, 6 patients restudied after relief of tamponade and 8 normal control subjects are listed in Table 2. Examples of one patient's Doppler recordings across all four cardiac valves during inspiration and expiration, before and after pericardiocentesis, are shown in Figure 2.

During tamponade, inspiration was associated with an 85 ± 46% (range 47 to 198) increase in the flow velocity integral across the pulmonary valve and an 81 ± 34% (range 28 to 116) increase across the tricuspid valve. Flow velocity integrals across the left-sided valves decreased during inspiration, with a 33 ± 13% (range 11 to 64) decrease across the aortic valve and a 35 ± 8% (range 21 to 46) decrease across the mitral valve. The magnitude of the respiratory variations observed across the left-sided valves was less than that seen across the right-sided valves. Phasic respiratory changes were also observed in the normal control subjects, but the changes were minimal compared with those seen in the patients with tamponade.

In the six patients studied after relief of tamponade, some respiratory variation persisted, especially across the tricuspid and pulmonary valves, but it was significantly less than that noted during tamponade (Fig. 3). Only a single recording from the tricuspid valve of a patient restudied after pericardiocentesis showed a respiratory variation within the range observed in the patients with cardiac tamponade. Other than this single exception, there were no Doppler tracings from subjects restudied after pericardiocentesis or from control subjects that showed respiratory variation within the range observed in the patients with cardiac tamponade.

The degree of pulsus paradoxus did not correlate with the percent inspiratory decrease in the aortic valve Doppler flow velocity integrals. Two of the patients with cardiac tamponade had a pulsus paradoxus of <10 mm Hg on both physical examination and intraarterial pressure recordings. These two patients still showed exaggerated respiratory variations

Table 2. Doppler Flow Velocity Integrals (cm) Across the Four Cardiac Valves

	n	During Tamponade	n	After Pericardiocentesis	n	Normal Group
Pulmonary expir	8	6.3 ± 2.5	4	13.3 ± 3.8	8	14.6 ± 1.8
Pulmonary insp	8	11.2 ± 3.8*	4	14.7 ± 4.4†	8	15.9 ± 2.4†
Tricuspid expir	12	6.6 ± 1.4	6	8.7 ± 2.3	8	10.3 ± 1.3
Tricuspid insp	12	11.6 ± 2.1*	6	10.4 ± 2.2†	8	10.7 ± 1.4
Aortic expir	12	15.5 ± 6.9	5	13.5 ± 1.3	8	20.0 ± 2.6
Aortic insp	12	10.2 ± 4.6*	5	12.8 ± 1.3	8	19.4 ± 2.7†
Mitral expir	13	13.3 ± 4.8	5	14.1 ± 4.1	8	14.6 ± 1.5
Mitral insp	13	8.6 ± 3.4*	5	13.5 ± 4.1†	8	13.5 ± 1.6†

*p < 0.001 versus expiration (expir); †p < 0.05 versus expiration; n = number of patients in each group in whom Doppler recordings of transvalvular flow velocity integrals were adequate for analysis; insp = inspiration.

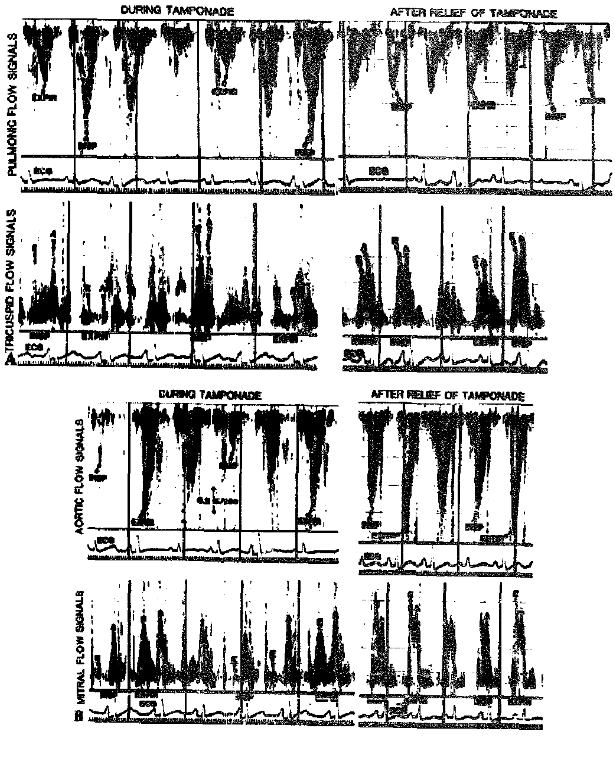
in transvalvular Doppler recordings and had elevation and equalization of intracardiac and intrapericardial pressures diagnostic of tamponade. The Doppler ultrasound and hemodynamic abnormalities resolved after pericardiocentesis.

Doppler indexes of left ventricular filling (Table 3). During tamponade, inspiration was associated with a decrease in the peak early/late diastolic (E/A) velocity ratio and an increase in the A area/mitral flow velocity integral ratio, reflecting a shift of left ventricular filling from early to late diastole. In normal control subjects and the patients restudied after pericardiocentesis, these Doppler indexes of left ventricular filling did not vary significantly during the respiratory cycle.

Figure 2. Pulsed Doppler recordings in a patient during and after relief of cardiac tamponade. A, Signals across the right-sided valves. B, Signals across the left-sided valves. In the transmitral recording during tamponade, inspiration (INSP) is associated with a prominent A (late diastolic velocity) wave and an attenuated E (early diastolic velocity) wave. ECG = electrocardiogram; EXPIR = expiration.

Discussion

The results of this study demonstrate that cardiac tamponade is associated with exaggerated respiratory variations in transvalvular Doppler flow velocity integrals. Doppler in-



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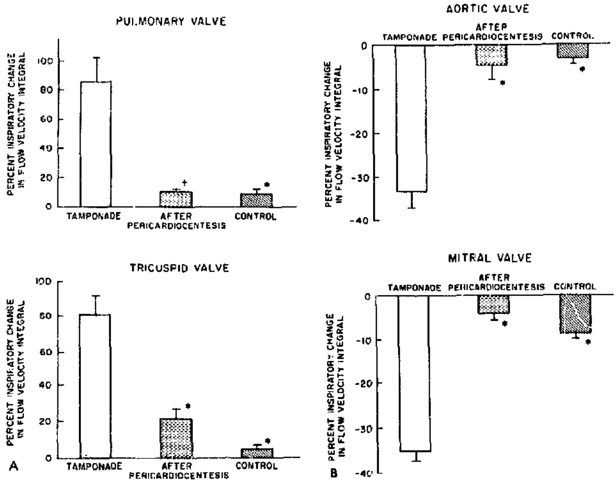

dexes of left ventricular filling also vary with respiration during tamponade, with a shift to greater dependence on atrial contraction during inspiration. These Doppler findings could serve as diagnostic signs of cardiac tamponade and may be particularly useful in settings in which movement of the right atrial and ventricular walls cannot be optimally assessed to detect collapse suggestive of tamponade or when fluid accumulation causing tamponade may be eccentric (for example, after cardiac surgery) (5).

Transvalvular flow in cardiac tamponade. This report extends the observations of Pandian et al. (1,2), who reported Doppler findings in three patients with cardiac tamponade. They noted that inspiration was associated with a 100% increase in peak flow velocity across the tricuspid valve and a 42% decrease in peak flow velocity across the mitral valve. Although we measured transvalvular flow velocity integrals rather than transvalvular flow, our findings are in accord with previous work (6–10) demonstrating that pulsus paradoxus during cardiac tamponade is associated with an exaggerated inspiratory increase in right-sided transvalvular flow and a decrease in left-sided transvalvular flow. In our study, the right-sided valves showed a greater respiratory variation in flow velocity integrals than did the left-sided valves. Previous echocardiographic studies in tamponade (11) indicated that the right ventricle has a greater percent variation in dimension

Figure 3. Inspiratory changes in Doppler flow velocity integrals across the (A) right-sided and (B) left-sided valves during cardiac tamponade and after pericardiocentesis and in control subjects. Data expressed as mean ± standard error. *p < 0.01 versus tamponade; †p < 0.05 versus tamponade.

with respiration than the left ventricle. The combination of these factors, therefore, suggests a far greater respiratory variation in blood flow across the right-sided valves than across the left-sided valves. Some right-sided variation in excess of that seen in normal subjects persists after relief of tamponade, and may be related to comorbid conditions other than pericardial disease, including metastatic pulmonary involvement and obstructive lung disease (10).

The data from our normal control group are consistent with those reported by Meijboom et al. (12). They evaluated Doppler recordings in four normal subjects, and reported that inspiration caused a small increase in blood flow velocity across the tricuspid valve and a corresponding decrease across the mitral valve.

Mechanism of pulsus paradoxus. In normal subjects, inspiration has been reported (13) to cause a 7% decrease in left ventricular stroke volume. The inspiratory decrease in left ventricular stroke volume is markedly increased in patients with cardiac tamponade (6,8,13). Using catheter-tipped electrodes to study vena caval blood flow velocity in normal subjects and patients with cardiac tamponade. Shabetai et al. (6) found that inspiration caused an increase in vena caval velocity in both groups. In a canine model of cardiac tamponade, Shabetai et al. (7,9) demonstrated that pulsus paradoxus depends on an inspiratory increase in right ventricular filling, and does not occur when venous return to the right heart is held constant throughout the respiratory cycle.

The mechanism of pulsus paradoxus in cardiac tamponade is complex and multifactorial (14–18). With inspiration, negative intrathoracic pressure is transmitted to the pericardial space, and intrapericardial and right atrial pressures decrease, resulting in an increase in flow from the venae cavae into the right heart chambers. This inspiratory increase in venous return causes an increase in right ventricular size. Because the total intrapericardial space is relatively fixed during tamponade, an increase in right ventricular volume causes a corresponding decrease in left ventricular volume. The interventricular septum is shifted to the left (19), resulting in a decrease in left ventricular compliance, left ventricular diastolic filling and, subsequently, inspiratory left ventricular stroke volume. Thus, during inspiration, right-sided flow increases at the expense of left-sided flow, and this respiratory variation is reflected in the Doppler tracings. Other factors, including inspiratory pooling of blood in the pulmonary vascular bed and an inspiratory increase in left ventricular afterload, are also thought to contribute to pulsus paradoxus (20).

Changes in left ventricular compliance. An important mechanism of pulsus paradoxus during cardiac tamponade is considered to be an inspiratory decrease in left ventricular compliance, mediated partially by a leftward shift of the interventricular septum. Previous echocardiographic studies (11,21) of cardiac tamponade reported an inspiratory decrease in the mitral valve EF slope, which may also reflect a decrease in left ventricular compliance. In this study, Doppler transmitral indexes of left ventricular filling, consisting of a decrease in the peak early/late diastolic (E/A) velocity ratio and an increase in the A area/mitral flow velocity integral ratio, also suggest a decrease in left ventricular compliance with inspiration during tamponade. Similar changes in Doppler indexes of early and late diastolic filling have previously been reported in other conditions where left ventricular compliance is decreased (22), including pathologic left ventricular hypertrophy (23), myocardial ischemia (24) and normal aging (25).

Table 3. Ratio of Peak Velocity During Early Diastole to Peak Velocity During Atrial Systole and of Area Under the Velocity Curve of Atrial Systole to Mitral Flow Velocity Integral

	During Tamponade (n = 13)	After Pericardiocentesis (n = 6)	Normal Group (n = 8)
E/A expir	1.18 ± 0.52	2.07 ± 0.71	1.82 ± 0.47
E/A insp	0.96 ± 0.44*	1.76 ± 0.81	1.73 ± 0.51
A area/FVI expir	0.32 ± 0.12	0.21 ± 0.06	0.23 ± 0.07
A area/FVI insp	0.41 ± 0.18*	0.23 ± 0.07†	0.22 ± 0.07†

*p < 0.05 inspiration versus expiration in each group; †p < 0.05 inspiratory values in patients after pericardiocentesis and in normal subjects versus inspiratory values in patients during tamponade. A = peak velocity during atrial systole; A area = area under the velocity curve of atrial systole; E = peak velocity during early diastole; FVI = flow velocity integral; other abbreviations as in Table 2.

Limitations of the study. One assumption of quantitative Doppler echocardiography is that the Doppler interrogation beam is aligned parallel to blood flow. A potential limitation of this study is that excessive swinging of the heart within a large pericardial effusion may have changed the angle between the direction of blood flow and the Doppler beam in some patients during the cardiac and respiratory cycles. However, because all patients showed reproducible changes that were in the same direction and of such significant magnitude, it is unlikely that this potential factor would alter the conclusions of the study.

Another limitation of our study is that we assessed velocity integrals rather than total transvalvular flow. To quantitate transvalvular flow, the flow velocity integral must be multiplied by the cross-sectional area of the valve orifice (26–28). In this study, the actual volume of transvalvular flow was not quantitated because the cross-sectional areas of the valve orifices were not measured. In most patients, a slight variation in cardiac position during the cardiac and respiratory cycles precluded optimal measurements of inflow and outflow areas. Settle et al. (11) and others (20) reported echocardiographic measurements during cardiac tamponade, demonstrating that inspiration produces a significant decrease in left ventricular internal dimension and a significant increase in right ventricular internal dimension. Similar studies have also documented an inspiratory decrease in mitral valve diastolic area and aortic valve systolic area (11,29) and an inspiratory increase in pulmonary artery diameter (14). Thus, although we did not measure inflow and outflow areas, which would have permitted calculation of actual transvalvular flow, previous evidence suggests that inspiration is associated with an increase in the area of right-sided valves and a decrease in the area of left-sided valves. The actual respiratory variation in transvalvular blood flow, therefore, would likely be greater than the respiratory variation in flow velocity integrals reported here.

Finally, this study did not address the specificity of the respiratory changes in flow velocities or flow velocity integrals. Hoit et al. (30) reported a similar "flow paradoxus" of respiratory origin in a patient with pulsus paradoxus and severe obstructive lung disease, suggesting that exaggerated respiratory changes in intrathoracic pressure might produce similar findings. It remains to be determined whether patients with pulsus paradoxus found in association with other illnesses (10), including asthma and acute pulmonary embolism, may also demonstrate changes in flow velocities and flow velocity integrals mimicking those of tamponade.

Conclusions. Cardiac tamponade is associated with exaggerated respiratory variation in transvalvular Doppler flow velocity integrals. These phasic respiratory changes are markedly reduced after relief of tamponade and are observed to only a minimal extent in normal subjects. Doppler echocardiography may, therefore, be a valuable tool in the diagnosis of cardiac tamponade, particularly in patients in whom the diagnosis is uncertain clinically and in whom imaging of the right atrial and right ventricular walls to detect collapse, characteristic of tamponade, is difficult. In addition, the changes in morphology of the mitral flow velocity profiles, suggesting an inspiratory decrease in left ventricular compliance, may assist in further understanding the mechanisms associated with pulsus paradoxus.

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