

A Mechanism for L-Wave Generation via Color M-Mode Imaging in a Patient with Mitral Regurgitation



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INTRODUCTION

Diastasis is the quiescent period of diastole between the early and late filling of the heart. However, an additional flow called an “L wave” may be present during this period in some patients.¹ In the present report, we describe examination of the L wave using color M-mode imaging in a patient with severe mitral regurgitation (MR) and propose a mechanism for this flow.

CASE PRESENTATION

An 82-year-old woman who had been treated for hypertension was brought to our hospital by ambulance because of severe dyspnea. She had felt exertional dyspnea for several days before admission. Physical examination revealed blood pressure of 182/114 mm Hg and a pulse rate of 114 beats/min. Auscultation showed expiratory wheezing at the bilateral lung field and systolic heart murmur at the apex and aortic area. Her oxygen saturation level measured using a pulse oximeter was 98% with inhalation of 10 L/min of oxygen from a reservoir mask. Analysis of a blood sample revealed mild liver injury and a high plasma brain natriuretic peptide level (359 pg/mL). Chest radiography revealed pulmonary congestion, with a cardiothoracic ratio of 66% (Figure 1, left). Twelve-lead electrocardiography showed transient atrial fibrillation that became a sinus rhythm the following day, high amplitude of the R wave in lead V₅, and negative T wave in leads V₂ to V₅ (Figure 1, right). Transthoracic parasternal two-dimensional echocardiography showed a left atrial (LA) enlargement of 52 mm. The thicknesses of the interventricular septum and left ventricular (LV) posterior wall were both 9 mm. The LV end-diastolic dimension, end-systolic dimension, and ejection fraction were 52 mm, 33 mm, and 66%, respectively. There was a flail mitral valve (MV) at the posterior leaflet, suggesting rupture of the mitral chordae (Figure 2, left). Color flow imaging revealed a flow of severe MR directed toward the LA anterior wall (Figure 2, right). Pulsed Doppler echocardiography at the mitral inflow showed short E-wave deceleration time (137

msec) and an obvious mid-diastolic L wave (Figure 3, arrows). A color M-mode recording along the line with mitral inflow revealed abnormal intra-atrial flow during mid-diastole. The early rapid filling flow was abruptly interrupted and then immediately restarted from the LA base. This flow propagated rather slowly and formed an L wave (Figure 4, arrows).

After treatment for congestive heart failure with diuretics, the patient underwent cardiac catheterization. A hemodynamic study showed a cardiac index of 2.1 L/min/m² and mean pulmonary capillary wedge pressure of 6 mm Hg. The results of coronary angiography and left ventriculography showed a normal coronary artery and normal LV wall motion with Sellar's grade III MR. She underwent surgical MV repair. Postoperative color flow imaging confirmed the disappearance of MR. M-mode echocardiography showed mildly restricted opening of MV and disappearance of the L wave (Figure 5A). Pulsed Doppler echocardiography at the mitral inflow showed increased E-wave deceleration time (342 msec) and disappearance of the L wave (Figure 5B). Continuous Doppler echocardiography showed mitral resistance increased by showing an estimated MV area of 2.6 cm² and a mean diastolic pressure gradient of 0.6 mm Hg without mitral stenosis.²

DISCUSSION

In the present case of severe MR, pulsed Doppler echocardiography at the mitral inflow showed an L wave. Keren *et al.*¹ first described the L wave as mid-diastolic flow after the E wave. They demonstrated that the L wave appeared in the condition of normal or high LV stiffness and small MV resistance, when a transient negative and then positive atrioventricular pressure gradient occurred after large rapid filling wave. They also considered that pulmonary venous flow contributed to the L wave. In the present case of severe MR, color M-mode echocardiography revealed an intriguing flow supporting their theory: an early rapid filling E wave was suddenly interrupted by an abrupt elevation of the early diastolic LV pressure and immediately restarted from the LA base because of a decrease in LV pressure. A gradual decrease in LV diastolic pressure resulted in a slow propagation in the LA cavity, causing a significant delay from the E wave and resulting in an L wave. Thus, the early diastolic E wave was divided into two parts, the latter of which formed the L wave. In the present case, although the pulmonary venous flow was not recorded, massive pulmonary venous flow due to the reservoir function of the pulmonary vasculature due to severe MR and LA conduit function may have caused the diastolic LA flow. Ghosh *et al.*³ proposed another mechanism of the L wave related to the LV diastolic intraventricular vortex. They observed that the early diastolic recirculating vortex ring entrained some blood at the MV and created the L wave. In the present case, massive rapid

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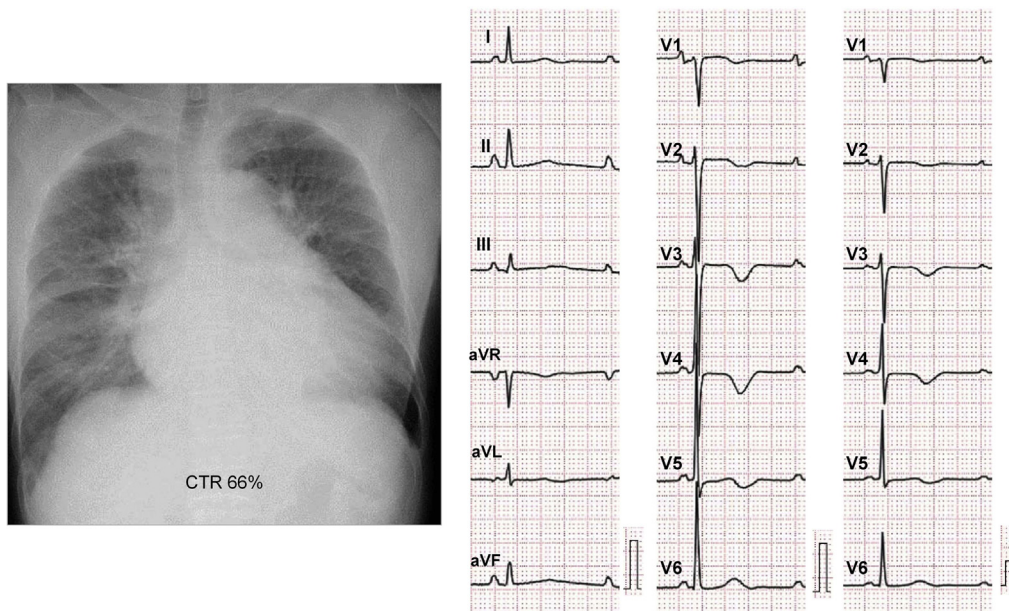


Figure 1 (Left) A chest radiograph showing pulmonary congestion with a cardiothoracic ratio of 66%. (Right) A 12-lead electrocardiogram taken the next day showing high amplitude of the R wave in lead V₅ and negative T wave in leads V₂ to V₅.

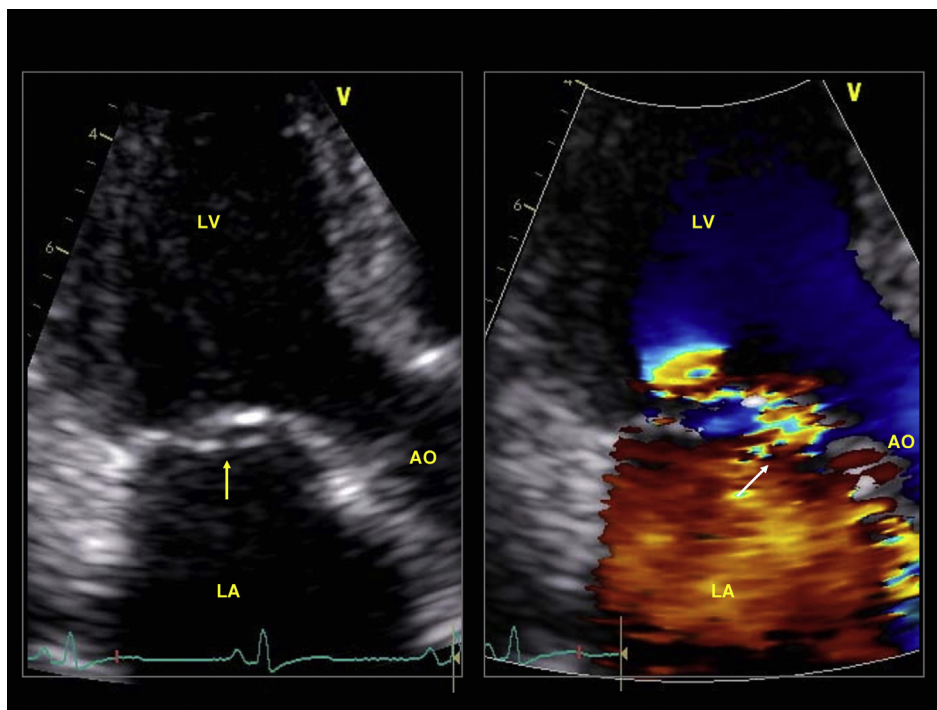


Figure 2 (Left) Apical long-axis two-dimensional echocardiography showing a flail MV at the posterior leaflet (arrow). (Right) Color flow imaging in the same view showed a severe mitral regurgitant flow directed toward the LA anterior wall (arrow).

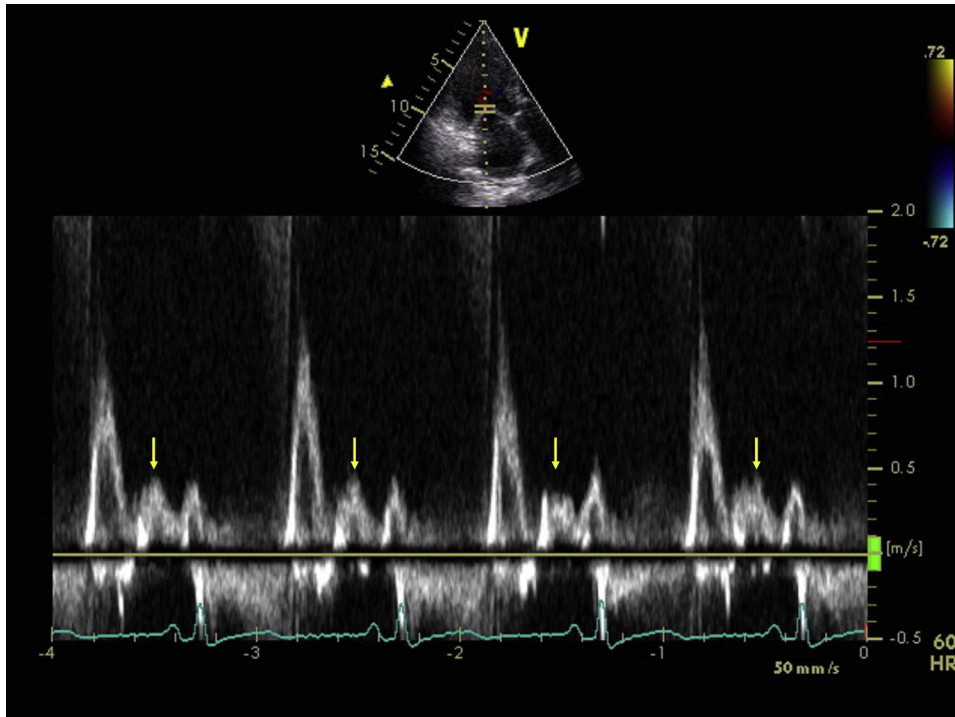


Figure 3 Pulsed Doppler echocardiography at the mitral inflow showed short E-wave deceleration time (137 msec) and an obvious mid-diastolic L wave (arrows).

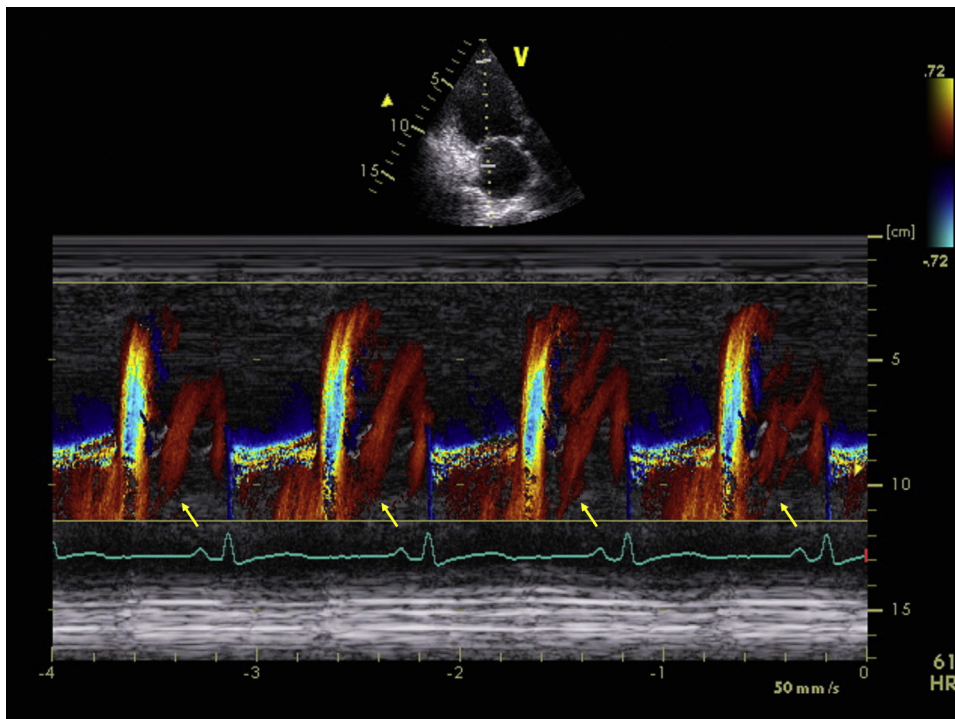


Figure 4 A color M-mode recording along the line with mitral inflow revealing an intra-atrial flow during the mid-diastole. The early rapid filling flow was abruptly interrupted and then immediately restarted from the LA base. This flow propagated rather slowly and formed the L wave (arrows).

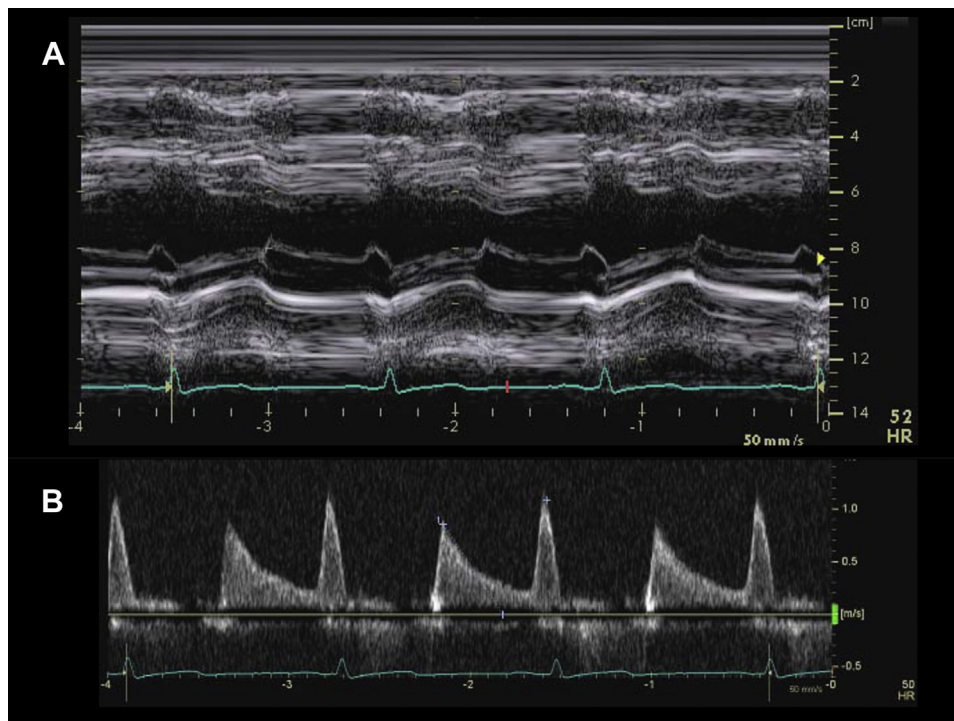


Figure 5 Transthoracic echocardiography after MV repair. **(A)** M-mode echocardiography showed mildly restricted opening of MV and disappearance of the L wave. **(B)** Pulsed Doppler echocardiography at the mitral inflow showed increased E-wave deceleration time (342 msec) and disappearance of L wave.

filling wave due to MR might have caused increased recirculating vortex and L wave.

After MV repair, although there was no significant mitral stenosis, mitral resistance increased. It is possible that restricted MV opening¹ as well as decreased pulmonary venous flow extinguished the L wave after MV repair.

CONCLUSION

This case report demonstrates the precision and usefulness of color Doppler M-mode echocardiography in examining the L wave.

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