

One-Dimensional Echocardiography: Still Needed or Already Outdated?

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There is a widespread lack of knowledge about one-dimensional echocardiography (M-mode) by the current generation of echocardiographers. Has two-dimensional echocardiography (B-mode) and Doppler made M-mode outdated? M-mode provides information that cannot be obtained by using B-mode or even Doppler—be it spectral, color, or tissue Doppler—for two reasons:

- 1) M-mode provides temporal resolution of a structure at 2,000 frames per second, whereas B-mode provides only 30–100 frames per second. Sudden movement or amplitude definition is compromised on B-mode, but not on M-mode;
- 2) Doppler defines the velocity of the flow, whereas the temporal variations of a structure visualized via M-mode show the volumetric changes of that flow.

Now let us consider some conditions that can only be detected by M-mode and not by B-mode or Doppler, which can aid in the diagnosis via B-mode or in the understanding of pathologies.

In **left ventricular (LV) diastolic dysfunction**, approximately 70% of LV filling occurs in the first third of diastole, the so-called rapid filling period of the LV. In impaired relaxation, this initial flow is slowed, and ventricular filling occurs predominantly in the last two-thirds of diastole at the expense of atrial contraction.^{1,2} M-mode continuously displays the left atrium (LA) in a left parasternal section behind the aorta from the end of LV ejection, when it is at its largest, to after atrial contraction, when it is at its smallest diameter. In this way, it is possible to determine whether LA emptying (and thus LV filling) occurs mainly in the first-third of diastole or in the last two-thirds. If it occurs in the last two-thirds, the rapid filling phase of the LV is absent, which suggests an impaired relaxation. This is also seen in the region where the posterior valve merges with the mitral annulus, forming a prominence between the ventricular and atrial walls. This elevation, seen on M-mode, is pulled toward the apex during systole and returns to its original position at the end of diastole. This return occurs as the LV relaxes. If it returns less in the first third and more in the last two-third, LV relaxation is impaired. The key point is not the rate of return (detectable

by tissue Doppler imaging), but rather the percentage of displacement indicative of LV cavity filling, taking as reference the amplitude of this systolic displacement in the patient rather than a value from a studied population. If relaxation is defined as abnormal on M-mode, it is considered pseudonormal if Doppler appears normal.

As LV end-diastolic pressure increases, the mitral valve closing motion may show an interruption of this displacement, forming a plateau (point B).³

In **LV systolic dysfunction**, there is a separation of the mitral E point from the interventricular septum (IVS) greater than 6 mm, or a “tilted pine tree” appearance.³ Aortic valve tends to close even during ejection at low output. These details are not captured by the low temporal resolution of B-mode.

Left bundle branch block (LBBB)^{4,5} alters the motion of the IVS. In the pre-ejection period, the IVS has a rapid posterior motion that occurs before the posterior wall moves. This is followed by a wide anterior displacement during ejection. At the end of ejection, the septum moves back posteriorly.

Type B **Wolff-Parkinson-White (WPW)**⁶⁻⁸ is characterized by abnormal septal motion similar to LBBB. In type A WPW, the IVS moves normally, and the posterior wall has premature contraction followed by normal anterior motion during the ejection phase. Relaxation of this wall may occur in two phases: an early phase and a late one.

Proper identification of chordae tendineae is important because they can be confused with the posterior wall endocardium if they originate from this wall, leading to underestimation of the ventricular cavity diameter and overestimation of the posterior wall thickness. On M-mode, the chordae tendineae have a smaller amplitude of systolic motion than the endocardium, resulting in a less steep trace compared to the endocardium. Thus, at the end of systole, the chordae tendineae are closer to the endocardium, where they may even touch; in diastole, they move apart. Despite these differences, it may not be possible to correctly separate these structures using B-mode.

In **right ventricular (RV) volume overload**,⁹ the IVS moves anteriorly into the RV cavity during systole, as opposed to its usual motion, which characterizes **paradoxical motion**. Paradoxical motion is better detected with M-mode than with B-mode.

In **pulmonary arterial hypertension**,¹⁰ A-wave disappears on pulmonary valve M-mode, so even in patients without tricuspid regurgitation, pulmonary artery systolic pressure can be assessed to indicate pulmonary hypertension.

In **coronary artery disease**, localized abnormalities of ventricular contraction in any wall cause diastolic tension

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to persist in that area for longer than in other regions of the myocardium. This causes the affected area to move inward into the LV cavity during the isovolumetric relaxation phase.^{11,12} Since both the aortic and mitral valves are closed during isovolumetric relaxation, the volume of the ventricular cavity cannot change; hence, corresponding to the inward movement of the affected area, other regions of the uninvolved walls expand to maintain the volume. This expansion causes the ventricular cavity to acquire a more circular configuration before the mitral valve opens, primarily through dilation of the basal regions of the LV. Consequently, valve opening occurs well after the posterior wall begins to move. Normally, valve opening and this movement occur almost simultaneously.

Acute aortic insufficiency¹³ results in premature closure of the mitral valve. Whereas in the chronic form, the LV gradually adapts to the increased diastolic volume, the acute form causes a significant increase in LV diastolic pressure, thus closing the mitral valve during diastole prior to the QRS complex. Acute aortic insufficiency is significant because, in chronic cases, the valve closes at its normal time irrespective of the severity of regurgitation.

In **mitral stenosis**, the more severe the stenosis, the greater the reduction in E to F slope velocity of the anterior valve.¹⁴⁻¹⁶ The presence of an anterior valve A-wave are a good indication that the stenosis is mild.

In **mitral prolapse**, in addition to the classic prolapse seen on M-mode, there are indirect signs that can help make this diagnosis in case of doubt on B-mode. One of these signs is the very posterior position of the mitral valve at the beginning of systole, intertwining with the posterior wall of the LV and preventing a perfect systolic visualization of the valves.¹⁷ Another indirect sign is the **atypical systolic anterior motion of the mitral valve**,¹⁸ in which there is a disproportion between the length of the chordae tendineae and the length of the long axis of the LV cavity due to abnormal chordae elongation. Under these conditions, a portion of the chordae remains during systole and projects into the LV outflow tract due to the Venturi effect. In most cases where this type of motion is seen on M-mode, B-mode will show mitral valve prolapse or, on repeat study at another time, the classic pattern of valve prolapses.

In **ruptured chordae**, short diastolic vibrations are observed in only a localized area of the mitral valve, during its opening detectable only on M-mode.¹⁹⁻²¹

Systolic vibrations of the mitral valve may indicate chordae rupture secondary to infectious endocarditis.²²

In **papillary muscle dysfunction**, the valve appears normal or “**tilted pine tree**” on M-mode. This indicates LV dysfunction, not specifically papillary muscle dysfunction.¹⁷

The appearance of **endocarditis vegetations** on M-mode is that of localized thickening only in certain areas of the valve, with a fluffy characteristic, as if the image is blurred. The edges of these thickenings have an irregular appearance like torn paper.^{17,23,24} These two signs aid in the differential diagnosis with calcification or fibrosis, which appear as a strong, bright trace with smooth edges on

M-mode. Because of its lower resolution, B-mode does not allow for these differentiations. In our experience, **Lambd's excrescences** can also be confused with vegetations on B-mode, but not on M-mode. Similar clinical scenarios include **suture threads on prosthetic valve rings**, mild **calcifications or degenerative valve thickening**, and **papillary fibroelastomas**.

In **dilated cardiomyopathy**, the M-mode of the aorta and aortic valve shows changes because of the decreased ejection volume found in these cases.^{25,26} The aortic walls move very little during ejection and the valves have a reduced opening. In more severe cases, the aortic valve tends to gradually close during systole because of the small ejection volume.

In **hypertrophic cardiomyopathies** with a subaortic gradient, the mitral valve moves toward the IVS immediately after the onset of systole and enters the LV outflow tract, which is characterized by the so-called **anterior systolic motion of the mitral valve (SAM)**.^{27,28} Patients can be classified into three categories based on the presence and intensity of SAM:

1) Patients with severe SAM, where it touches the IVS: These patients have a subaortic gradient.

2) Patients with mild SAM: There is no contact between the SAM and the IVS. There is no gradient in the LV, but SAM becomes more pronounced due to increased subaortic obstruction that occurs after maneuvers or certain medications.

3) Patients without SAM: These patients do not have subaortic obstruction.

Patients with mitral prolapse may have SAM.¹⁸ In such patients, SAM can be distinguished from hypertrophic cardiomyopathy because the anterior valve does not retract at the end of systole. The M-mode of the aortic valve is altered in approximately one-third of patients with hypertrophic cardiomyopathy.²⁹ The aortic valves may show midsystolic closure followed by late systolic reopening; however, random movements of closure and reopening of these valves throughout the ejection phase are equally common. Midsystolic closure of the aortic valve is always associated with a subaortic gradient.³⁰

For **prosthetic valves**, M-mode enhances the ability to correlate events with the time factor and, because of its higher repetition frequency compared to B-mode, allows for a more detailed analysis of the fine movements of mechanical prostheses.

In **constrictive pericarditis**, there is rapid motion of the IVS into the LV during early diastole with a characteristic sharp V-shaped appearance.³¹ During inspiration, there is dilation of the RV and reduction of the LV.

As the diastolic pressures of the RV and pulmonary artery equalize, the pulmonary valve may open early, partially or fully before the QRS complex.

On M-mode, **fixed membranous subaortic stenosis** shows pathognomonic abnormalities of the aortic valve.^{32,33} After opening, the aortic valve closes prematurely and remains semi-closed for the remainder of systole. This helps

to differentiate fixed membranous subaortic stenosis from hypertrophic cardiomyopathy, in which the closure occurs midsystolic and the valve reopens at the end of systole.

In **pulmonary stenosis**, there is low pressure in the pulmonary artery and a strong right atrial contraction that raises RV end-diastolic pressure above pulmonary artery pressure, opening the pulmonary valve before ventricular ejection.³⁴

In **infundibular stenosis**, the M-mode of the pulmonary valve RV shows systolic vibrations that extend for a long time

into diastole.³⁵ These oscillations are thought to be due to the turbulent jet impinging on the valves. The A-wave has normal amplitude in mild obstruction and disappears in severe obstruction.

M-mode suggests the presence of **Ebstein anomaly** because the tricuspid valve is more easily visualized than the mitral valve. The tricuspid valve shows delayed closure compared to the mitral valve,^{36,37} which may also be seen in a **ruptured sinus of Valsalva aneurysm** into the right atrium.³⁸

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