Echo in Cardiomyopathy

January 22, 2008

Joe M. Moody, Jr, MD
UTHSCSA and STVAHCS

Connolly HM and Oh, JK. Ch 14 in Braunwald's 8th ed. 2008
Ch. 17 in Feigenbaum's 6th ed. 2005

Outline

- Echo is Critically Important in Diagnosis
  - Dilated Cardiomyopathy
  - Hypertrophic Cardiomyopathy
  - Restrictive Cardiomyopathy
  - Arrhythmogenic RV Cardiomyopathy
  - Ventricular noncompaction
- Echo in Prognosis

Echo Features in Dilated Cardiomyopathy

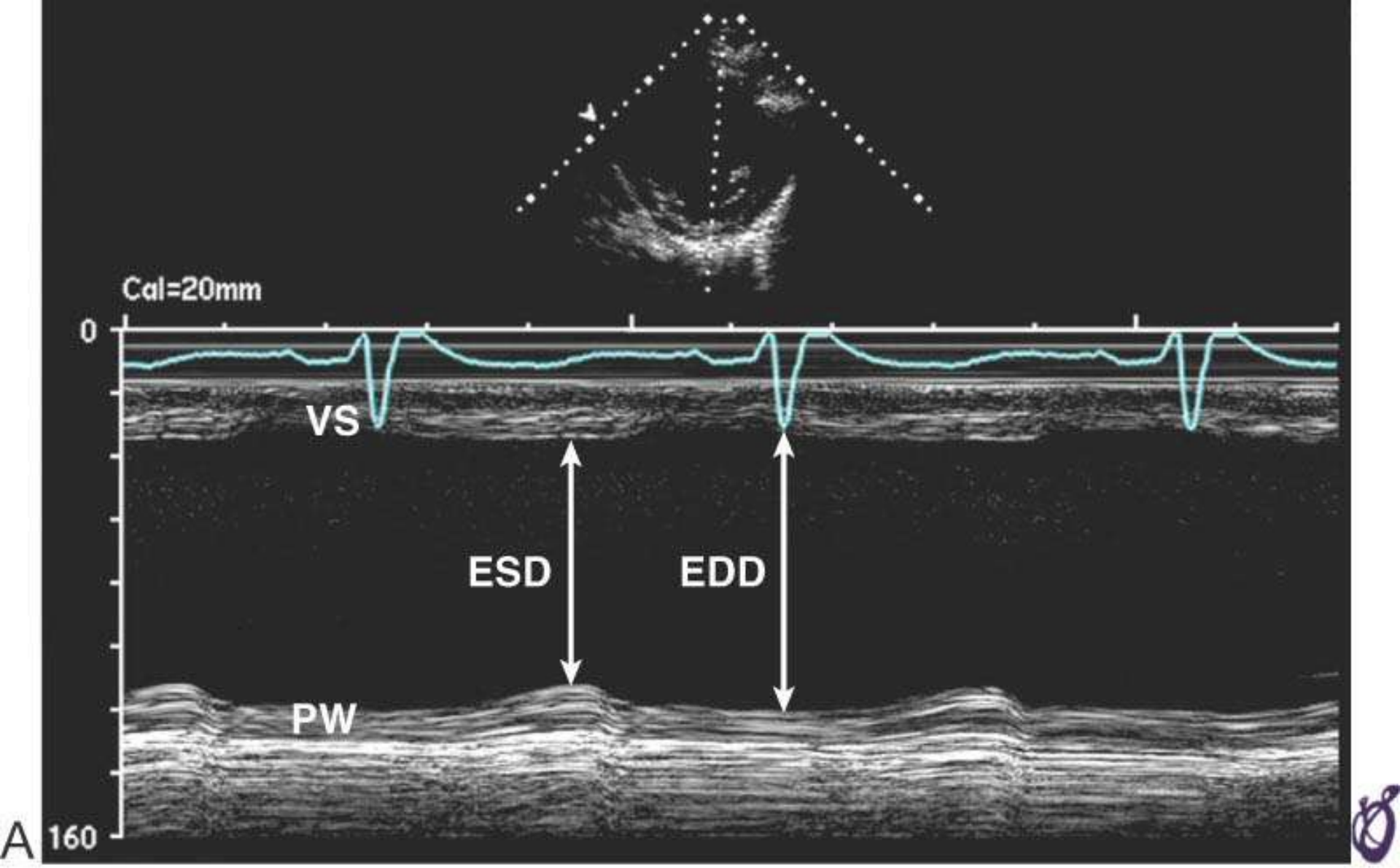
- Increased LV diastolic and systolic volumes
- Decreased LV systolic function
- Increased sphericity (long-axis dimension/short-axis dimension usually >1.5, but approaches 1.0 in DCM)
- Increased LV mass (eccentric LVH)
- Normal wall thickness
- Regional wall motion abnormalities can be present

Secondary Echo Findings in Dilated Cardiomyopathy

- Mitral Annular Dilation
- Evidence of Low cardiac output (decreased MV excursion)
- Atrial enlargement
- RV enlargement
- Apical thrombus
- Intraventricular mechanical dyssynchrony

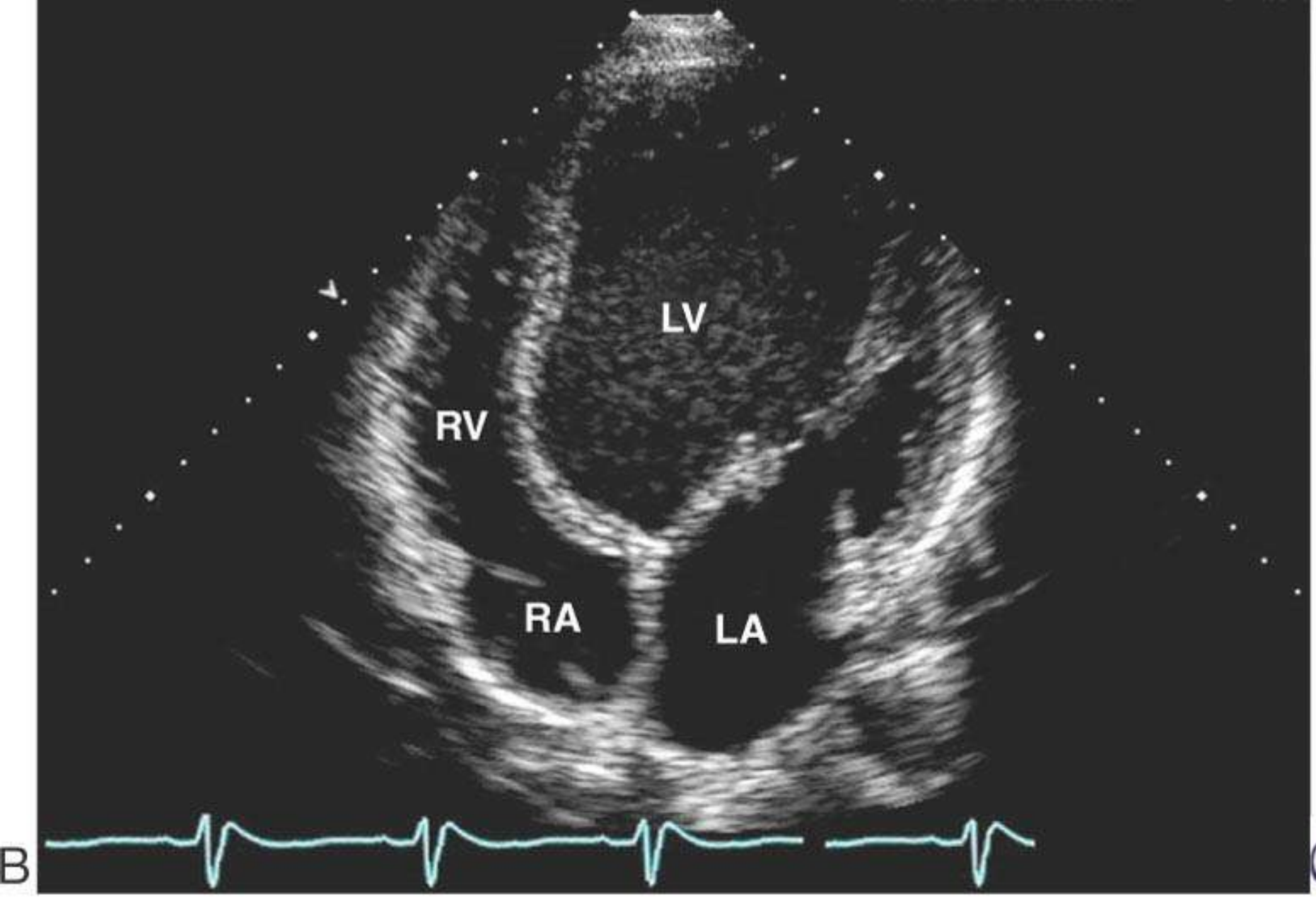
Doppler Findings in DCM

- Cardiac Output
  - SV times HR
  - SV = (VTI of LVOT) *(CSA of LVOT)
  - SV is somewhat technically demanding
- LV filling pressures (diastolic function)
  - Prognostic information
- TR velocity > 3 m/s adverse prognosis



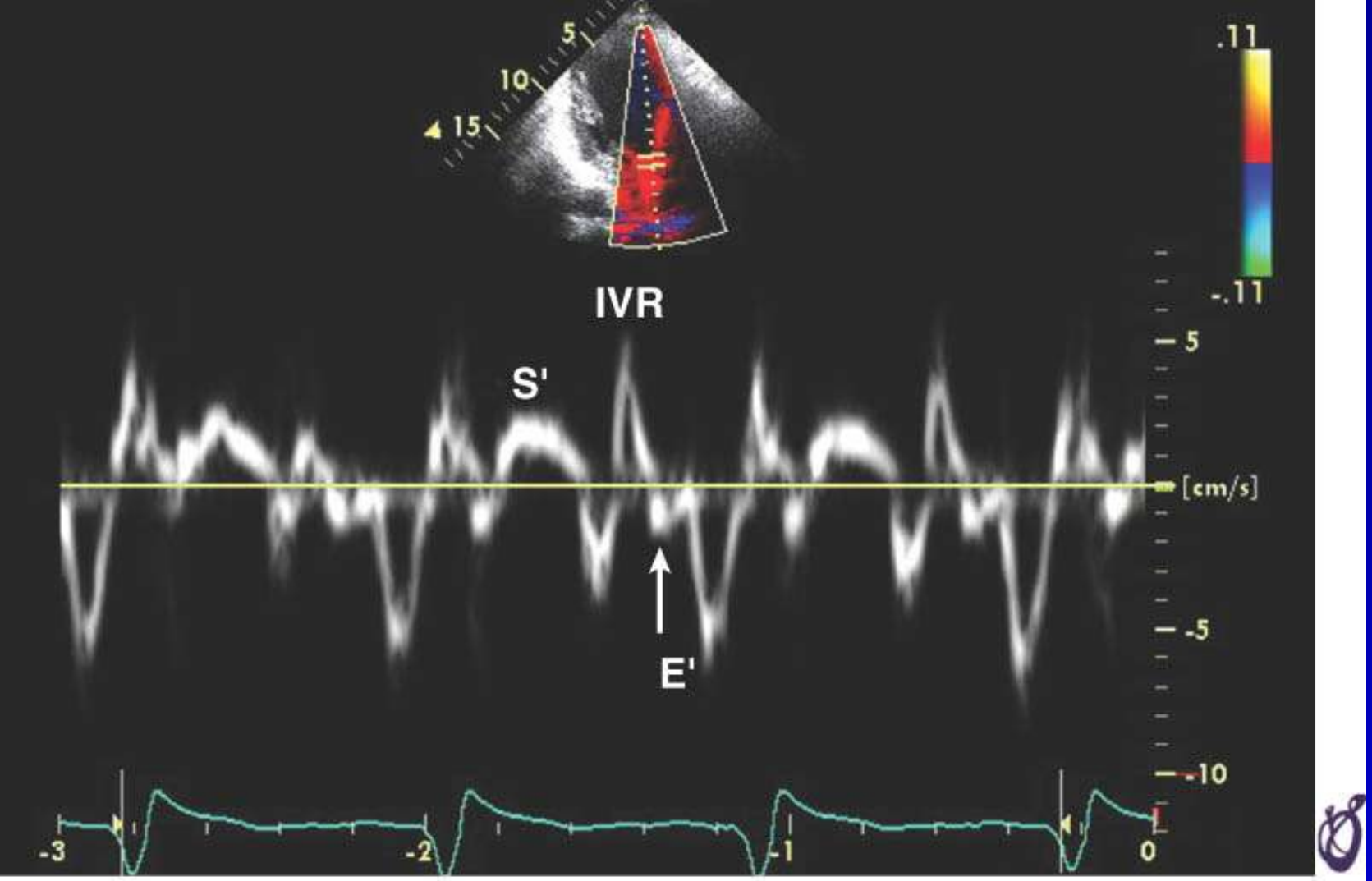
(From Oh JK, Seward JB, Tajik AJ: The Echo Manual. 3rd ed. Philadelphia, Lippincott Williams & Wilkins, 2006. Used with permission of Mayo Foundation for Medical Education and Research.)

Fig 14-71A, Braunwald's 8th ed.



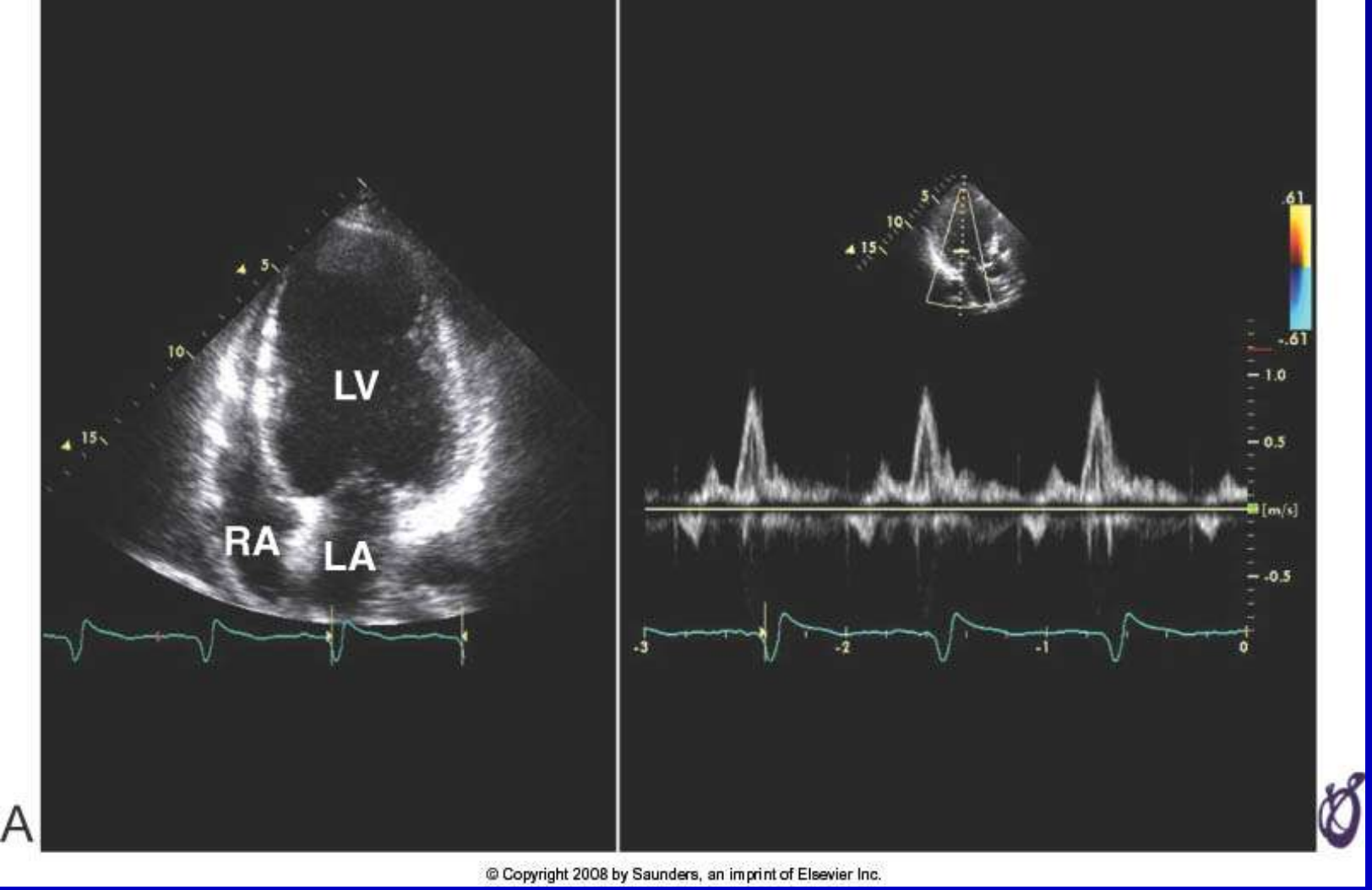
(From Oh JK, Seward JB, Tajik AJ: The Echo Manual. 3rd ed. Philadelphia, Lippincott Williams & Wilkins, 2006. Used with permission of Mayo Foundation for Medical Education and Research.)

Fig 14-71B, Braunwald's 8th ed.



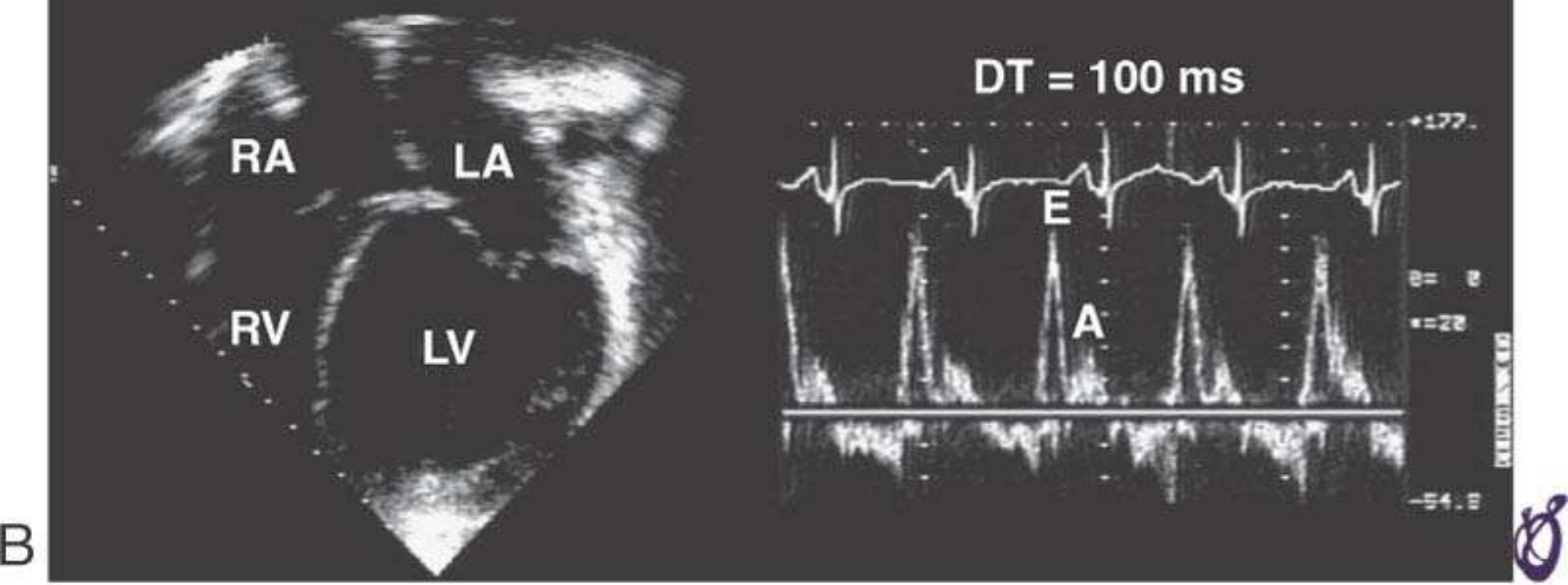
Tissue Doppler recording of the septal mitral annulus showing decreased systolic velocity (*S'*), early diastolic velocity (*E'*), and prominent bidirectional velocities during isovolumic relaxation (*IVR*)

Fig 14-72, Braunwald's 8th ed.



Dilated ischemic cardiomyopathy showing a relaxation abnormality pattern with increased A velocity. Patients with this type of diastolic filling pattern usually have minimal to mild symptoms, despite severe left ventricular (LV) systolic dysfunction.

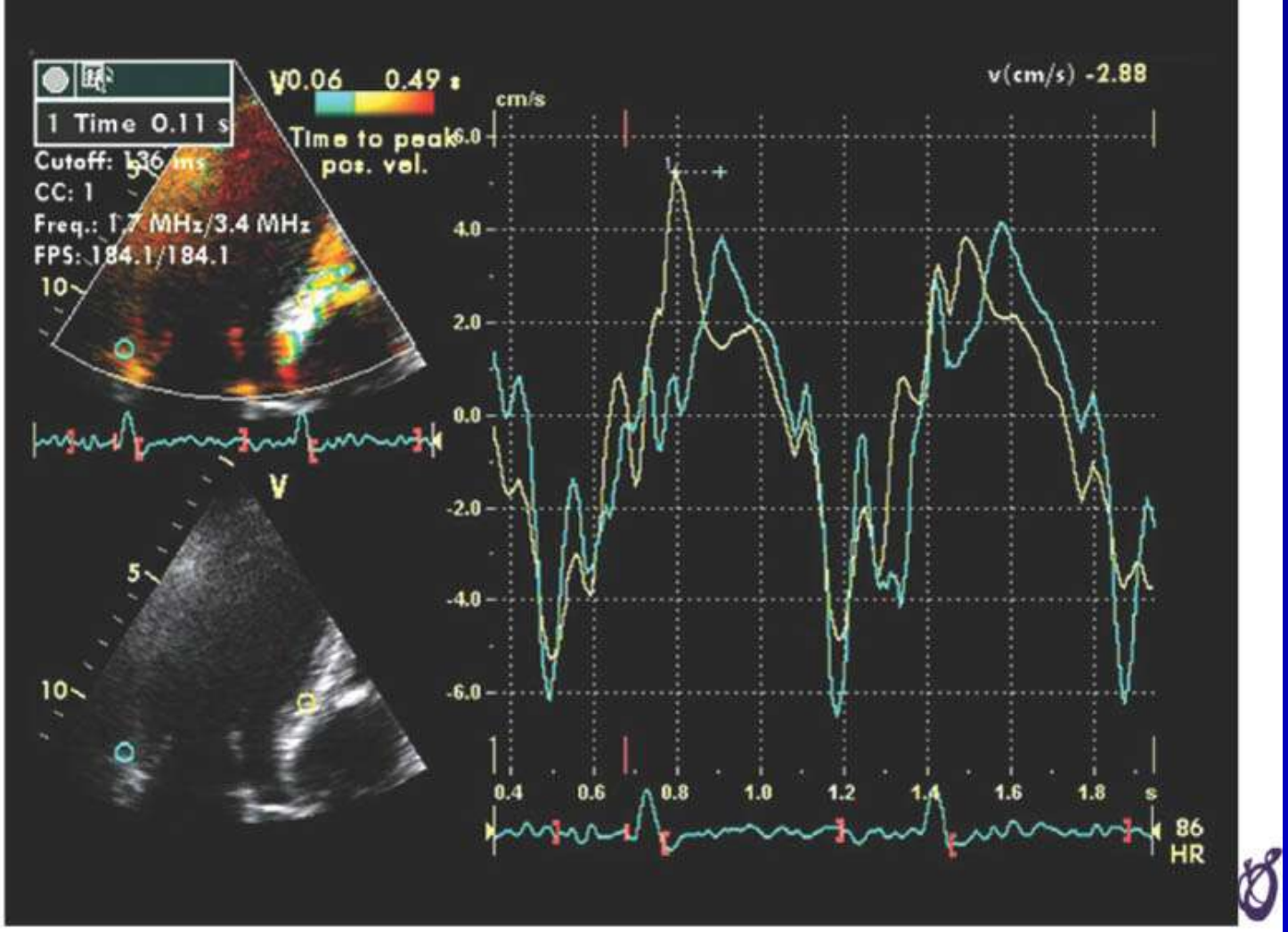
Fig 14-73A, Braunwald's 8th ed.



(From Oh JK, Seward JB, Tajik AJ: The Echo Manual. 3rd ed. Philadelphia, Lippincott Williams & Wilkins, 2006. Used with permission of Mayo Foundation for Medical Education and Research.)

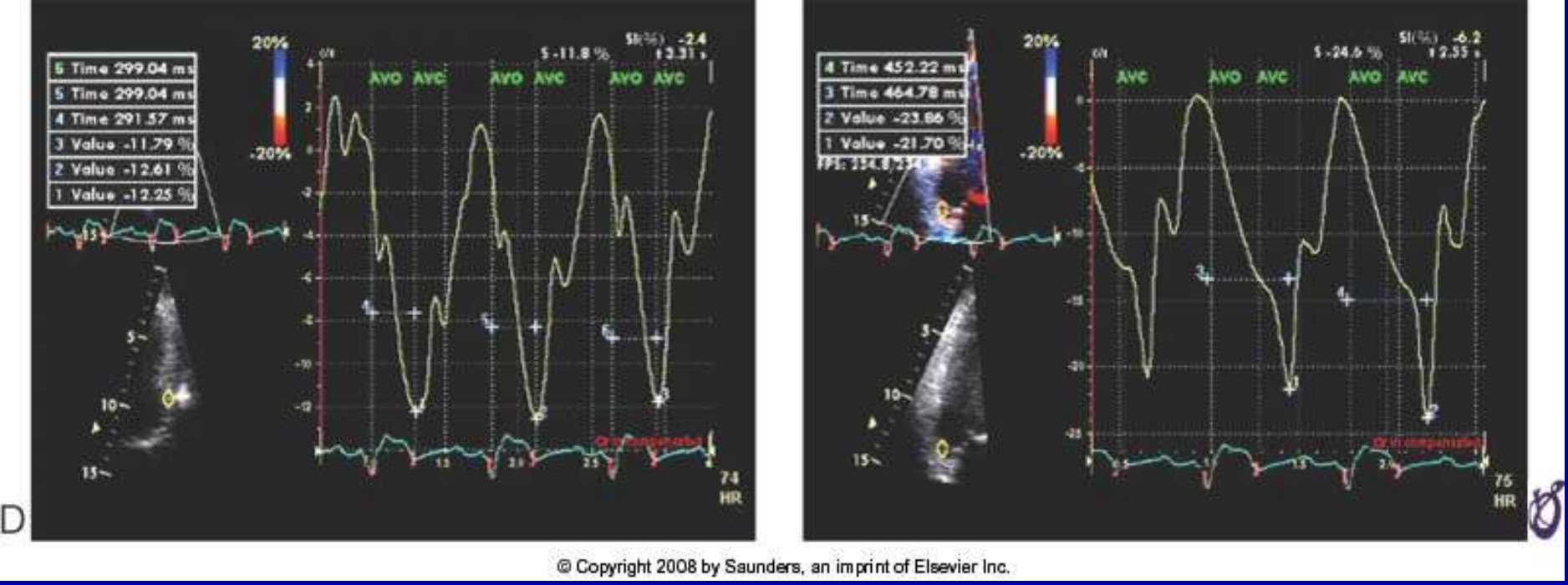
In this patient with dilated cardiomyopathy, the MV inflow velocity pattern shows restrictive physiology, with a markedly decreased A velocity and an increased E/A ratio. Deceleration time (DT) of mitral E velocity is shortened. Patients with this type of diastolic filling have increased filling pressure and symptomatic congestive heart failure.

Fig 14-73B, Braunwald's 8th ed.



Tissue Doppler velocity recordings from the basal septal **(yellow)** and basal lateral **(blue)** walls. Septal peak velocity occurs first, then lateral peak velocity. The timing difference (+----+) is 110 msec, indicating dyssynchronous contraction.

Fig 14-73C, Braunwald's 8th ed.



Intraventricular dyssynchrony can be measured with strain imaging, which we prefer. **Left,** Strain recording from the basal septal wall. The time from the onset of QRS to peak negative strain (when the maximum contraction occurs) is measured from three cardiac cycles. The time intervals measured for the three cycles are 299, 299, and 291 msec. **Right,** Strain recording from the basal lateral wall. The peak strain occurs after aortic valve closure (AVC), which is termed *postsystolic shortening.* The time interval measured from the onset of QRS to peak negative is 452 and 464 msec, much later than that of the septal wall. The timing difference between the septal and lateral segments is more than 150 msec, indicating marked dyssynchrony.

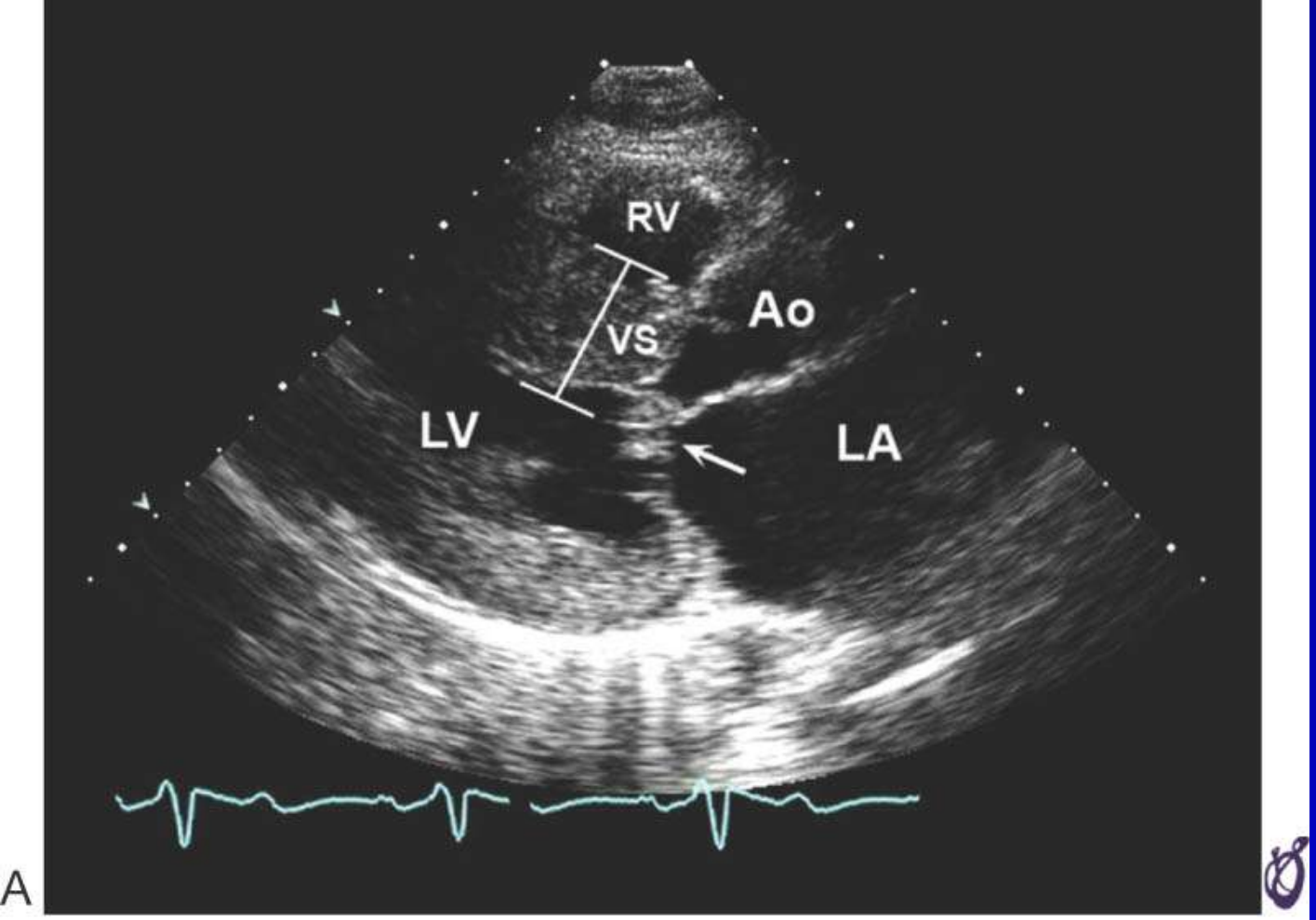
Fig 14-73D, Braunwald's 8th ed.

Echo Findings in Hypertrophic Cardiomyopathy

- Asymmetrical Septal Hypertrophy is most common (diffuse hypertrophy of septum and anterolateral free wall accounts for 70-75% in the West, and Basal septal in 10-15%)
- Also possible:
  - Concentric LVH (5%)
  - Apical HCM (<5%)
  - Lateral wall LVH (1-2%)

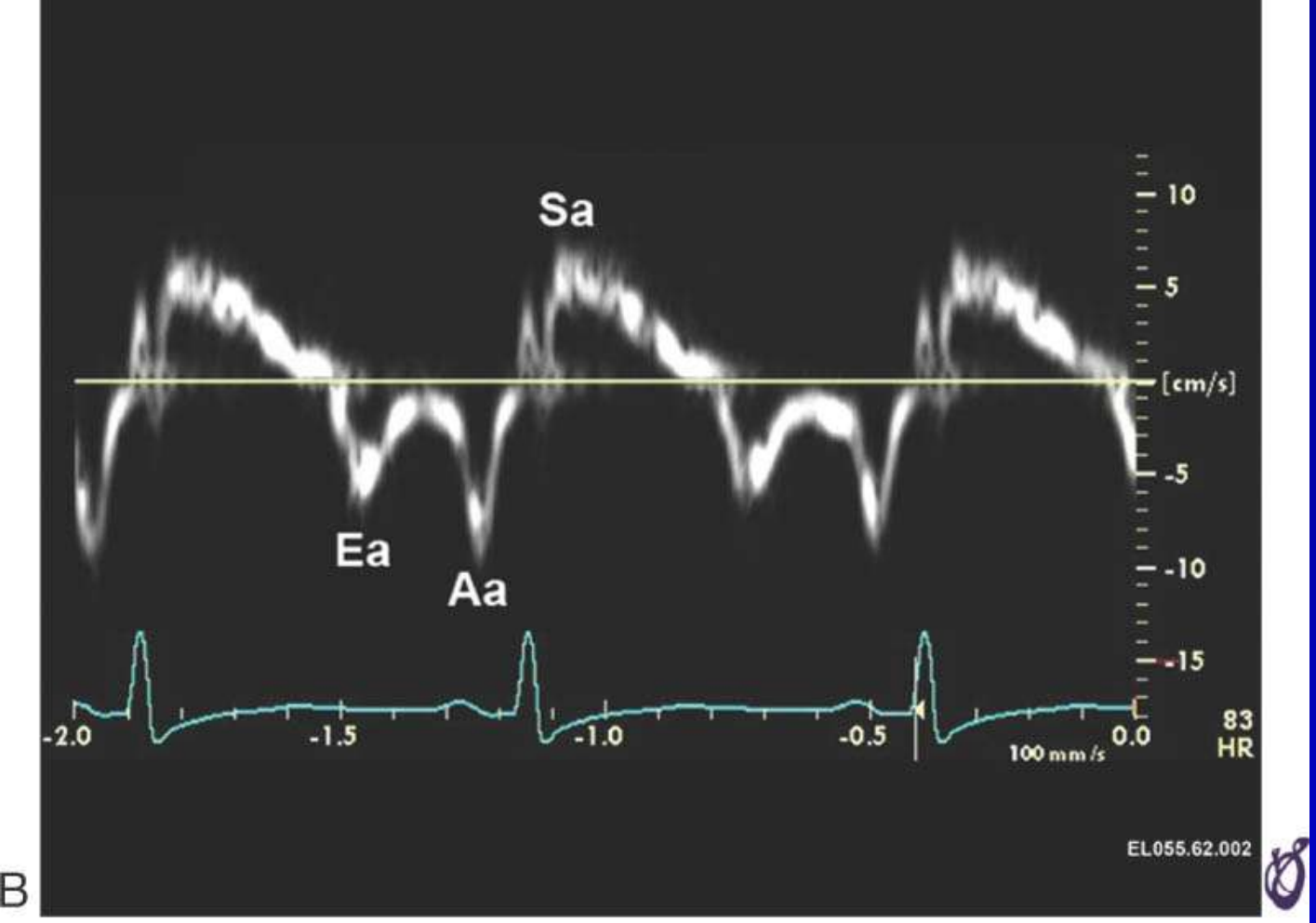
Doppler Findings in Hypertrophic Cardiomyopathy

- Dynamic LVOT obstruction
  - Broad-blade dagger
  - LVOTO is not diagnostic for HCM
    - Elderly hypertensive on vasodilator or diuretic
    - Postop patients on inotropes and volume depleted
    - Postop AS with AVR or MVP with MV repair
    - Membranous subaortic stenosis (but no late peak – looks like regular AS Doppler)
    - Acute anteroapical MI or apical ballooning



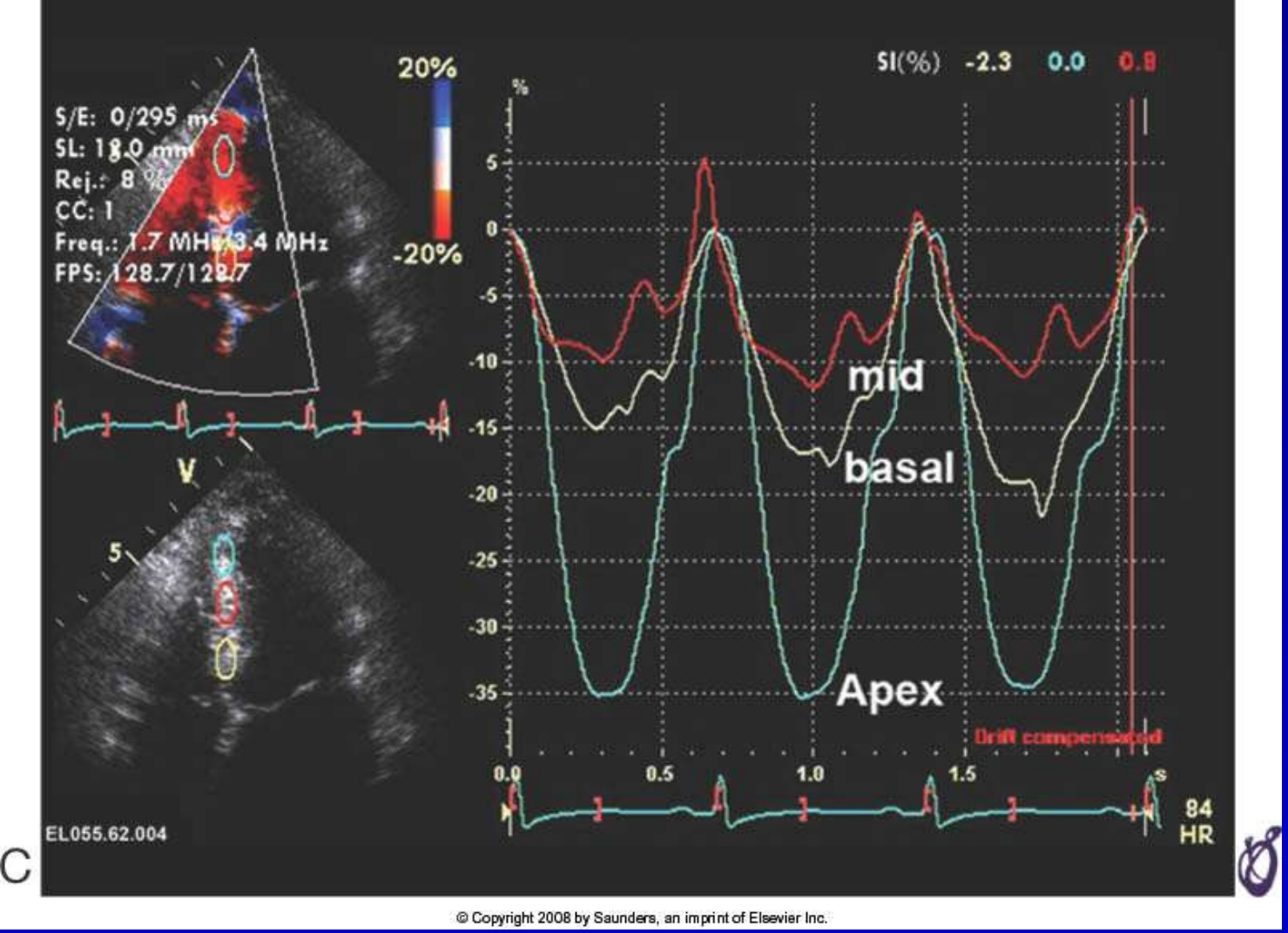
2D echocardiogram of hypertrophic obstructive cardiomyopathy (systolic frame). The ventricular septum (VS) is markedly thickened (25 mm) and has an abnormal myocardial texture. Systolic anterior motion of the anterior mitral leaflet is shown, contributing to the obstruction of the left ventricular outflow tract (arrow).

Fig 14-74A, Braunwald's 8th ed.



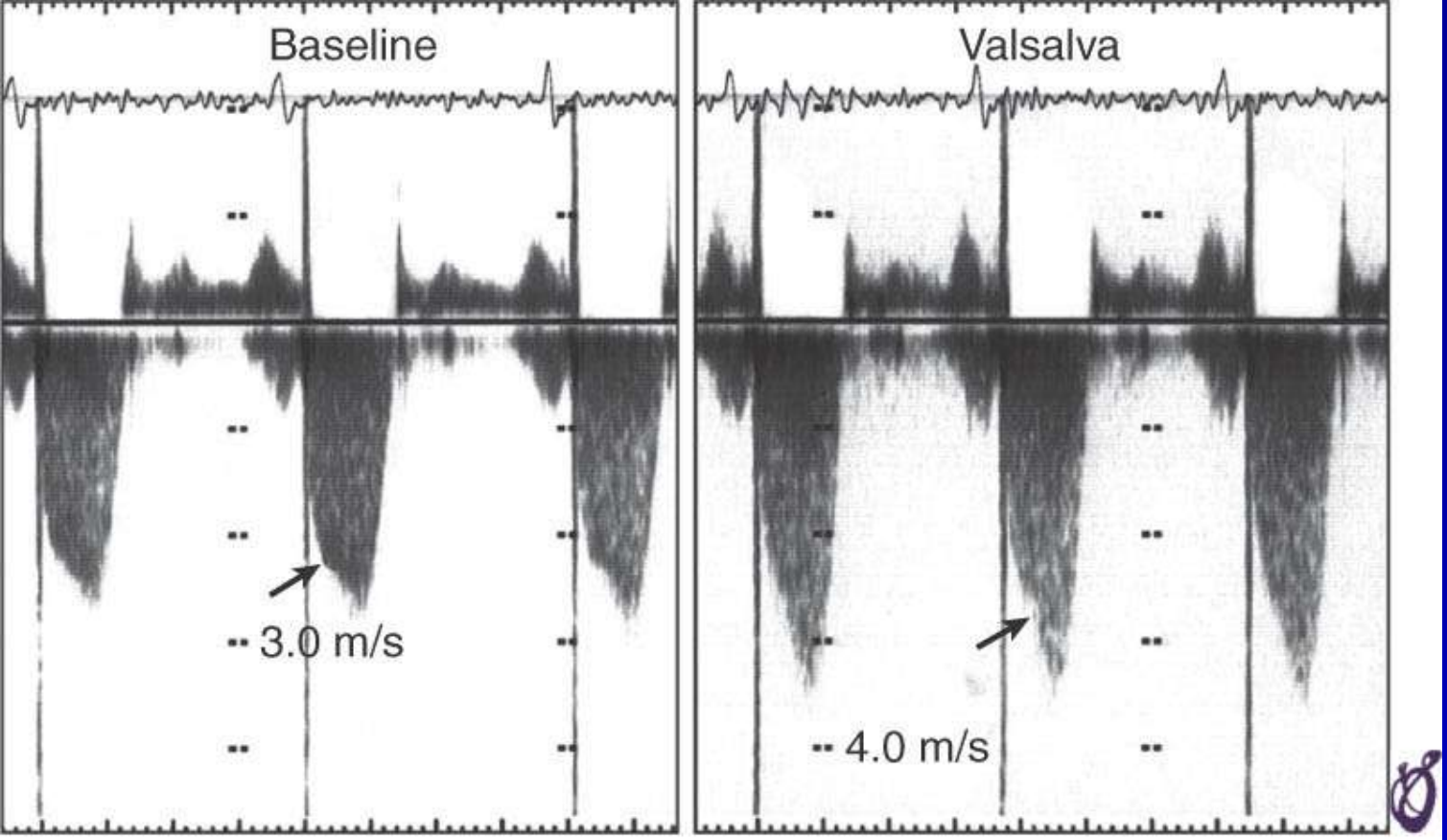
Septal mitral annulus velocity recording by tissue Doppler imaging in a 28-year-old asymptomatic patient with family history of hypertrophic cardiomyopathy. Early diastolic velocity is reduced to 6 cm/sec.

Fig 14-74B, Braunwald's 8th ed.



Strain from mid-septum (red curve with peak negative strain of 10 percent) is markedly reduced and apical strain (blue curve with 35 percent) is increased.

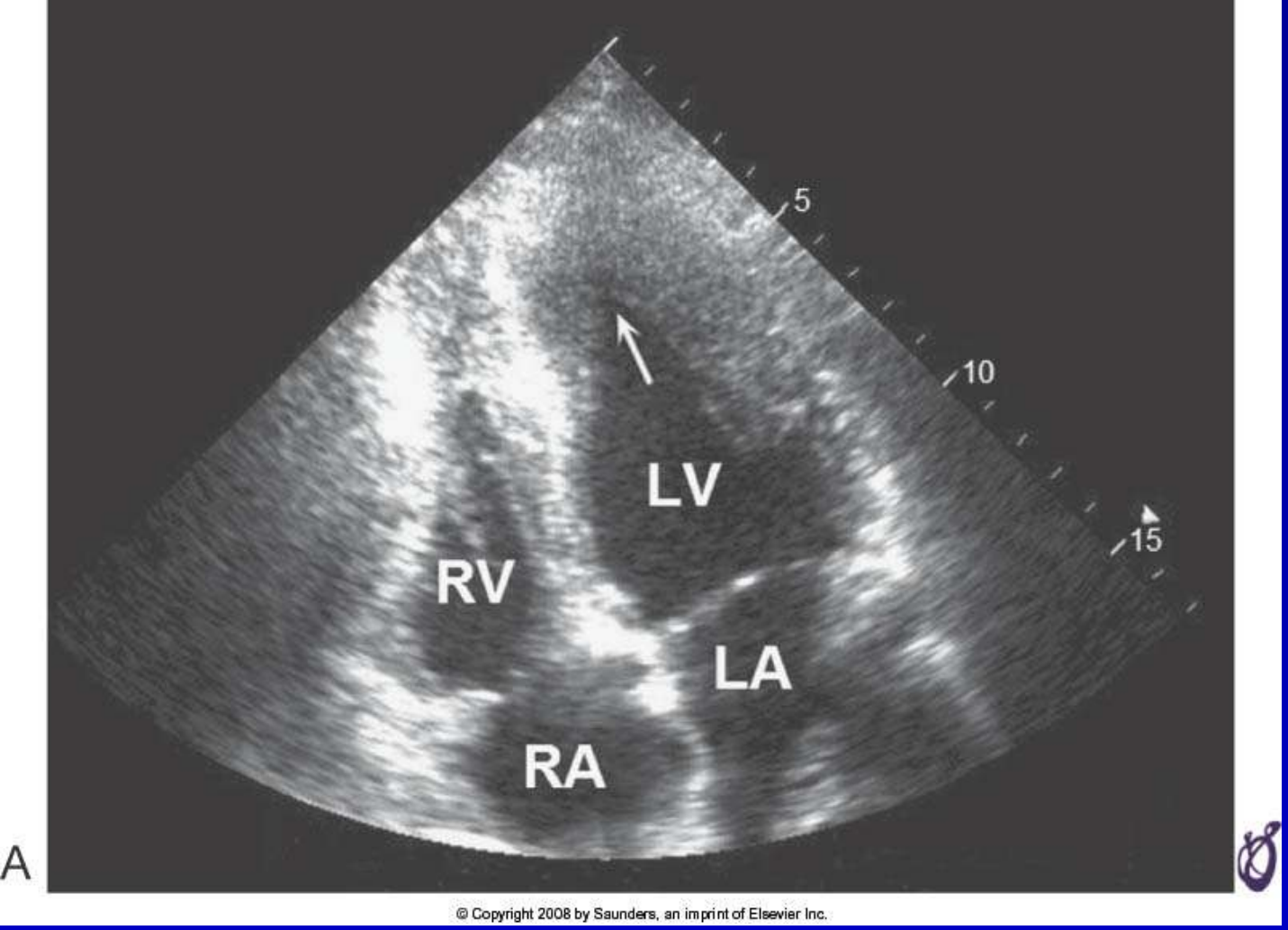
Fig 14-74C, Braunwald's 8th ed.



(From Oh JK, Seward JB, Tajik AJ: The Echo Manual. 3rd ed. Philadelphia, Lippincott Williams & Wilkins, 2006. Used with permission of Mayo Foundation for Medical Education and Research.)

CW Doppler from the apex showing dynamic LVOT obstruction. Note the typical late-peaking configuration resembling a dagger or ski slope (arrow). The baseline **(left)** velocity is 2.8 m/sec, corresponding to the peak left ventricular outflow tract gradient of 31 mm Hg (= 4 × 2.82). With the Valsalva maneuver **(right),** the velocity increased to 3.5 m/sec, corresponding to the gradient of 50 mm Hg

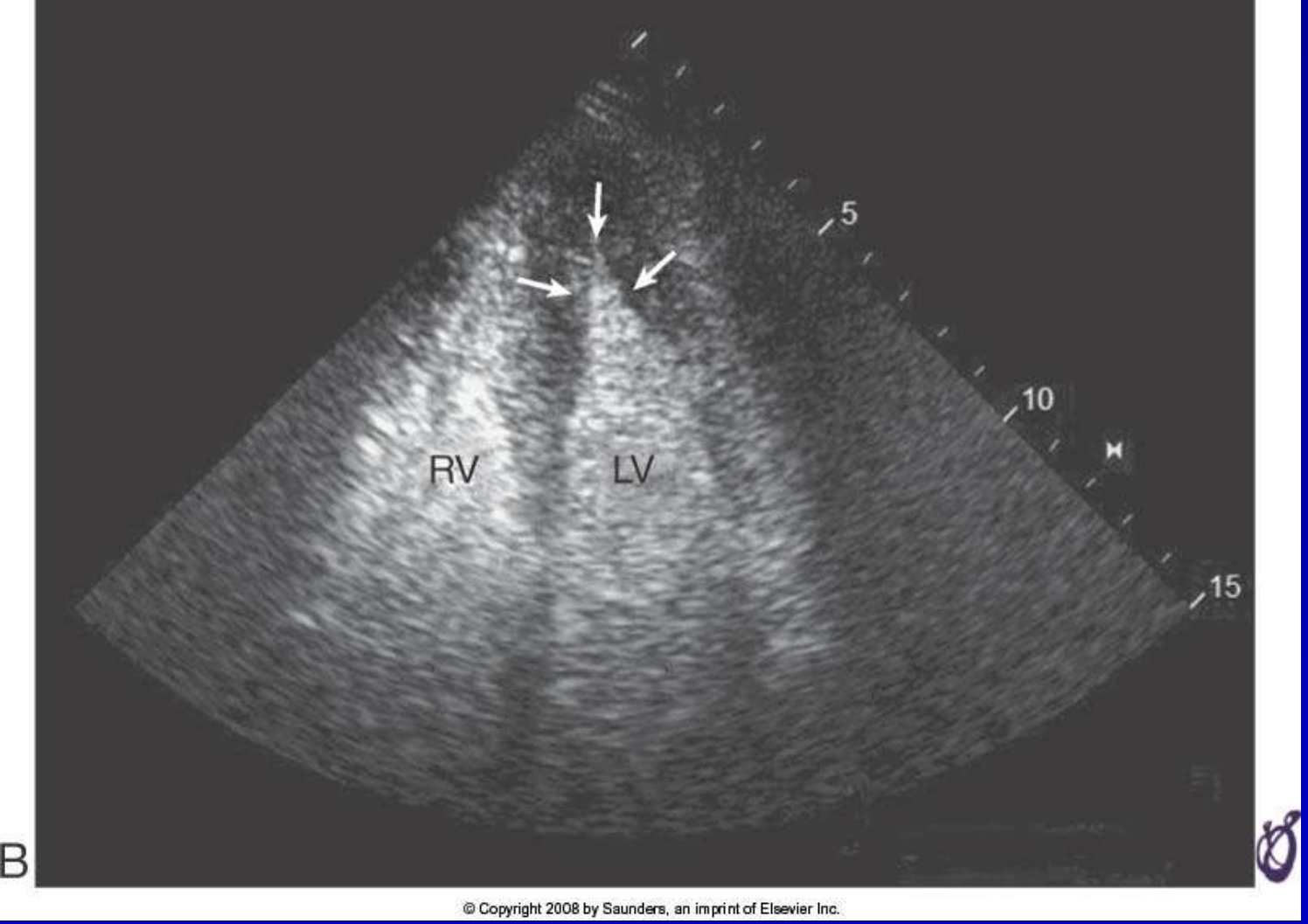
Fig 14-75, Braunwald's 8th ed.



Apical HCM is regularly missed by parasternal echo window; epicardium may seem dyskinetic. DDX: hypereosinophilic syndrome or noncompaction. Association with giant negative precordial T waves.

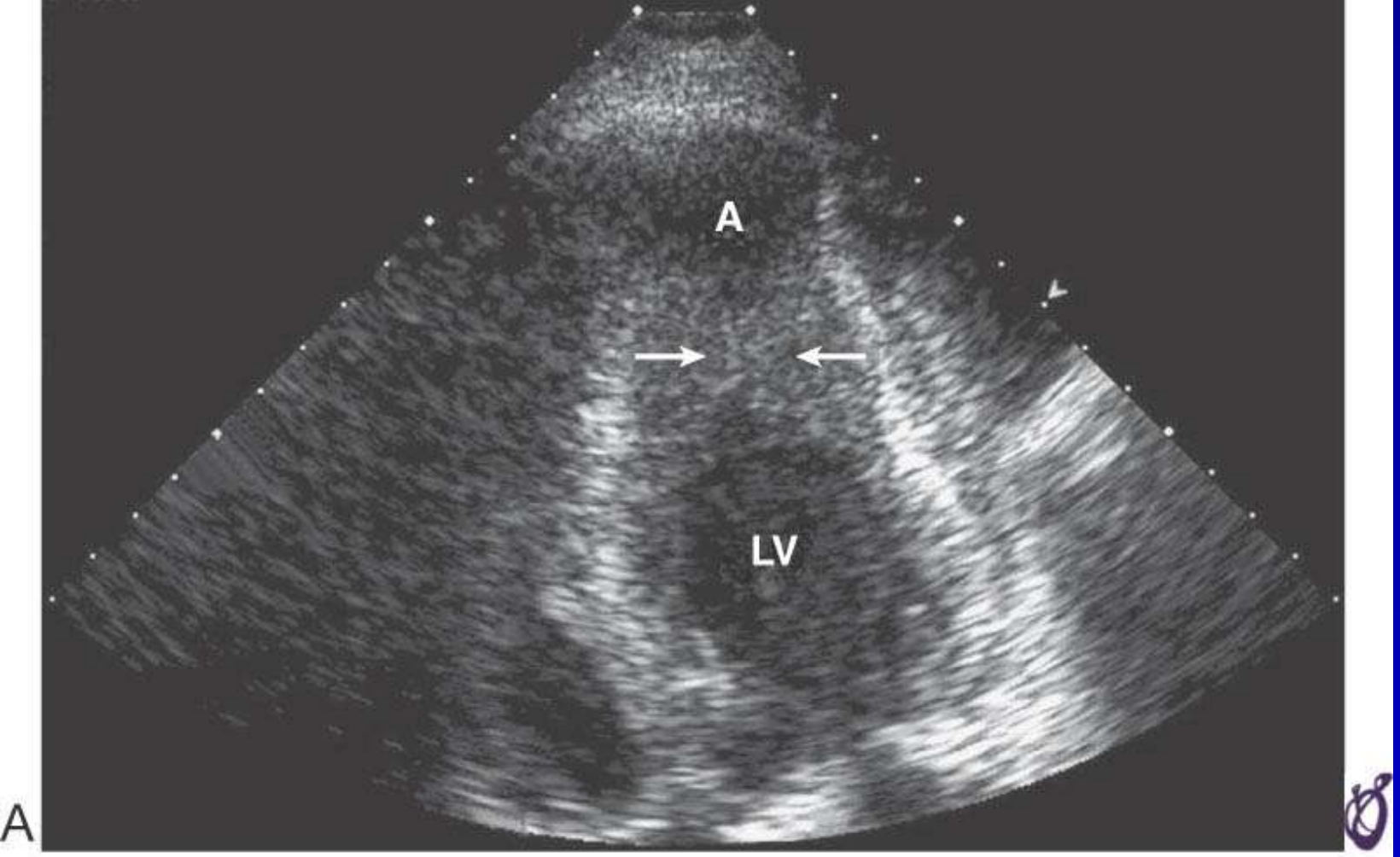
2D apical four-chamber view during diastole in a patient with apical hypertrophic cardiomyopathy. The apical wall thickness during diastole is markedly increased (arrow), and the apical cavity is nearly obliterated except for a small slit during diastole.

Fig 14-76A, Braunwald's 8th ed.



2D apical four-chamber view with contrast shows apical hypertrophy and obliteration of the apical cavity (arrows).

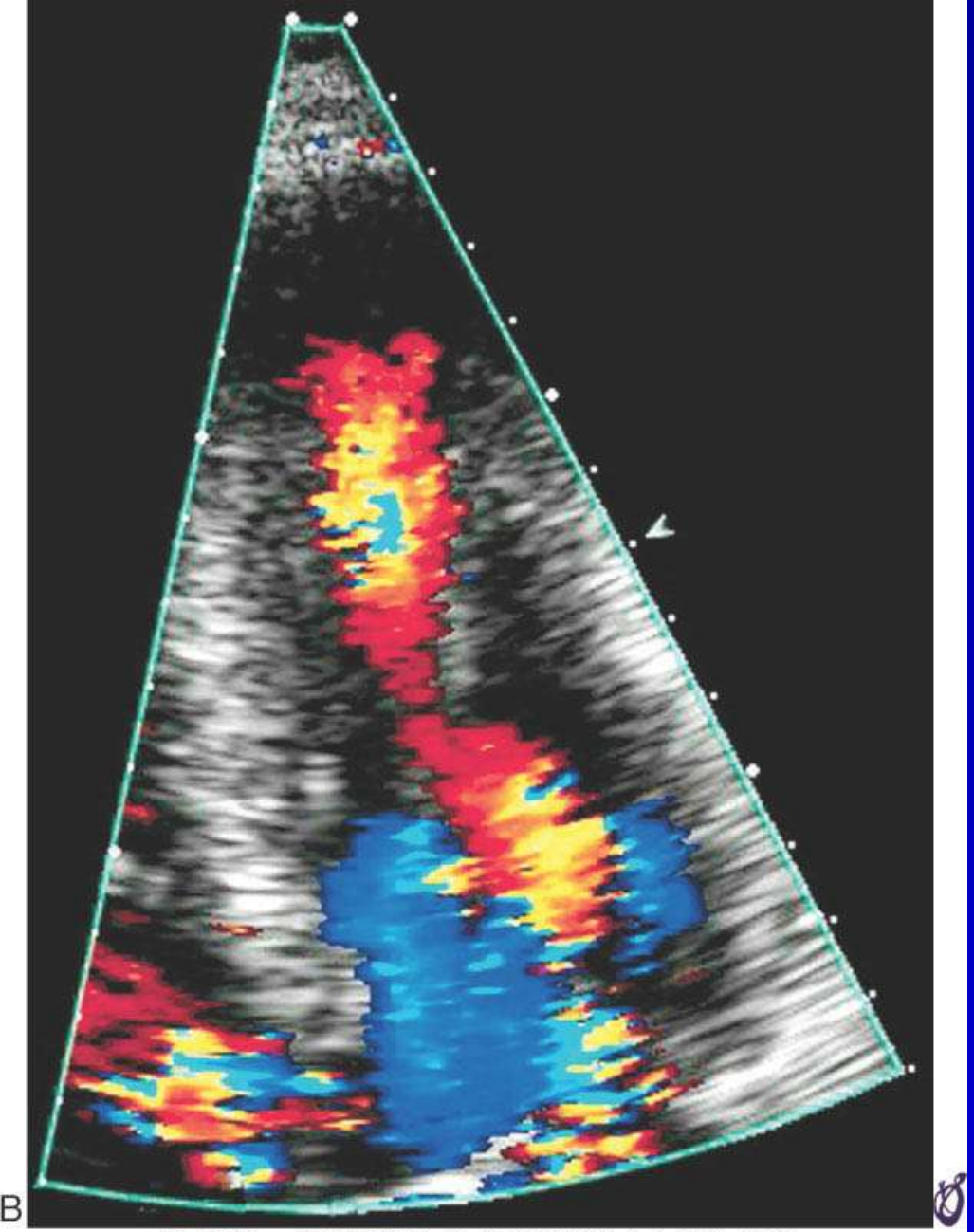
Fig 14-76B, Braunwald's 8th ed.



(From Oh JK, Seward JB, Tajik AJ: The Echo Manual. 3rd ed. Philadelphia, Lippincott Williams & Wilkins, 2006. Used with permission of Mayo Foundation for Medical Education and Research.)

2D echocardiography shows increased left ventricular (LV) wall thickness and systolic obstruction (arrows) at mid ventricle. The apical portion (A) is not well seen.

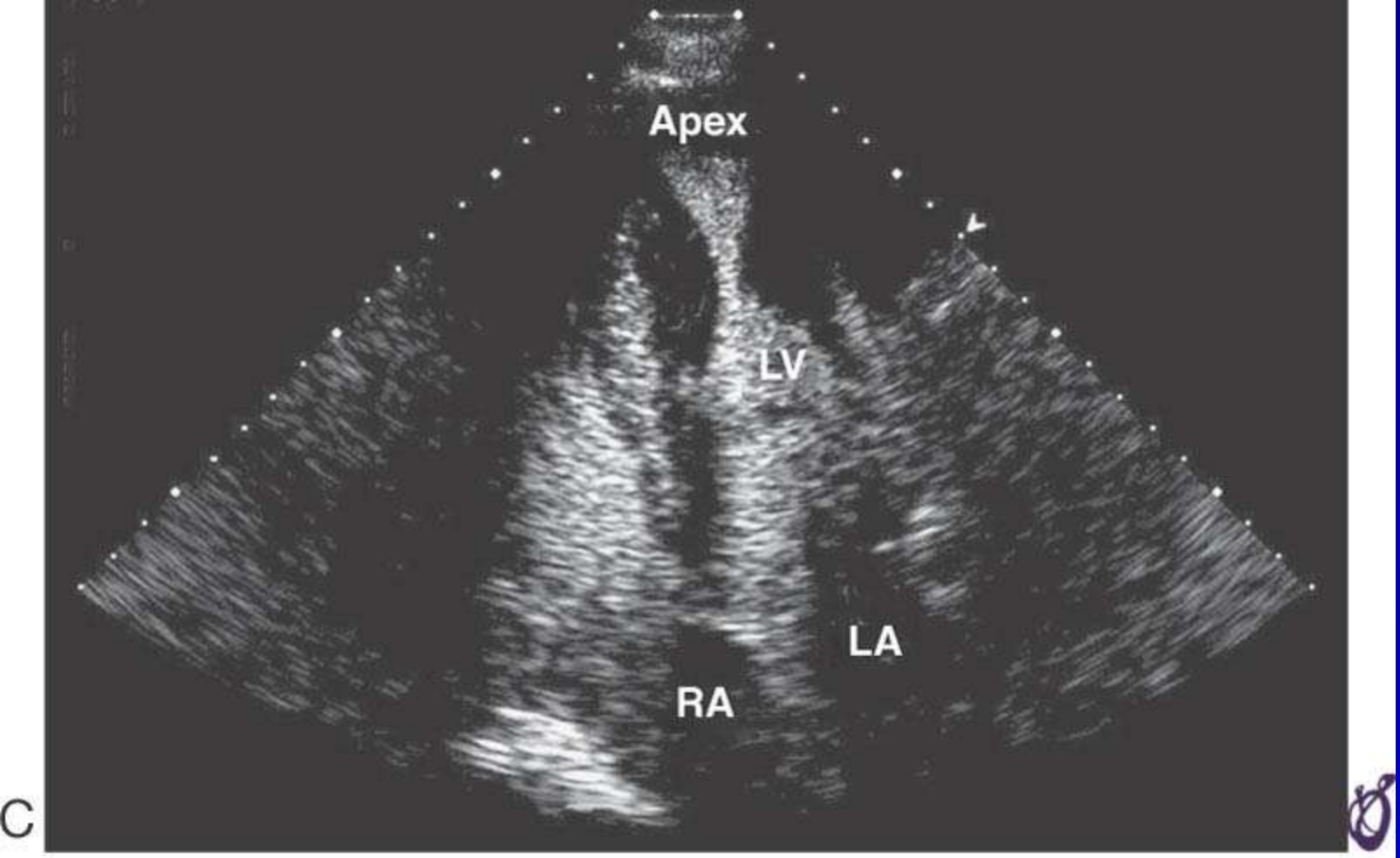
Fig 14-77A, Braunwald's 8th ed.



(From Oh JK, Seward JB, Tajik AJ: The Echo Manual. 3rd ed. Philadelphia, Lippincott Williams & Wilkins, 2006. Used with permission of Mayo Foundation for Medical Education and Research.)

Color flow imaging shows increased flow velocity at the obstructed mid cavity.

Fig 14-77B, Braunwald's 8th ed.



(From Oh JK, Seward JB, Tajik AJ: The Echo Manual. 3rd ed. Philadelphia, Lippincott Williams & Wilkins, 2006. Used with permission of Mayo Foundation for Medical Education and Research.)

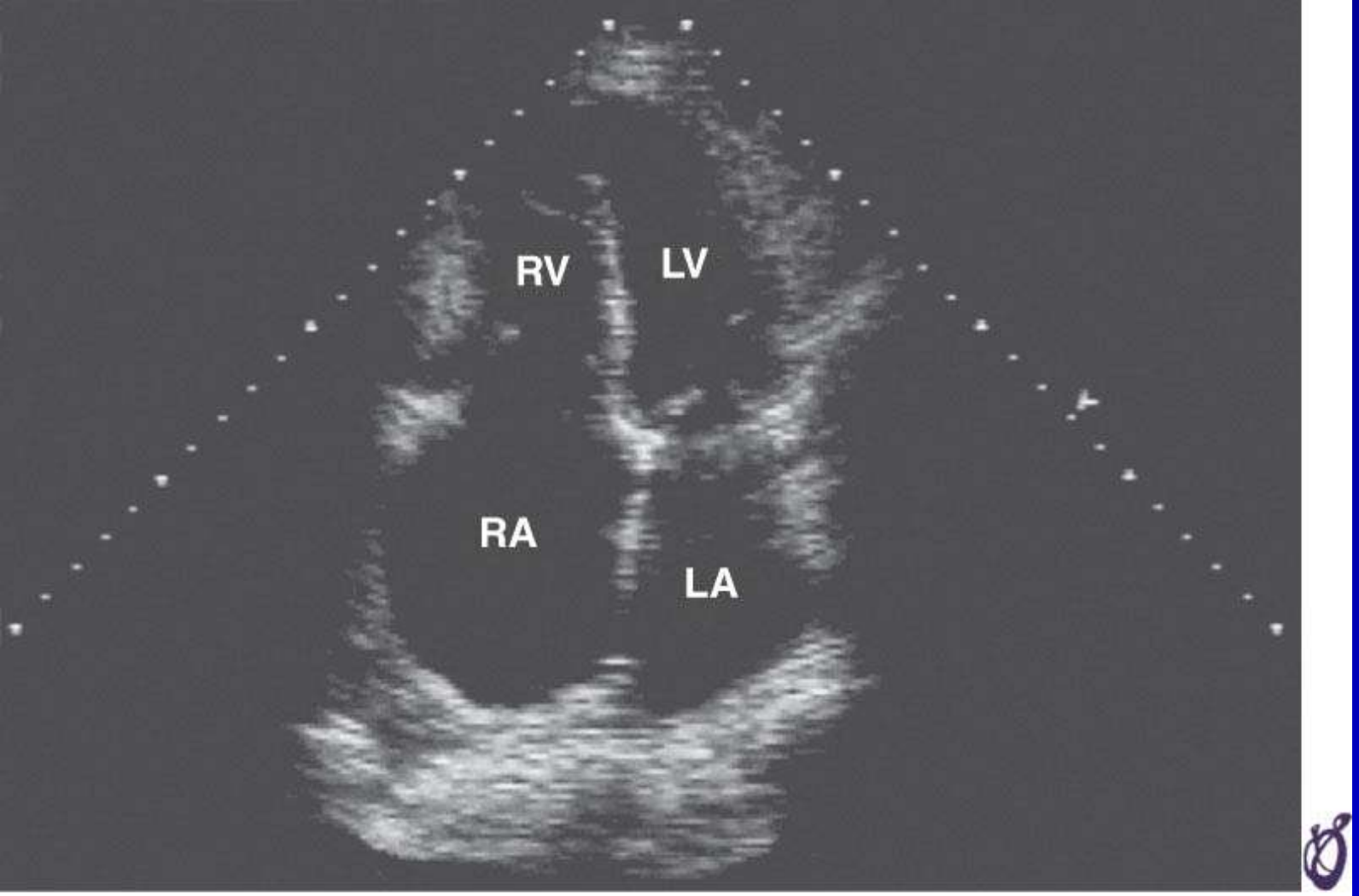
Contrast echocardiography for LV opacification shows the apical cavity best.

Fig 14-77C, Braunwald's 8th ed.

Athlete's Heart is Different from HCM

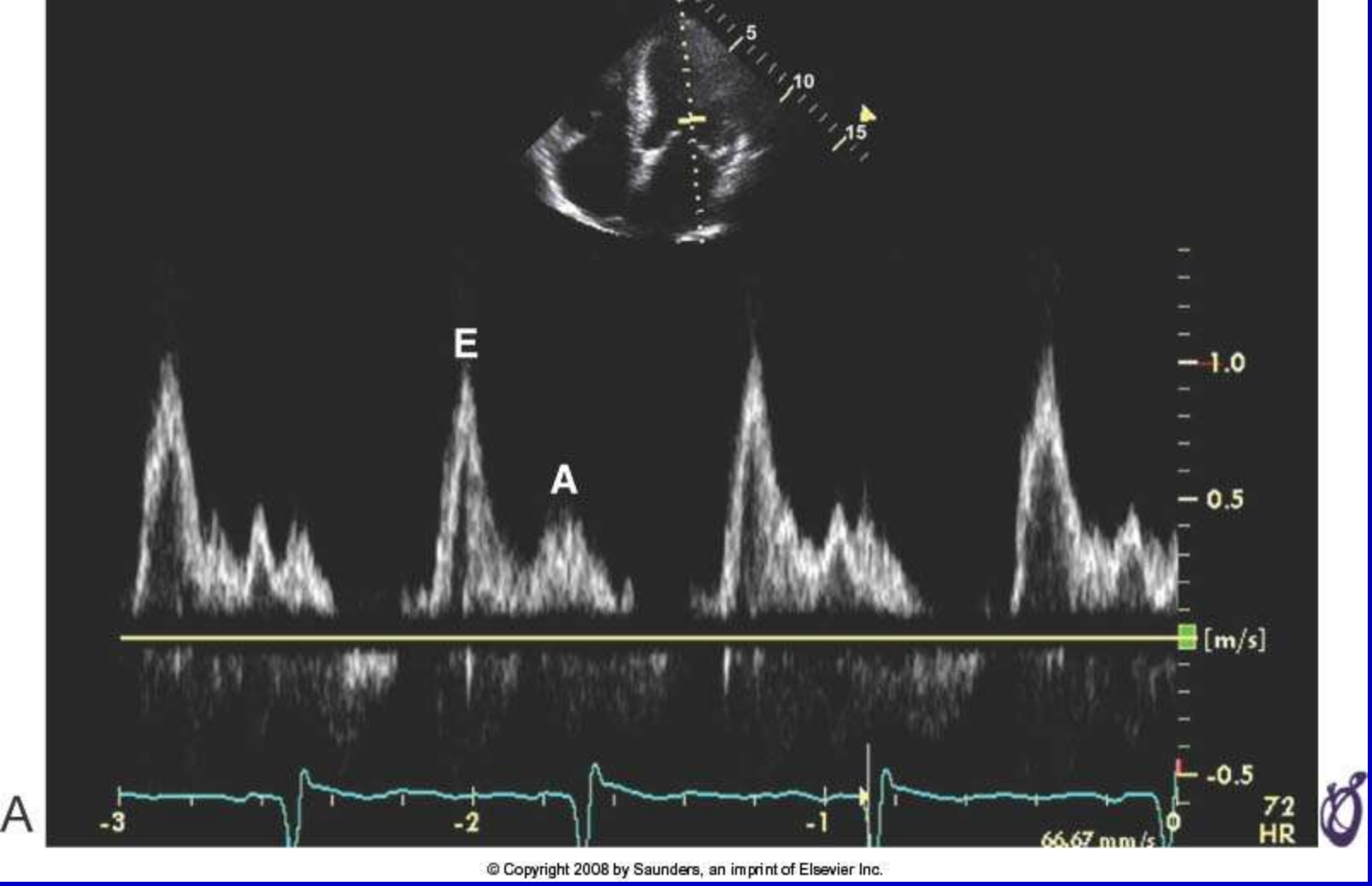
- Athlete's heart is symmetric hypertrophy
- Rarely >17 mm thick
- LV cavity increased
- Diastolic function is normal (Ea > 7 cm/s)
- Tissue Doppler velocities and strain values are normal

Restrictive Cardiomyopathy



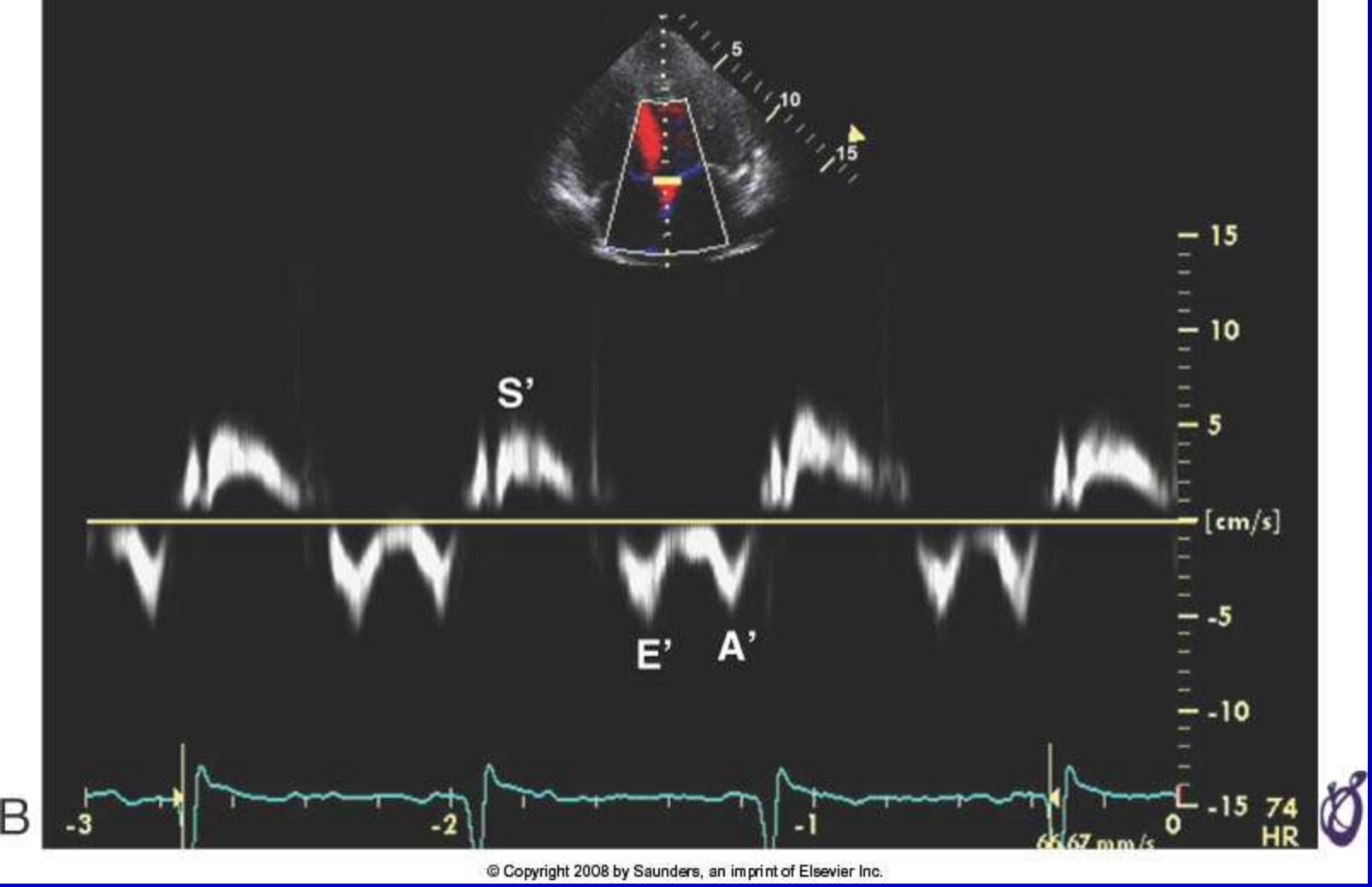
Apical four-chamber view of typical restrictive cardiomyopathy with normal left ventricular (LV) cavity size, normal systolic function, and marked biatrial enlargement.

Fig 14-78, Braunwald's 8th ed.



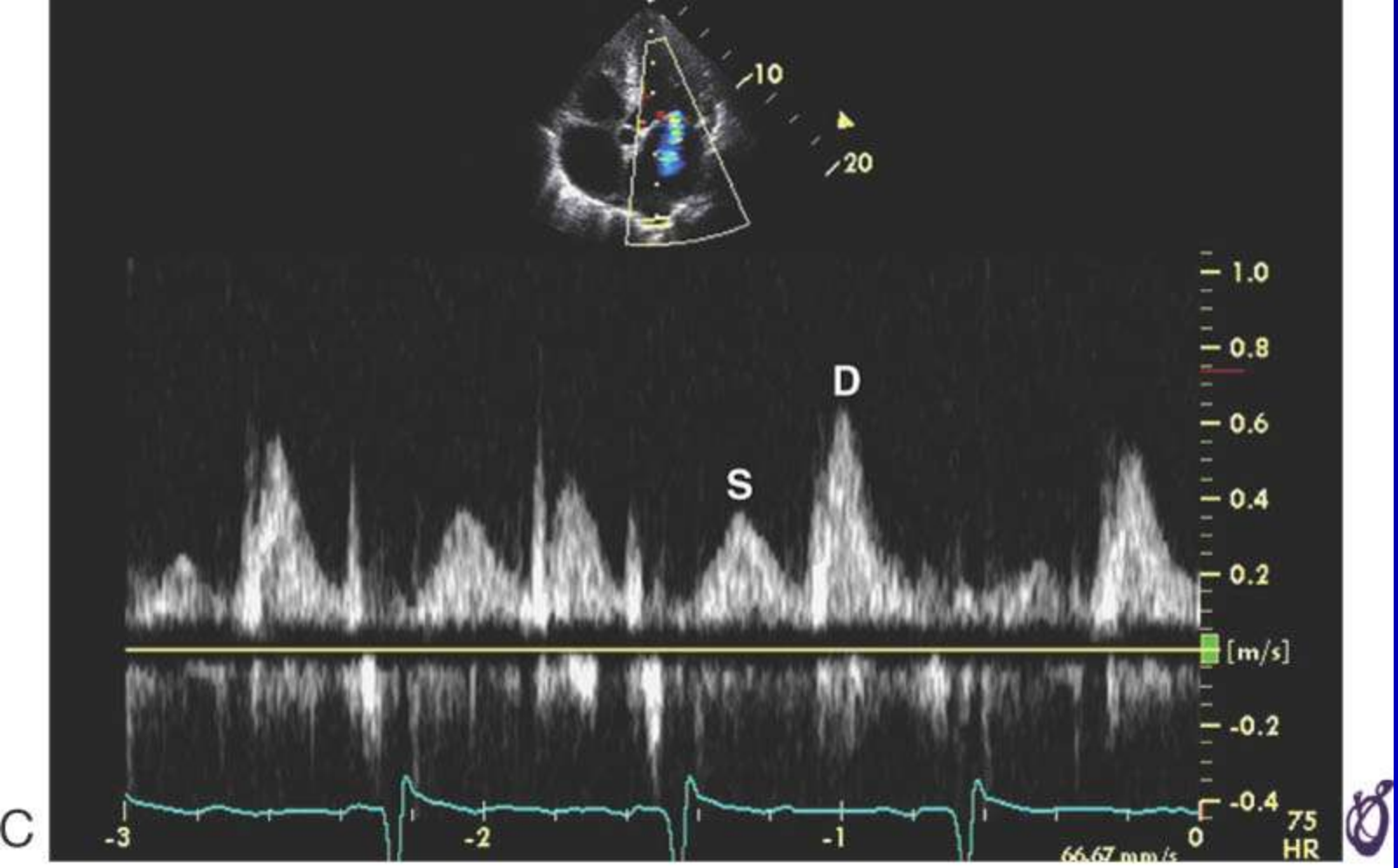
Mitral inflow in a patient with restrictive cardiomyopathy. E/A ratio is greater than 2, and deceleration time is short

Fig 14-79A, Braunwald's 8th ed.



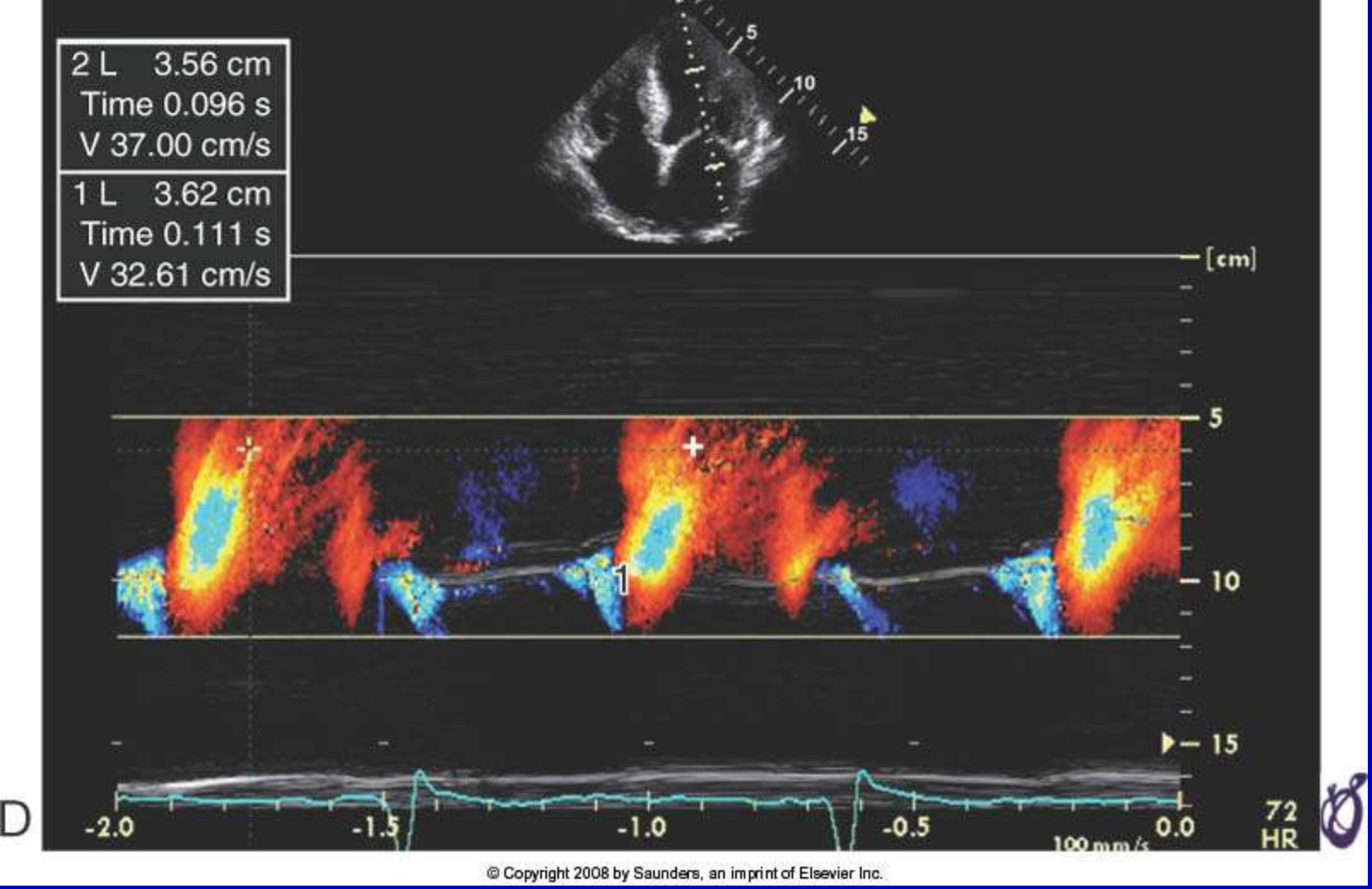
tissue Doppler septal mitral annulus in a patient with restrictive cardiomyopathy. E′ is decreased because of abnormal relaxation; S′ is also decreased

Fig 14-79B, Braunwald's 8th ed.



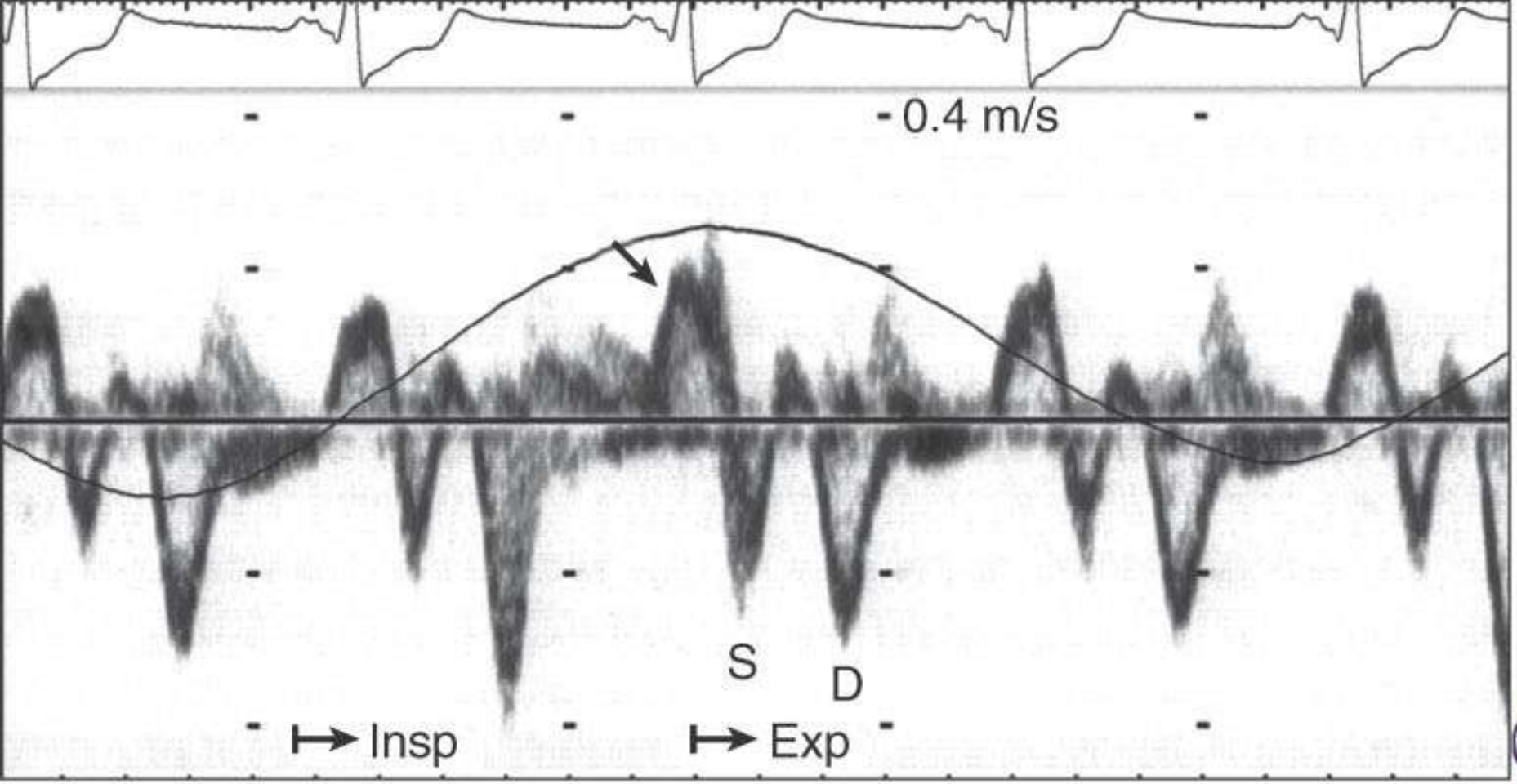
pulmonary vein in a patient with restrictive cardiomyopathy. Pulmonary vein systolic velocity (S) is reduced and diastolic velocity (D) deceleration time is shortened

Fig 14-79C, Braunwald's 8th ed.



color M-mode of mitral inflow velocity in a patient with restrictive cardiomyopathy. Mitral inflow color propagation velocity (V) is reduced to 32-37 cm/sec.

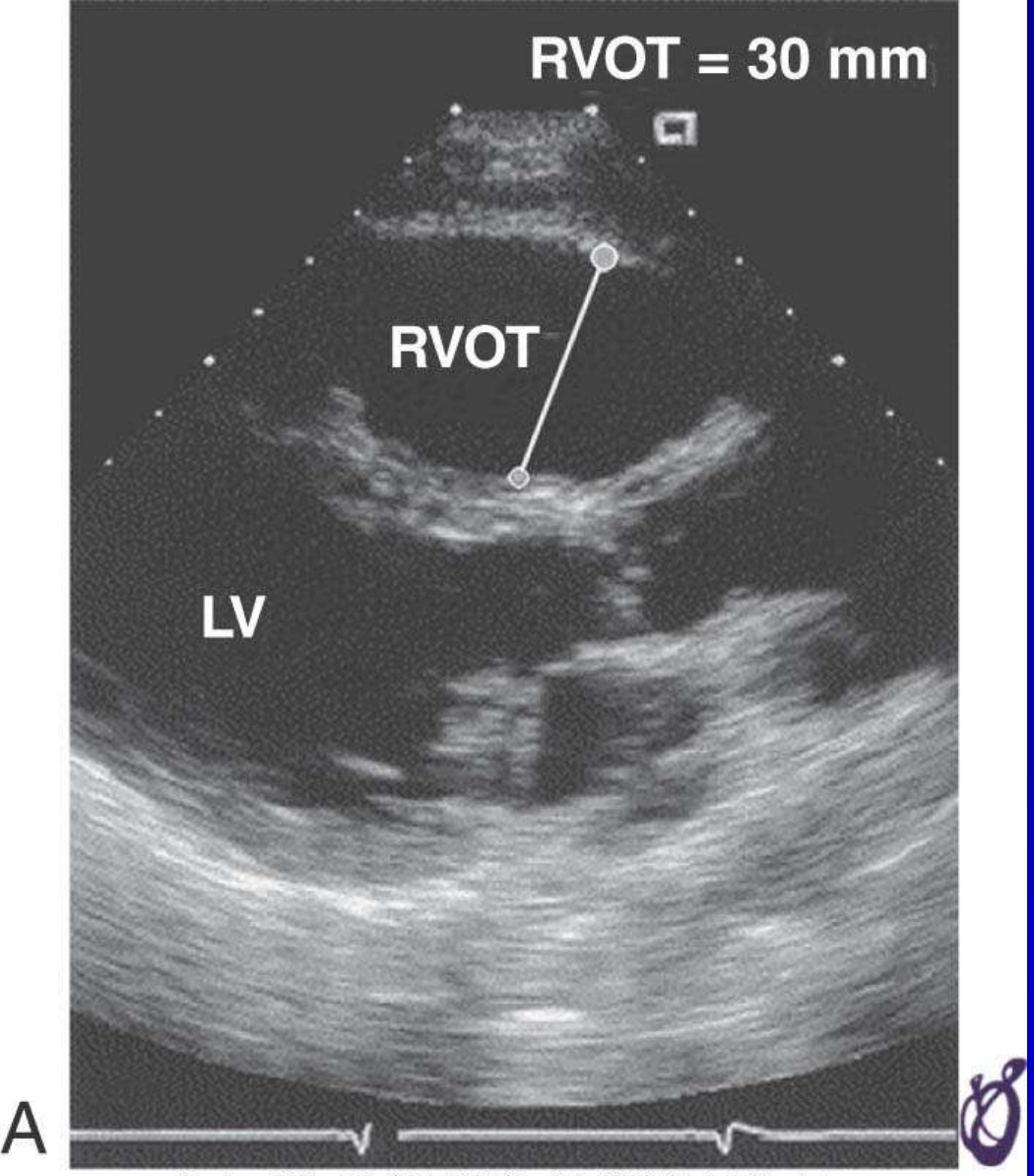
Fig 14-79D, Braunwald's 8th ed.



Hepatic vein pulsed wave Doppler recording together with respirometer recording from a patient with restrictive physiology. Note the higher diastolic velocity (D) than systolic velocity (S) and greater reversal of diastolic flow during inspiration (Insp) (arrow).

Fig 14-80, Braunwald's 8th ed.

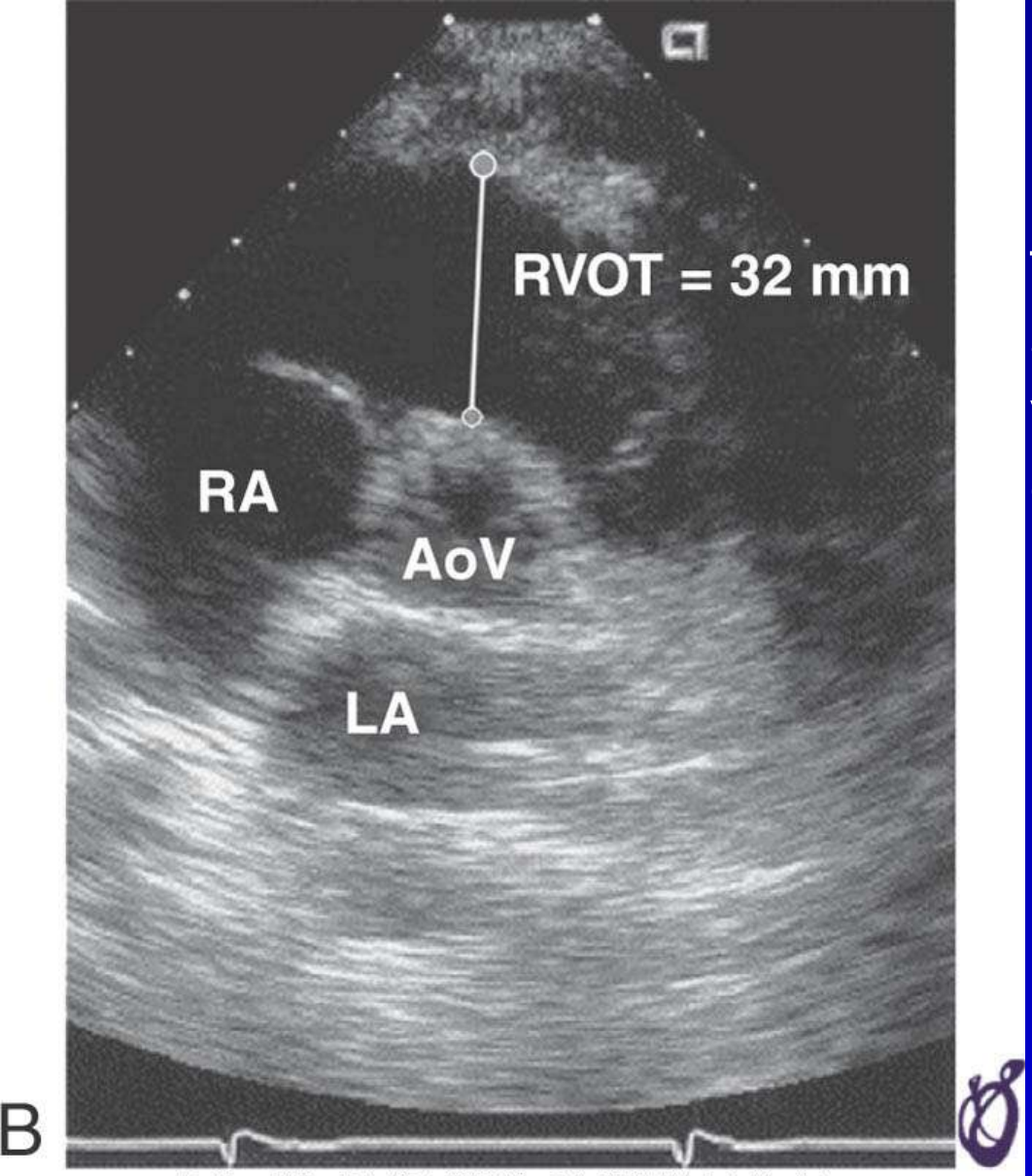
Arrhythmogenic RV Cardiomyopathy



(From Yoerger DM, Marcus F, Sherrill D, et al, Multidisciplinary Study of Right Ventricular Dysplasia Investigators: Echocardiographic findings in patients meeting task force criteria for arrhythmogenic right ventricular dysplasia: New insights from the multidisciplinary study of right ventricular dysplasia. J Am Coll Cardiol 45:860, 2005. Used with permission.)

Echocardiographic views from a patient with arrhythmogenic right ventricular dysplasia. Right ventricular outflow tract (RVOT) enlargement from the parasternal long-axis view

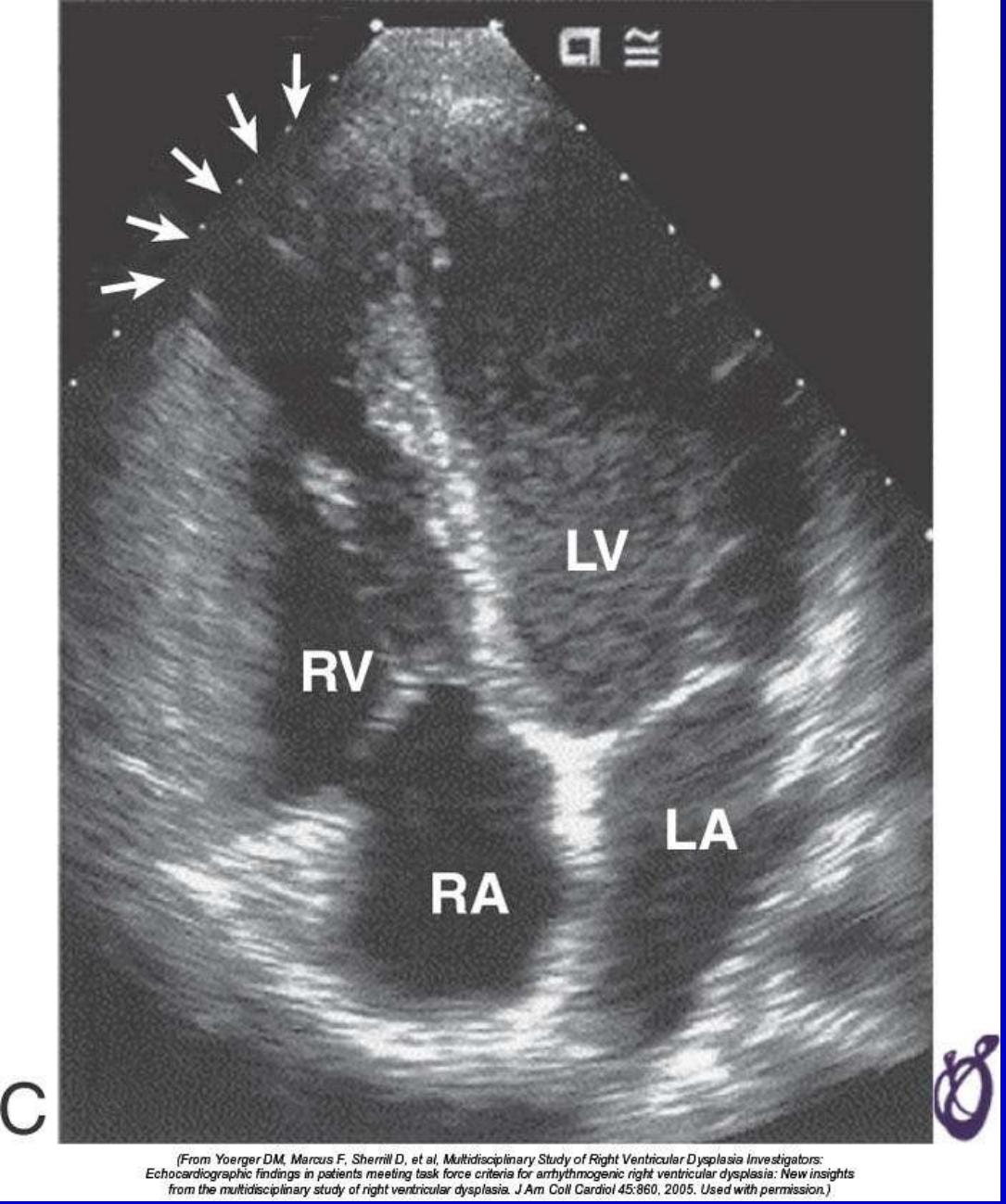
Fig 14-81A, Braunwald's 8th ed.



(From Yoerger DM, Marcus F, Sherrill D, et al, Multidisciplinary Study of Right Ventricular Dysplasia Investigators: Echocardiographic findings in patients meeting task force criteria for arrhythmogenic right ventricular dysplasia: New insights from the multidisciplinary study of right ventricular dysplasia. J Am Coll Cardiol 45:860, 2005. Used with permission.)

Echocardiographic views from a patient with arrhythmogenic right ventricular dysplasia. RVOT enlargement from the parasternal short-axis view.

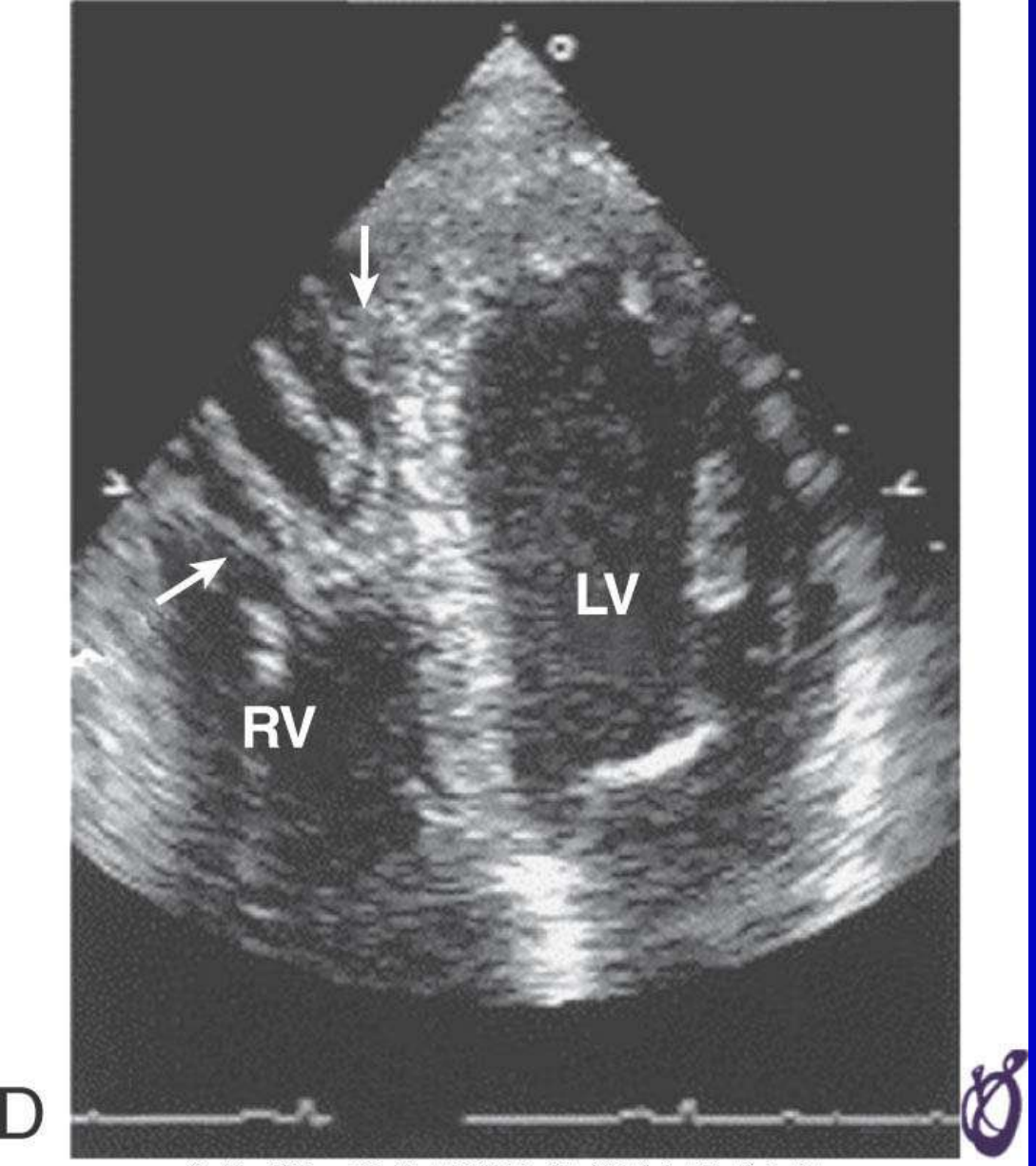
Fig 14-81B, Braunwald's 8th ed.



(From Yoerger DM, Marcus F, Sherrill D, et al, Multidisciplinary Study of Right Ventricular Dysplasia Investigators: Echocardiographic findings in patients meeting task force criteria for arrhythmogenic right ventricular dysplasia: New insights from the multidisciplinary study of right ventricular dysplasia. J Am Coll Cardiol 45:860, 2005. Used with permission.)

Echocardiographic views from a patient with arrhythmogenic right ventricular dysplasia. Note the focal right ventricular (RV) apical aneurysm (arrows).

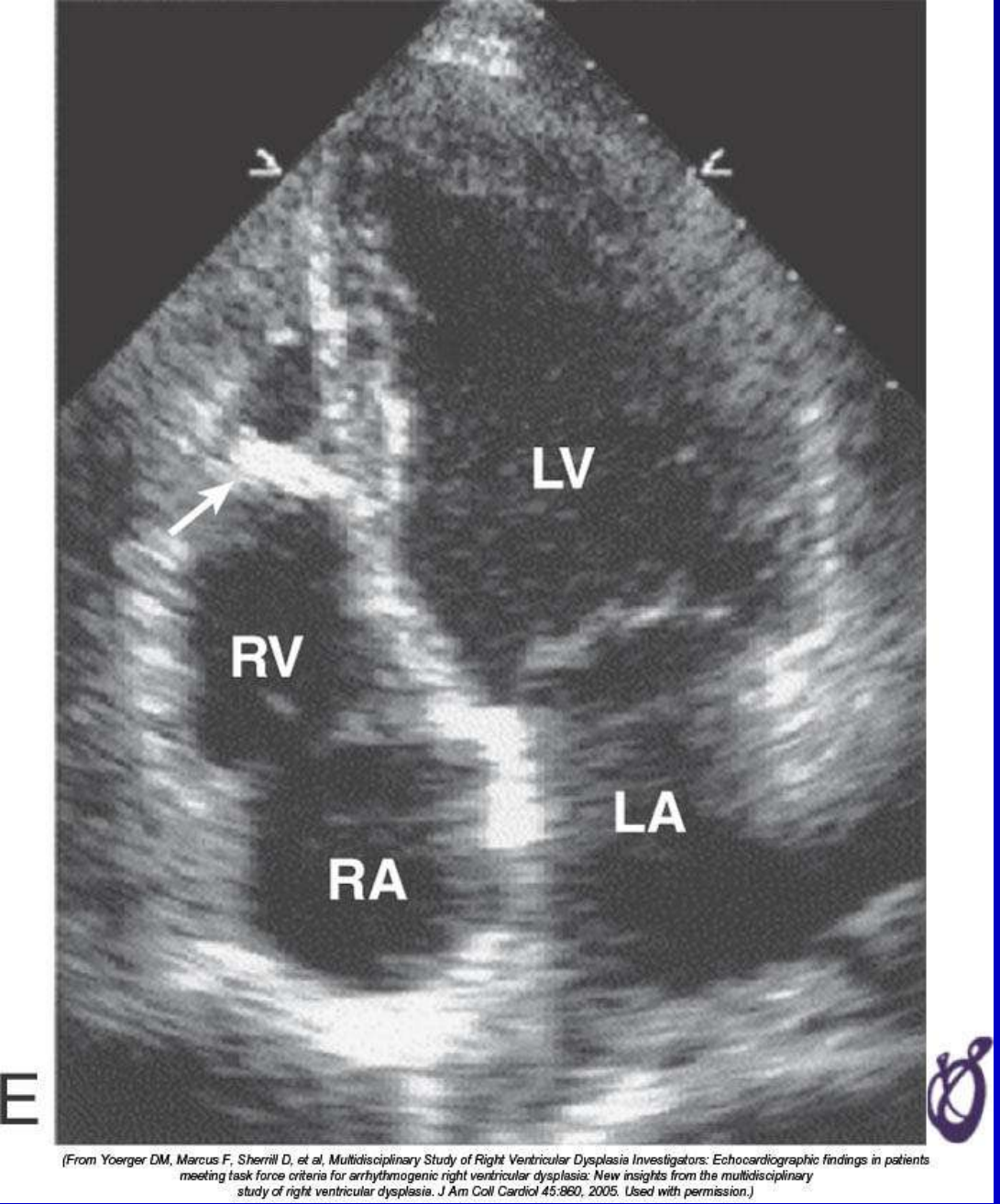
Fig 14-81C, Braunwald's 8th ed.



(From Yoerger DM, Marcus F, Sherrill D, et al, Multidisciplinary Study of Right Ventricular Dysplasia Investigators: Echocardiographic findings in patients meeting task force criteria for arrhythmogenic right ventricular dysplasia: New insights from the multidisciplinary study of right ventricular dysplasia. J Am Coll Cardiol 45:860, 2005. Used with permission.)

Echocardiographic views from a patient with arrhythmogenic right ventricular dysplasia. Note excessive trabeculations (arrows).

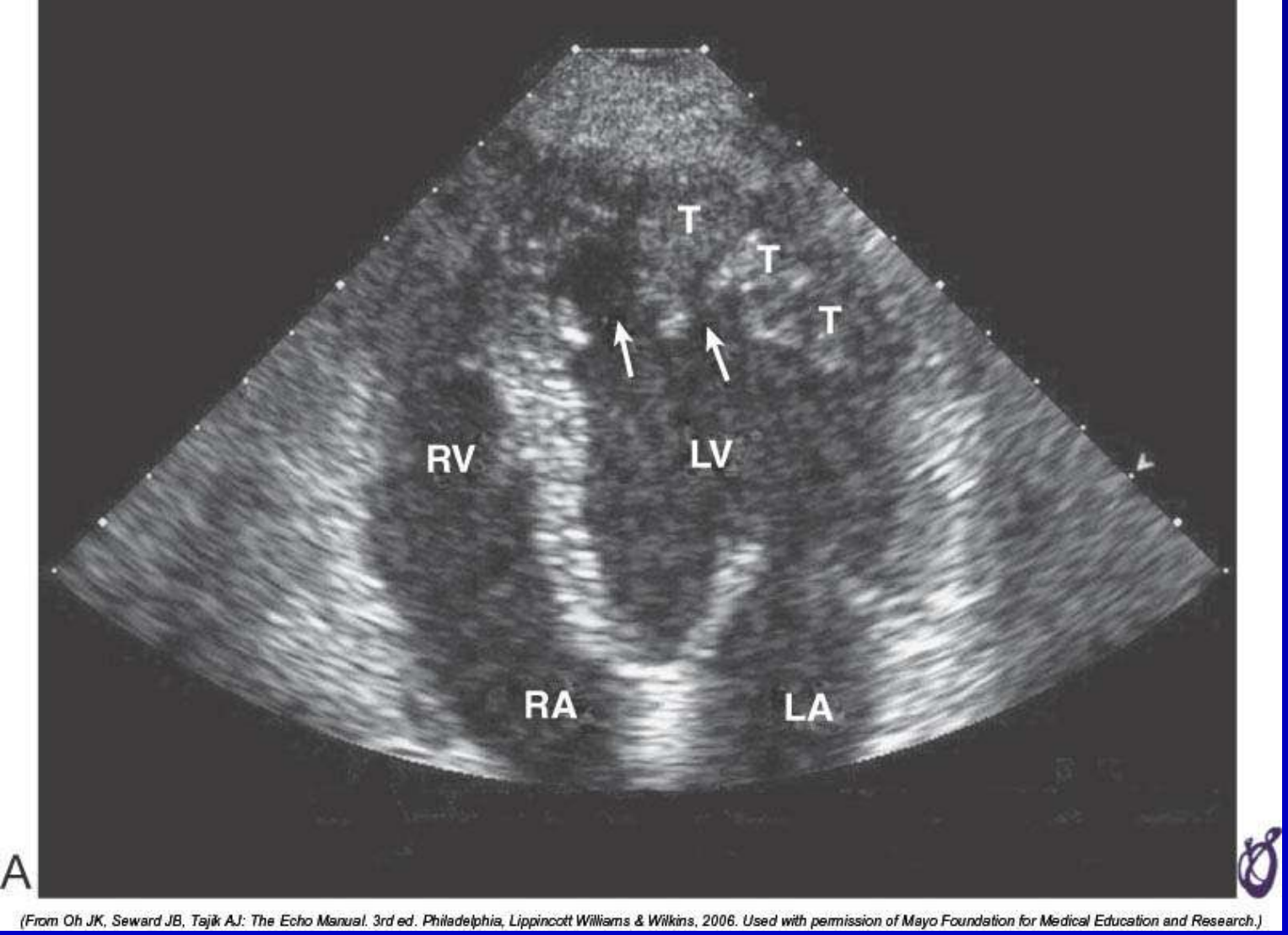
Fig 14-81D, Braunwald's 8th ed.



(From Yoerger DM, Marcus F, Sherrill D, et al, Multidisciplinary Study of Right Ventricular Dysplasia Investigators: Echocardiographic findings in patients meeting task force criteria for arrhythmogenic right ventricular dysplasia: New insights from the multidisciplinary study of right ventricular dysplasia. J Am Coll Cardiol 45:860, 2005. Used with permission.)

Echocardiographic views from a patient with arrhythmogenic right ventricular dysplasia. Note hyperreflective moderator band (arrow).

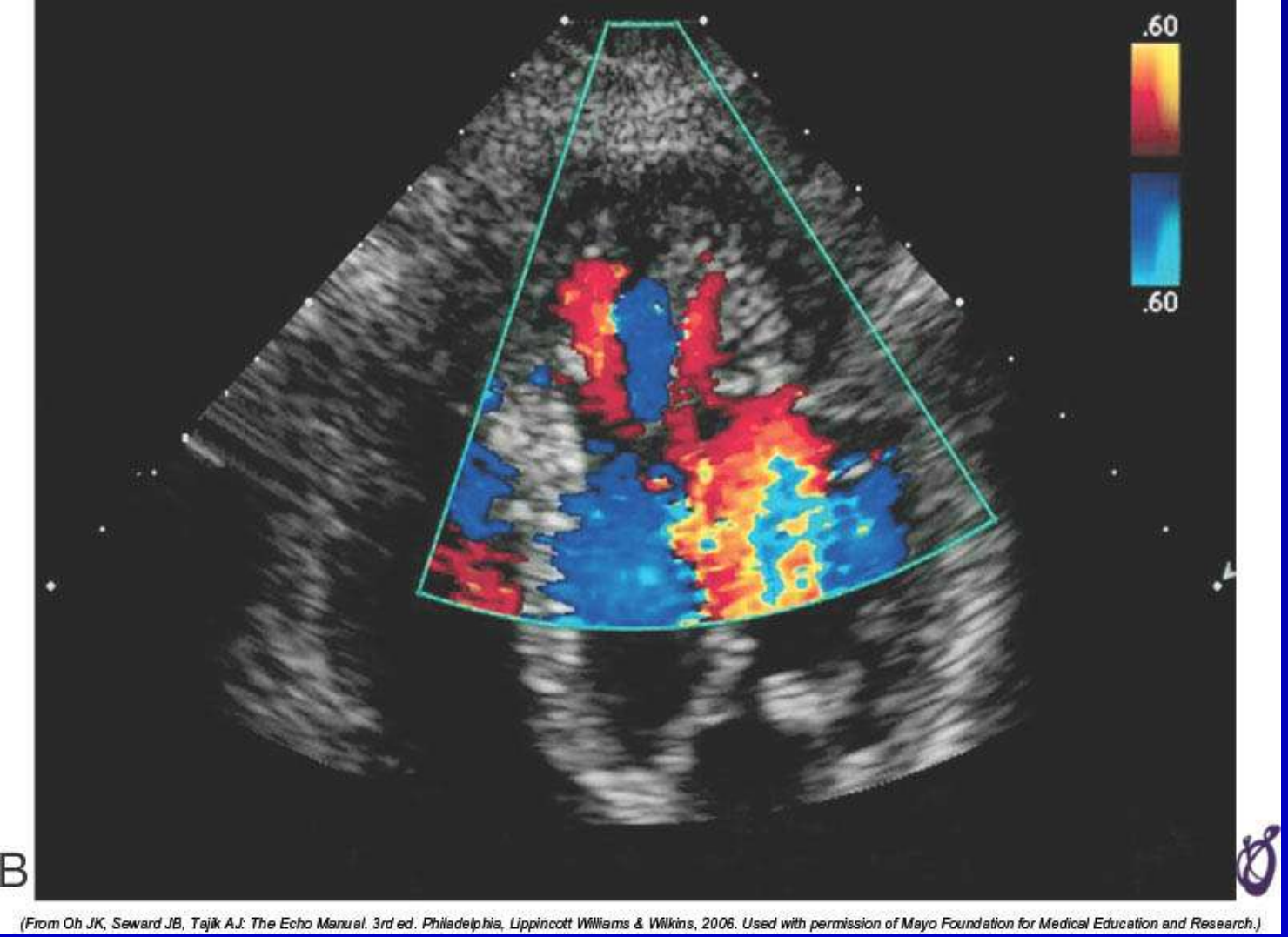
Fig 14-81E, Braunwald's 8th ed.



(From Oh JK, Seward JB, Tajik AJ: The Echo Manual. 3rd ed. Philadelphia, Lippincott Williams & Wilkins, 2006. Used with permission of Mayo Foundation for Medical Education and Research.)

Apical four-chamber 2D echocardiographic view showing characteristic increase in trabeculations (T) and deep recesses (arrows) in noncompaction cardiomyopathy

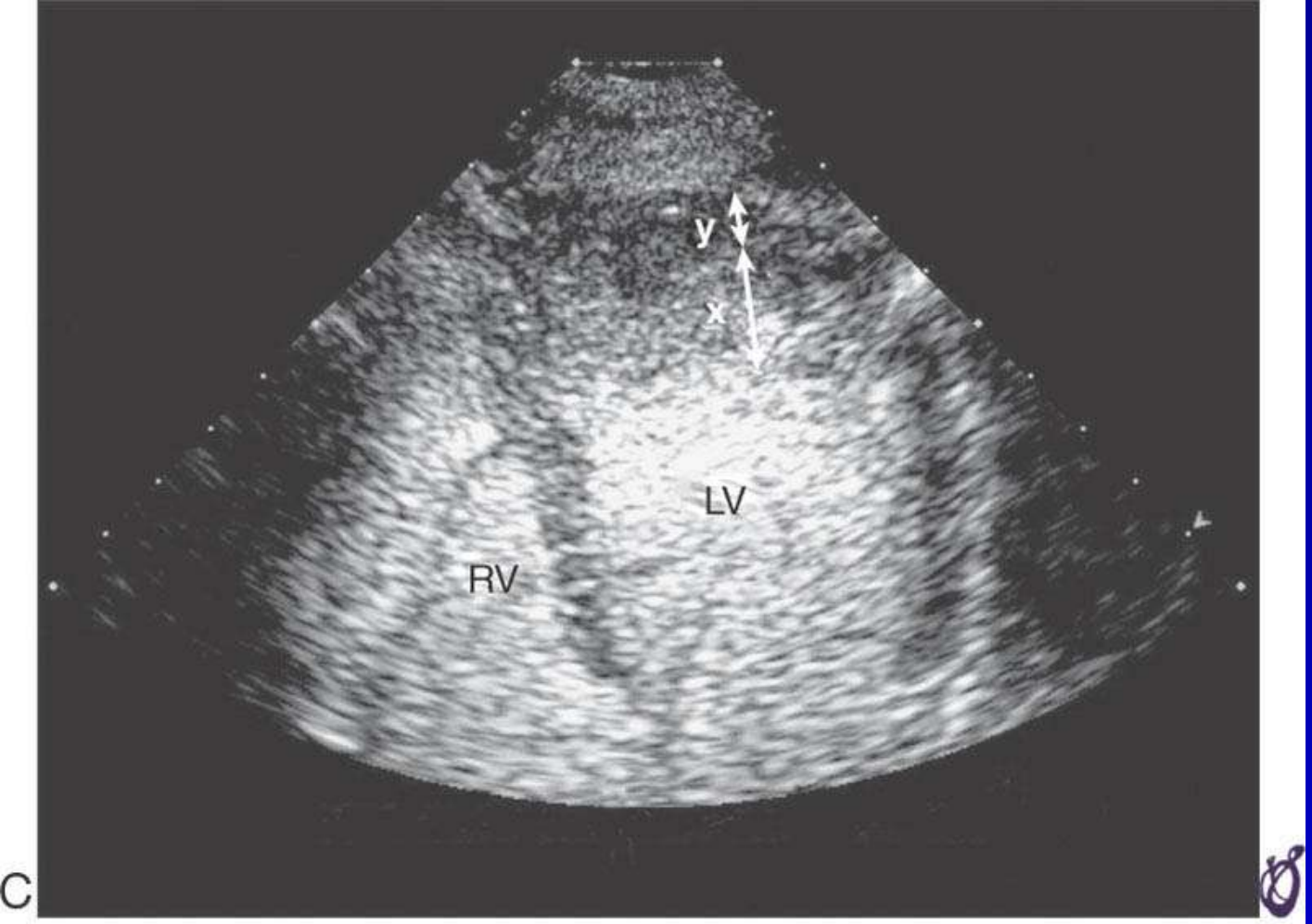
Fig 14-82A, Braunwald's 8th ed.



(From Oh JK, Seward JB, Tajik AJ: The Echo Manual. 3rd ed. Philadelphia, Lippincott Williams & Wilkins, 2006. Used with permission of Mayo Foundation for Medical Education and Research.)

Apical four-chamber 2D echocardiographic view in noncompaction cardiomyopathy. Color flow imaging showing flow into the intratrabecular recesses.

Fig 14-82B, Braunwald's 8th ed.



(From Oh JK, Seward JB, Tajik AJ: The Echo Manual. 3rd ed. Philadelphia, Lippincott Williams & Wilkins, 2006. Used with permission of Mayo Foundation for Medical Education and Research.)

Contrast administration shows trabeculations at the apex; the appearance is different from that of apical hypertrophic cardiomyopathy. With contrast enhancement, the ratio of noncompacted layer (x) to compacted layer (y) can be calculated. In this patient, x = 1.8 cm, y = 0.8 cm, with x/y = 2.3.

Fig 14-82C, Braunwald's 8th ed.