Coronary Anomalies & Hemodynamic Identification

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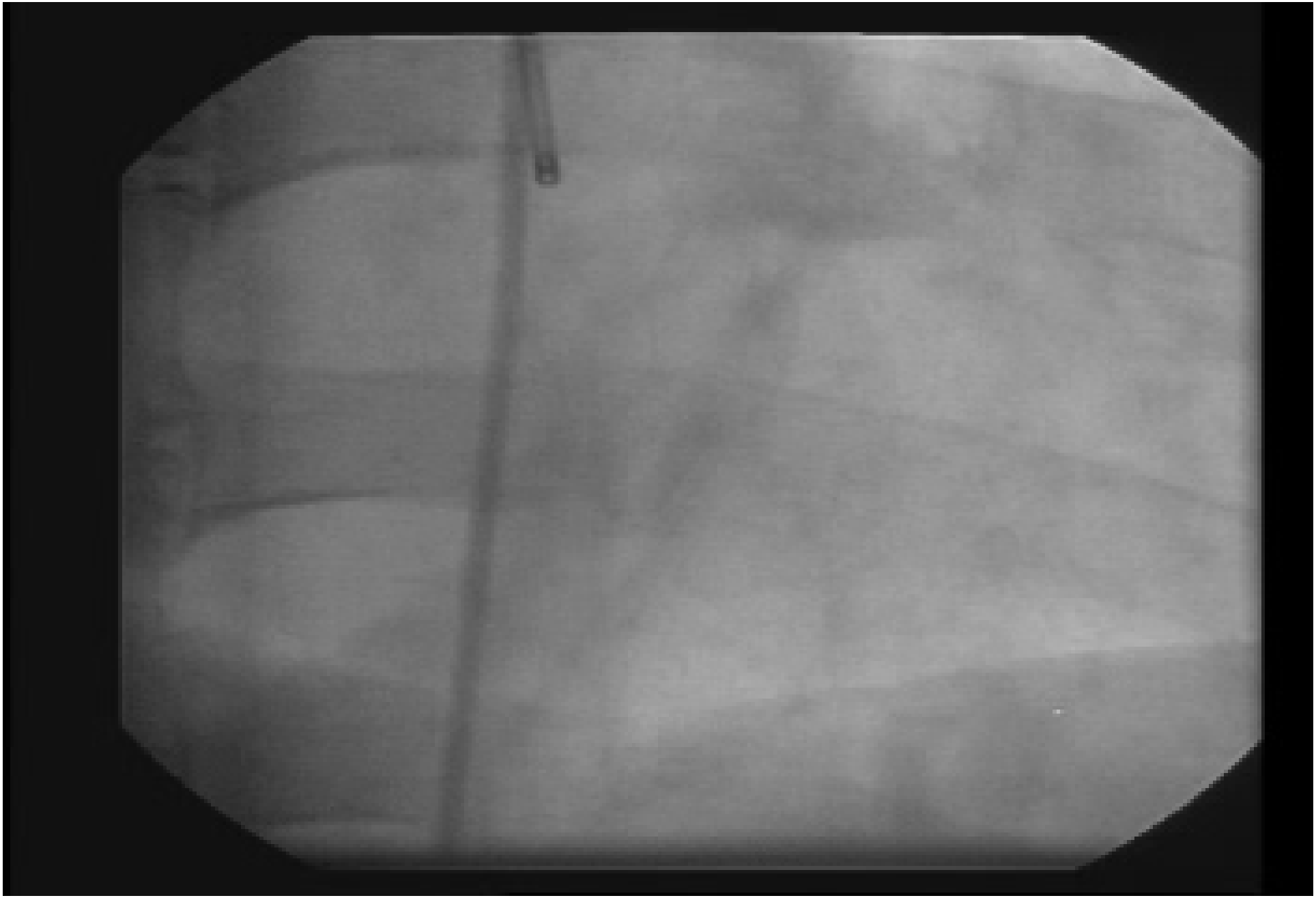
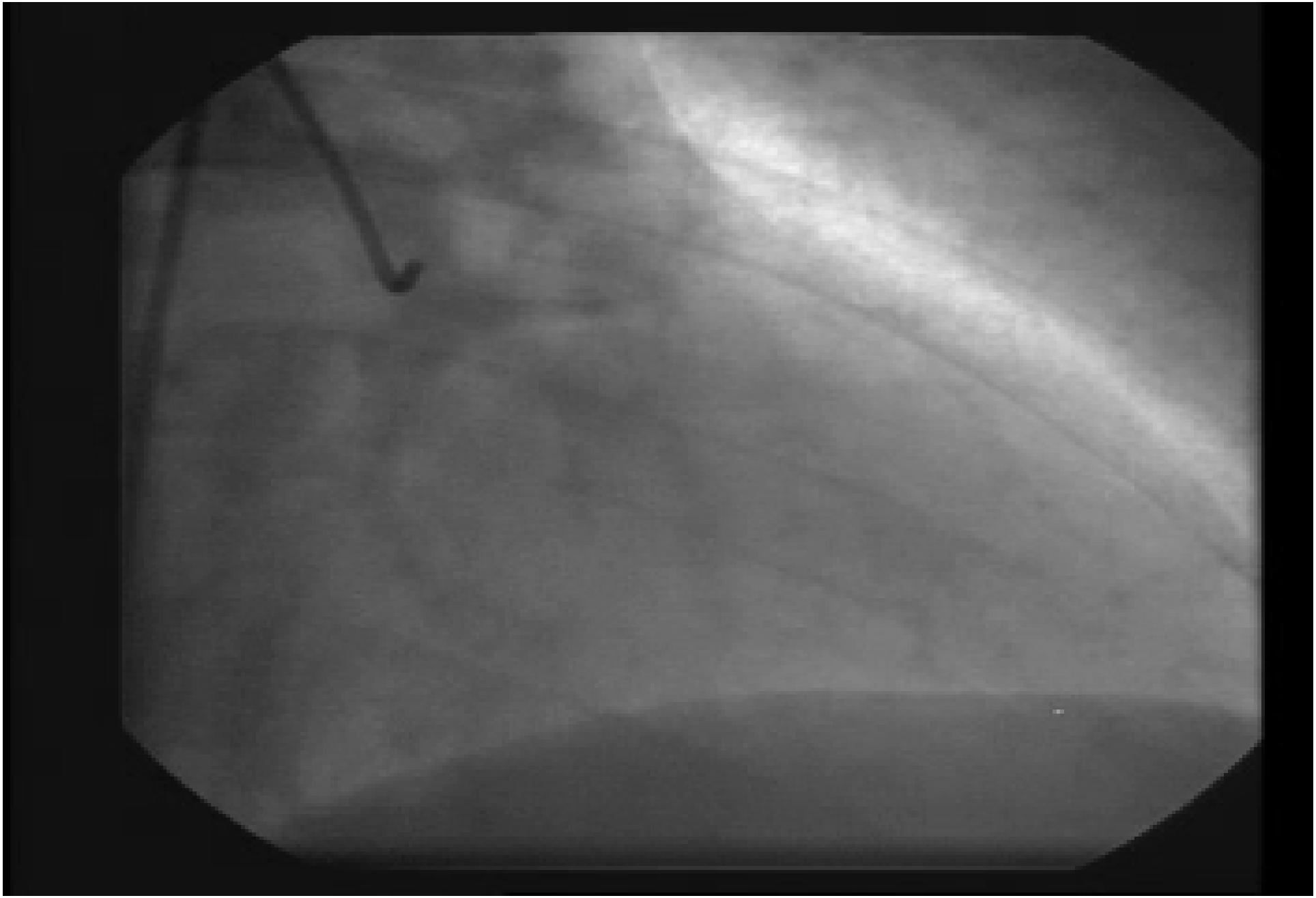
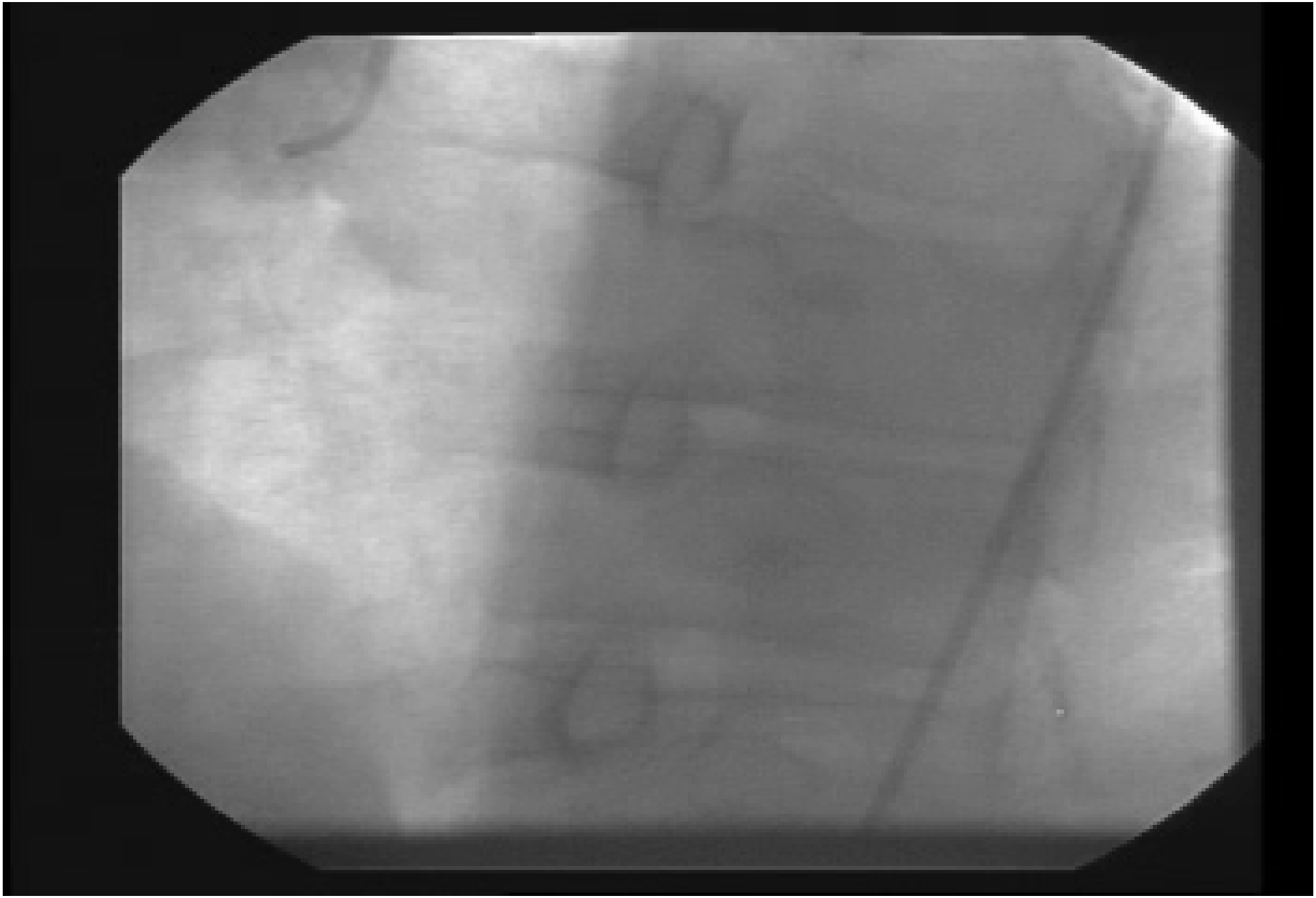
Cardiology Fellow, PGY 6

May 2, 2006

Anomaly #1







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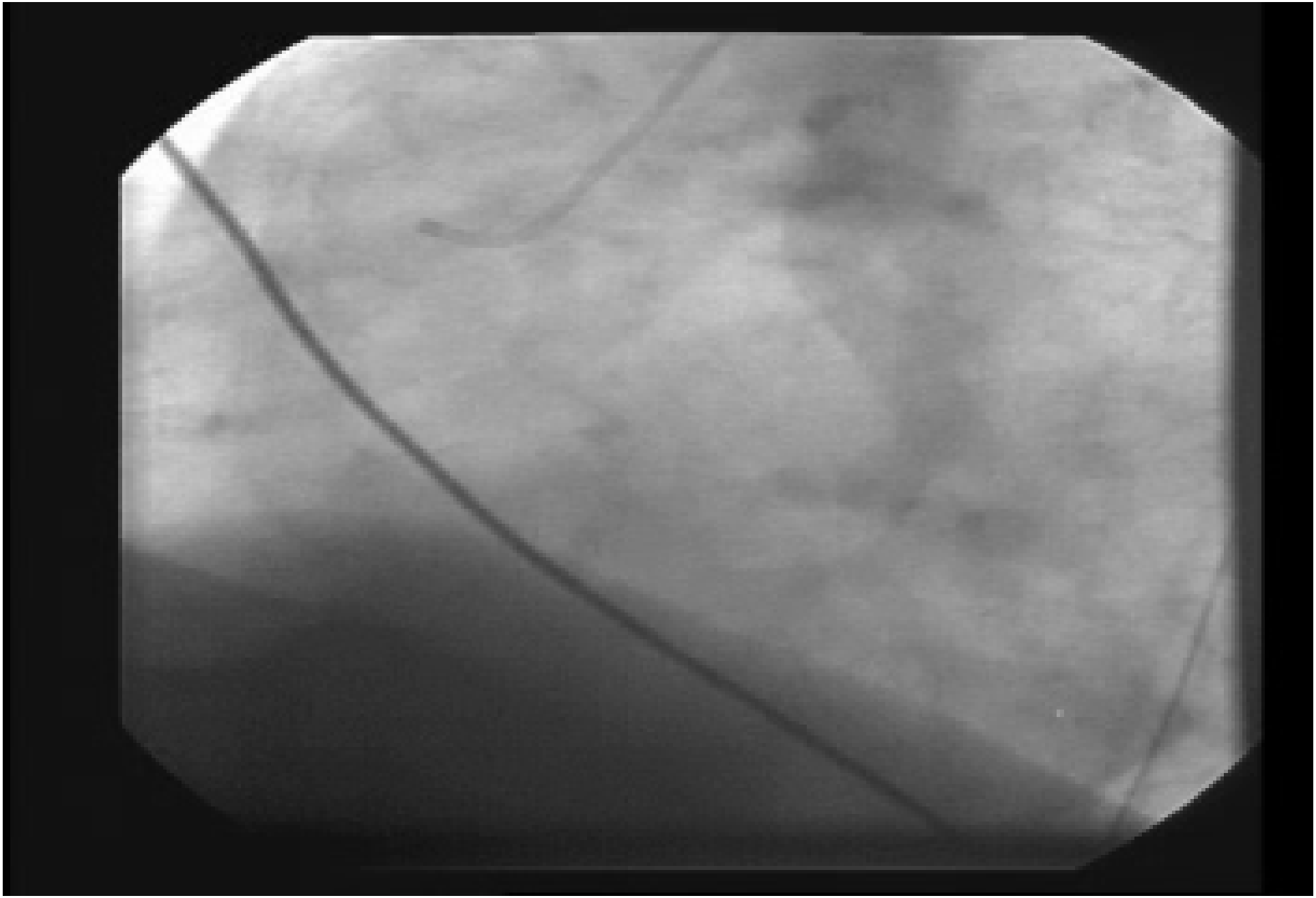

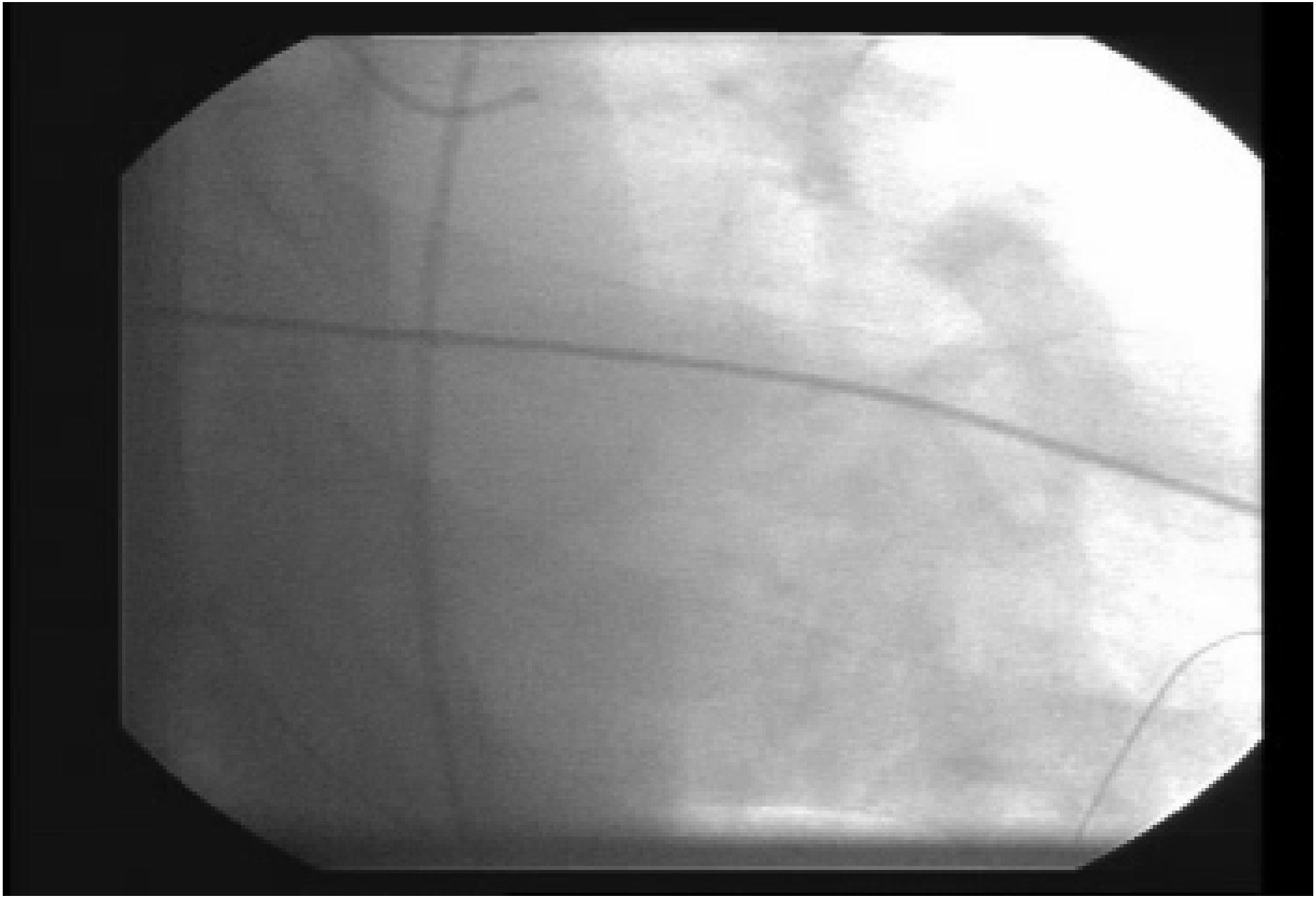

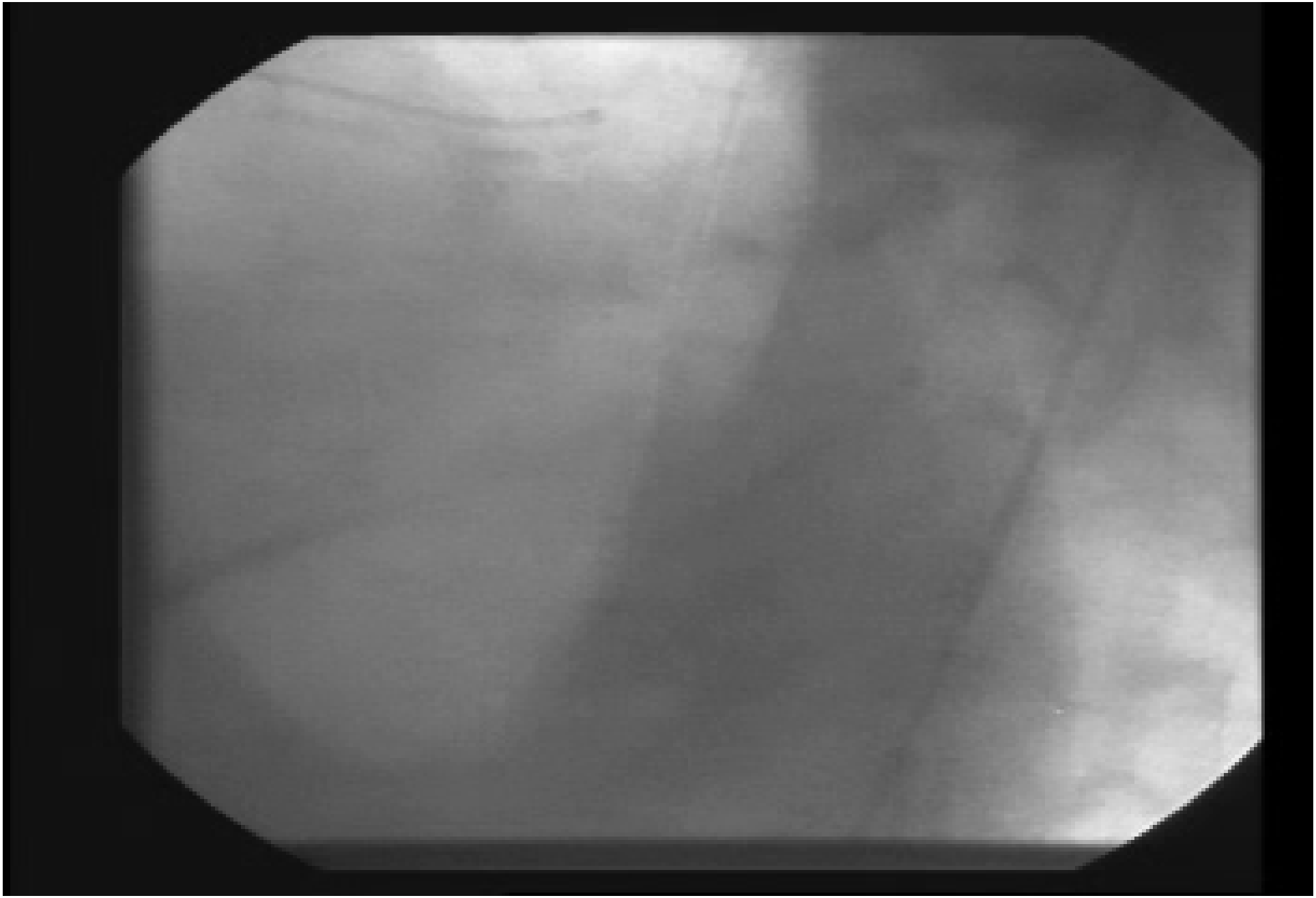

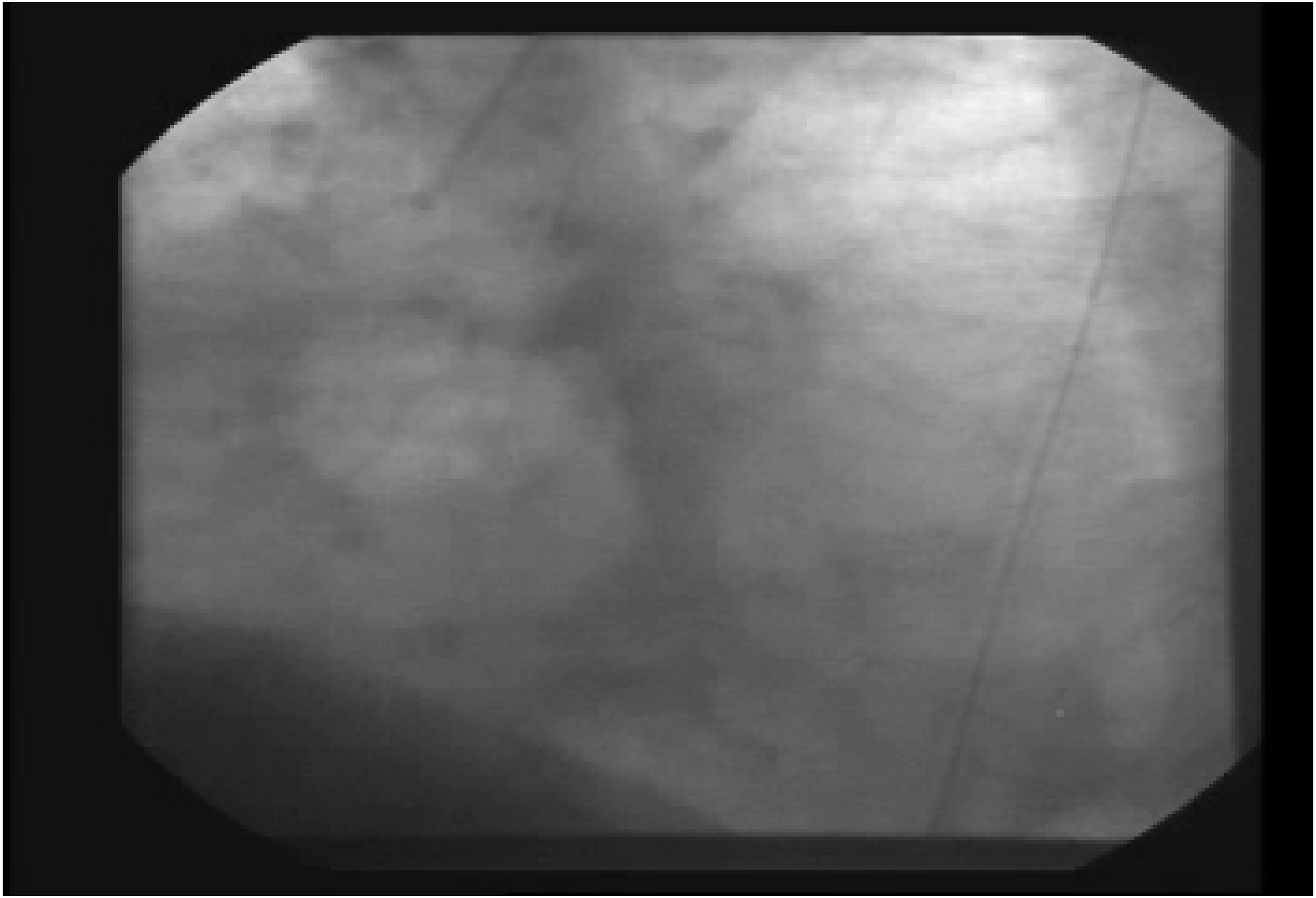

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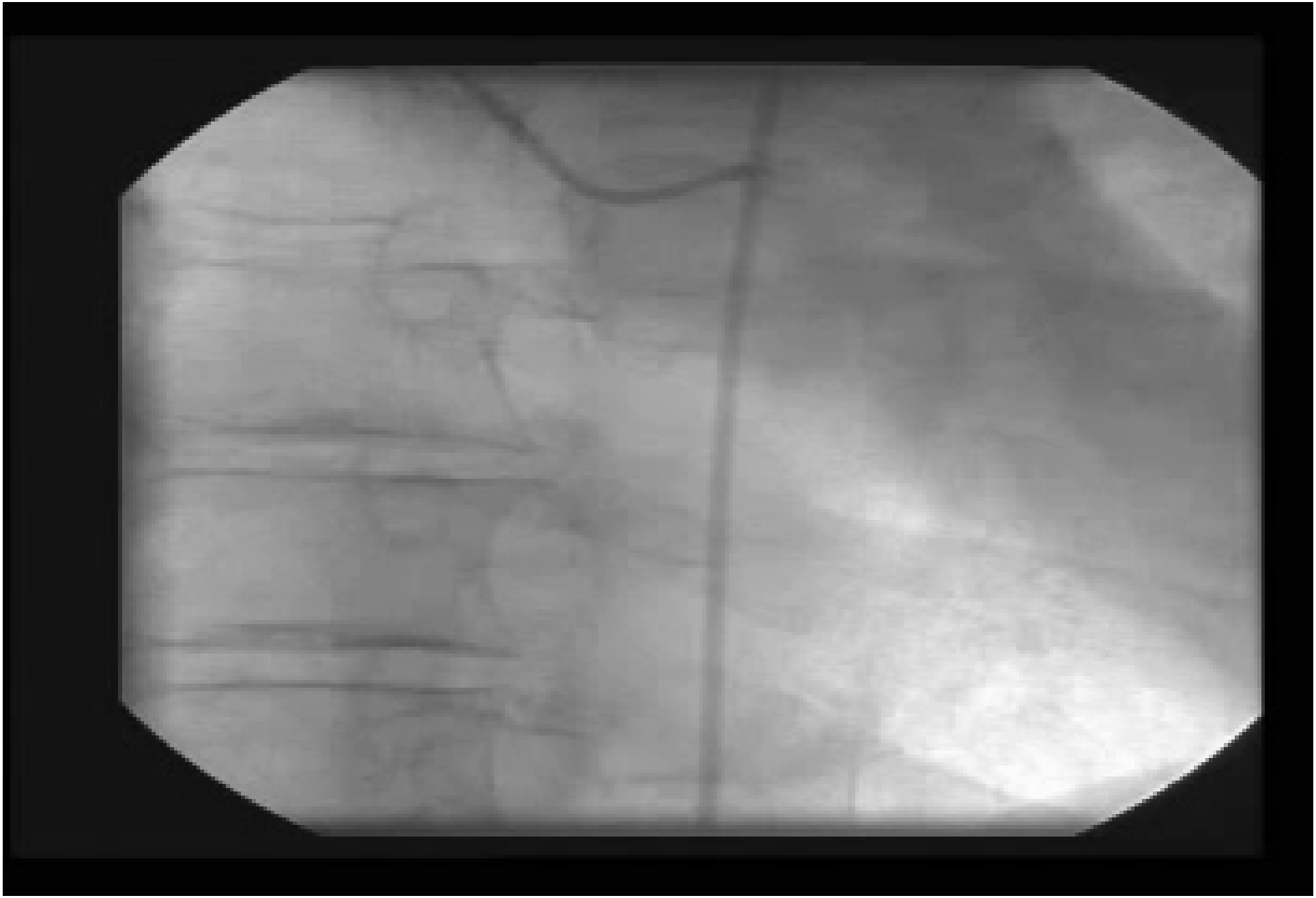
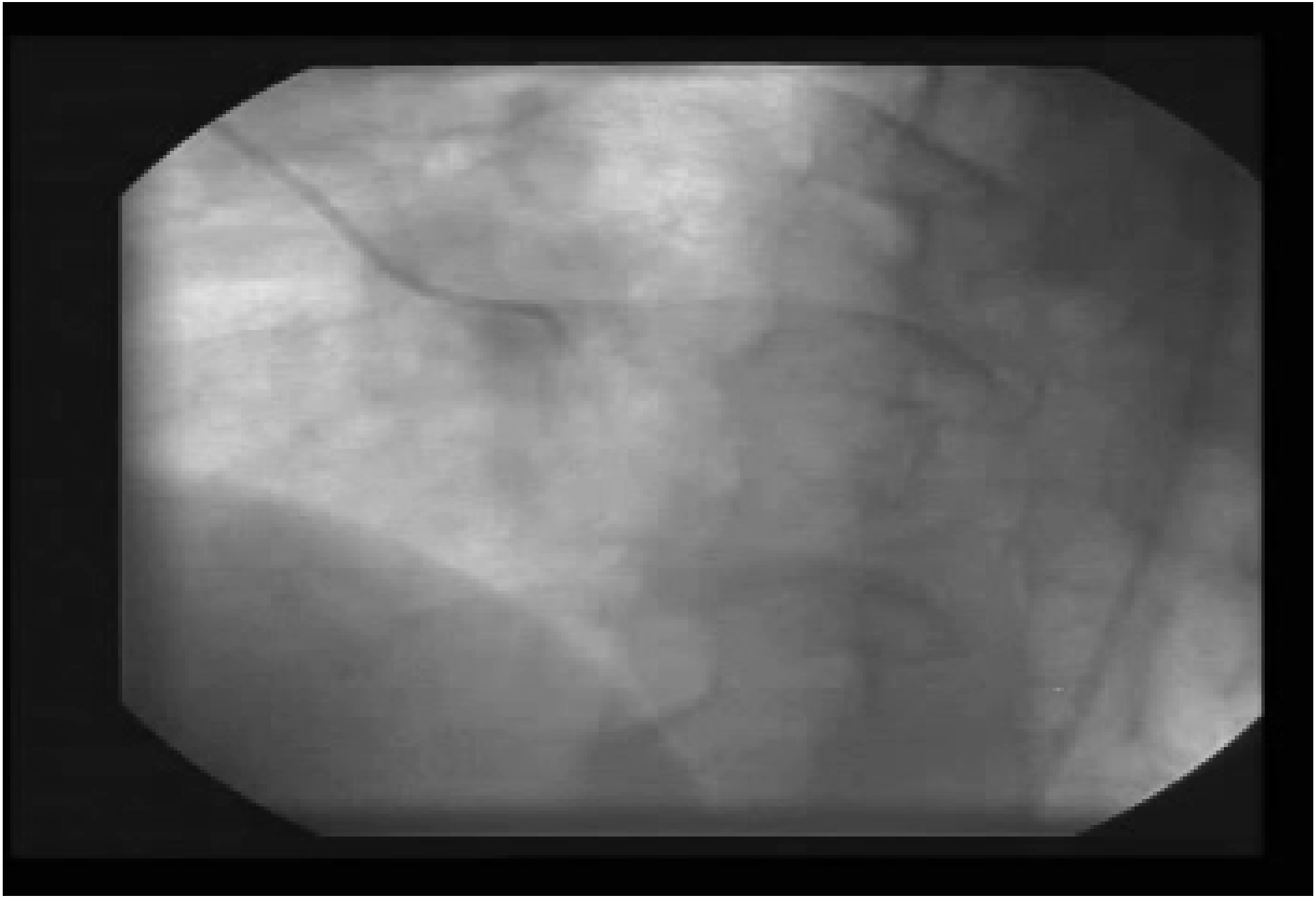
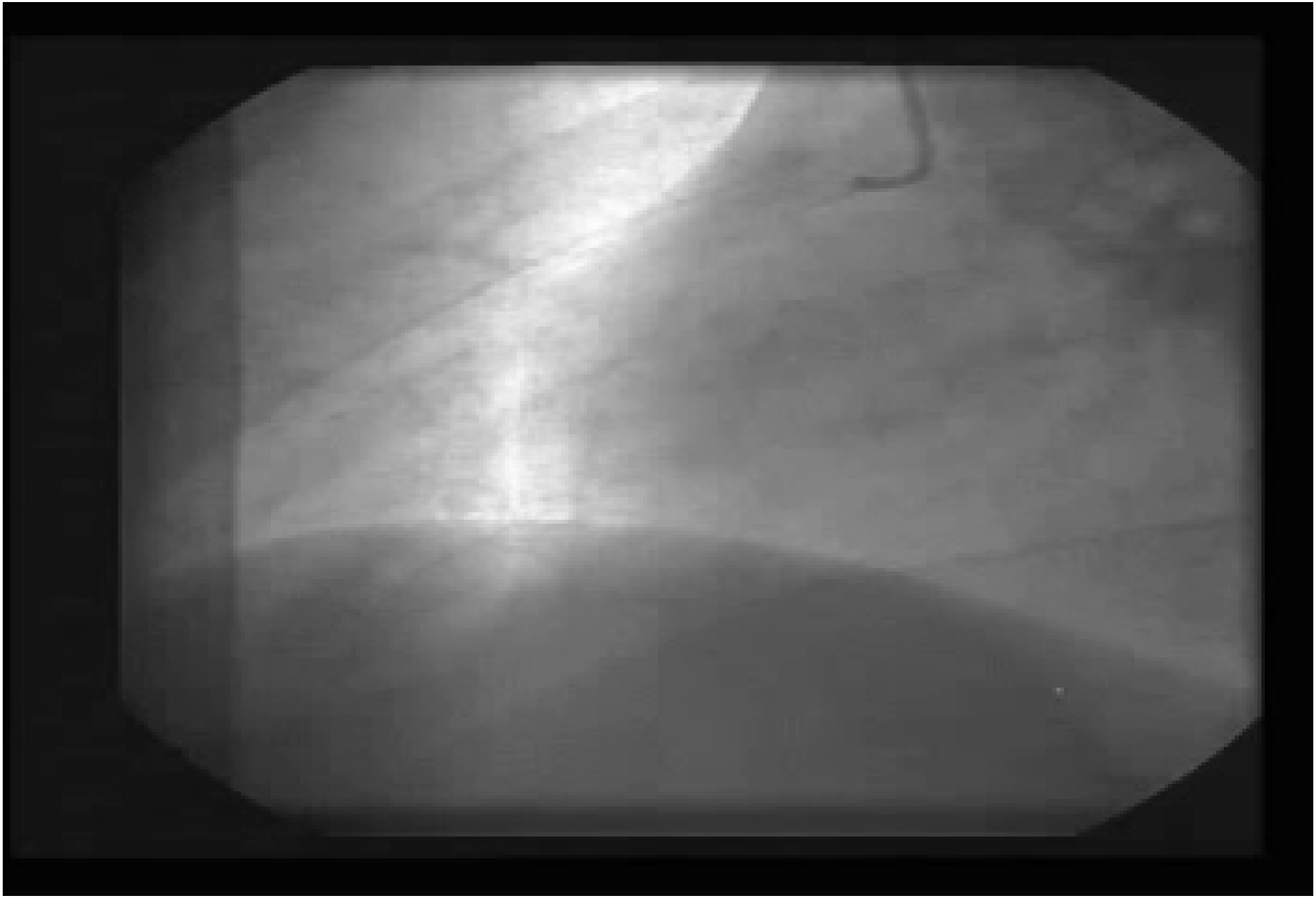
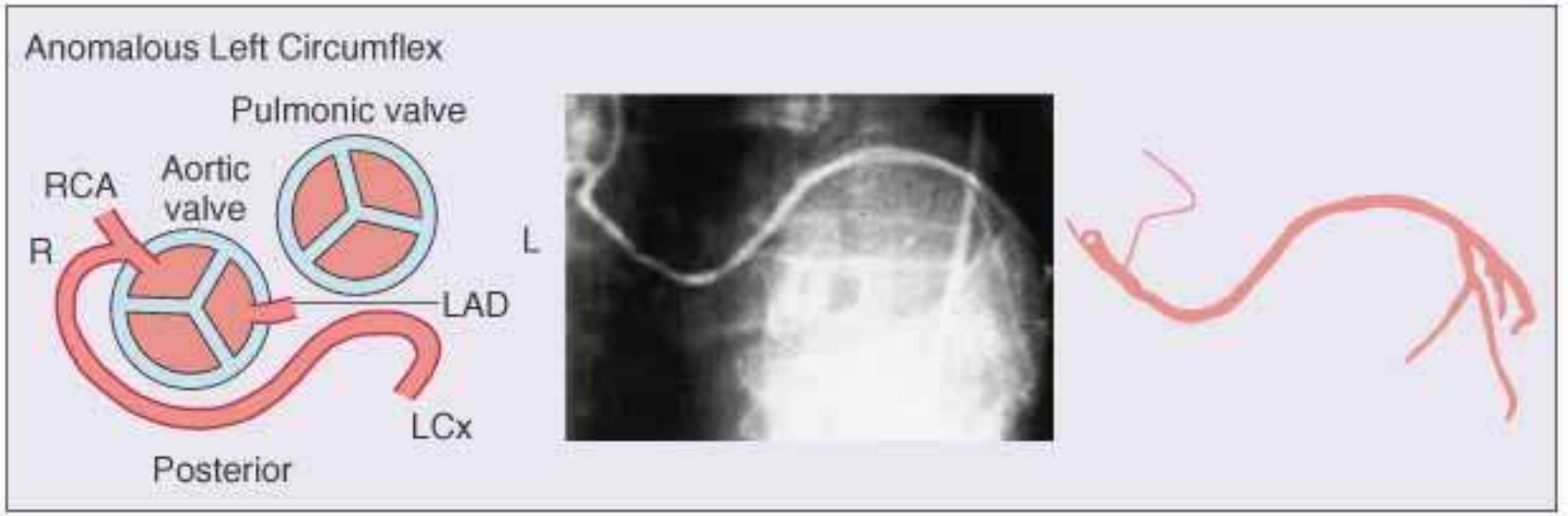


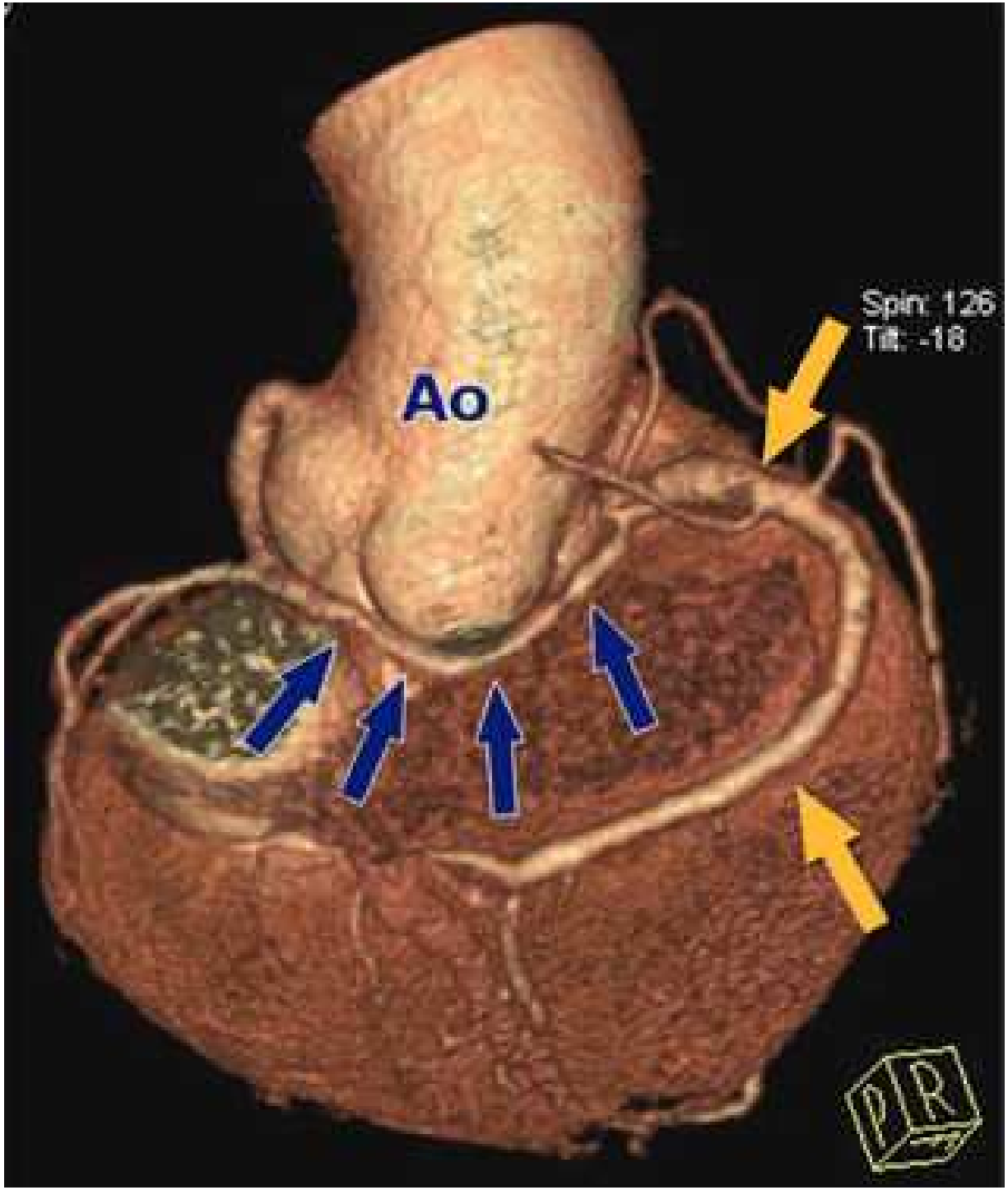




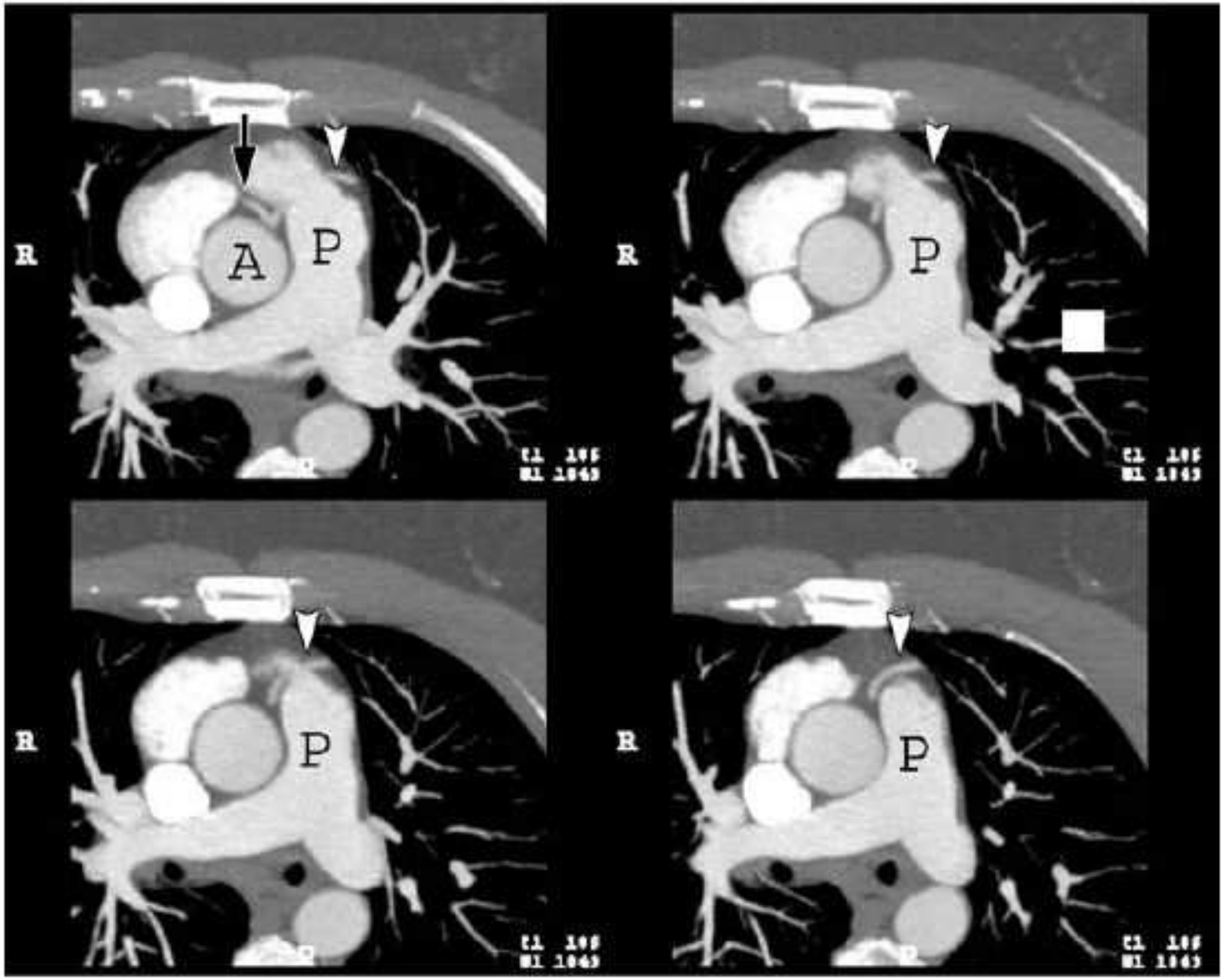


- **Figure 18-27** Anomalous origin of the left circumflex artery. The caudocranial cross-sectional view at the level of the semilunar valves shows the common course of the left circumflex coronary artery aberrantly arising from the right sinus of Valsalva. The left circumflex artery passes behind the aortic root and runs to the left atrioventricular groove following an initial course identical to that for the anomalous left coronary artery arising from the right sinus of Valsalva that follows a posterior, retroaortic course. LAD = left anterior descending; LCx = left circumflex; RCA = right coronary artery.

Cx from RCA Posterior course



Single coronary, right cusp
Cx anterior to pulmonary artery



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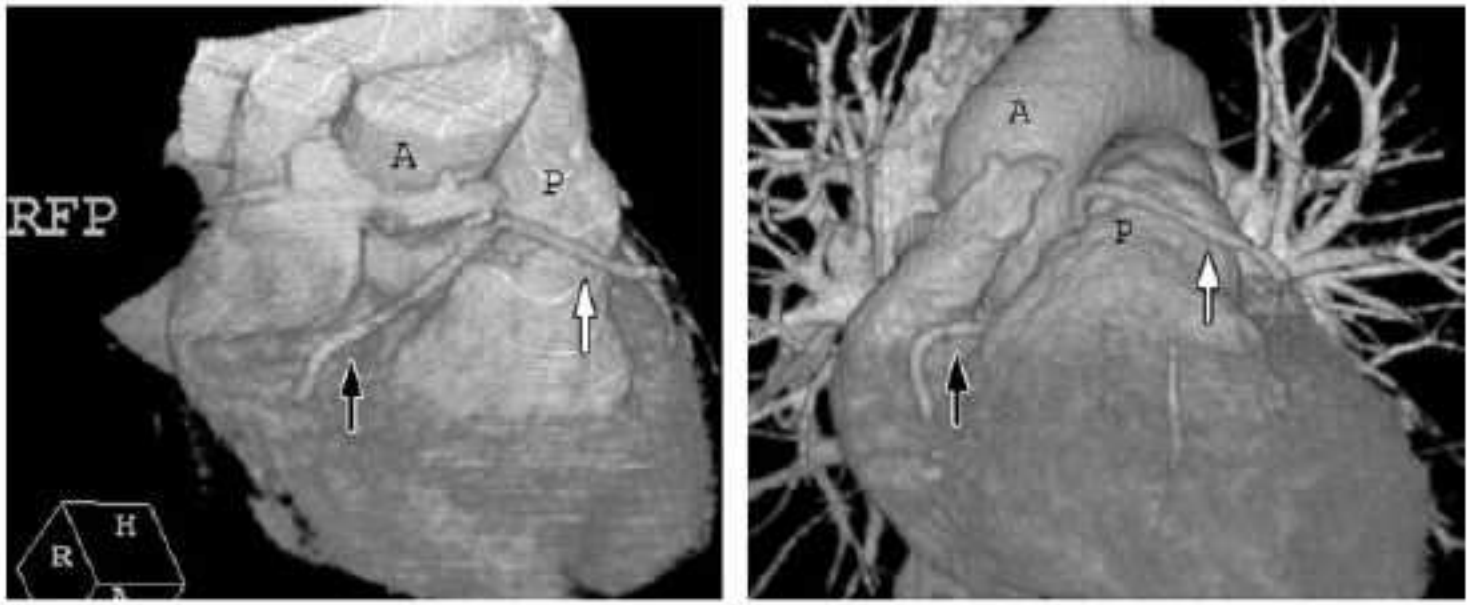
Single coronary, right cusp
Cx Anterior to pulmonary artery



b. c.

Figure 2. Images obtained in a 48-year-old woman who underwent coronary angiography for progressive angina and was found to have a single coronary artery arising from the right cusp. *A* = aorta. **(a)** Transverse CT scans obtained with a four-section multi–detector row CT unit show the origin of the common coronary artery and the separate courses of the right (arrow) and left (arrowhead) coronary arteries. The left coronary artery crosses anterior and superior to the main pulmonary artery *(P)*. **(b, c)** Two volume-rendered images obtained with oblique (left) and anterior (right) projections show the left coronary artery (white arrow) passing anterior and superior to the main pulmonary artery *(P)*. Black arrow indicates the right coronary artery.

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Anomaly #4
a different kind


ARITAN HOSP R2


ARITAN HOSP R2


ARITAN HOSP R2

Left Main from right coronary cusp

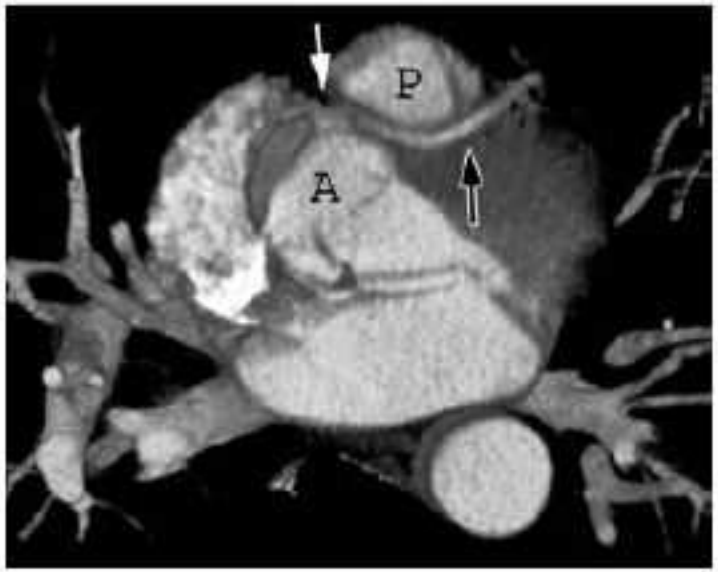

Figure 3. Patient 5. Reconstructed image obtained in a 44-year-old man with chest pain. Coronary angiography showed a shared origin of the right and left coronary arteries from the anterior cusp. CT was performed to define the course of the left main artery. Thick-slab transverse reconstruction (10-mm-thick section) shows the shared anterior orifice (white arrow) and the left coronary artery (black arrow) coursing between the aorta *(A)* and the main pulmonary artery *(P)*. Note the acute angulation of the left coronary artery at its origin. The patient underwent bypass grafting.

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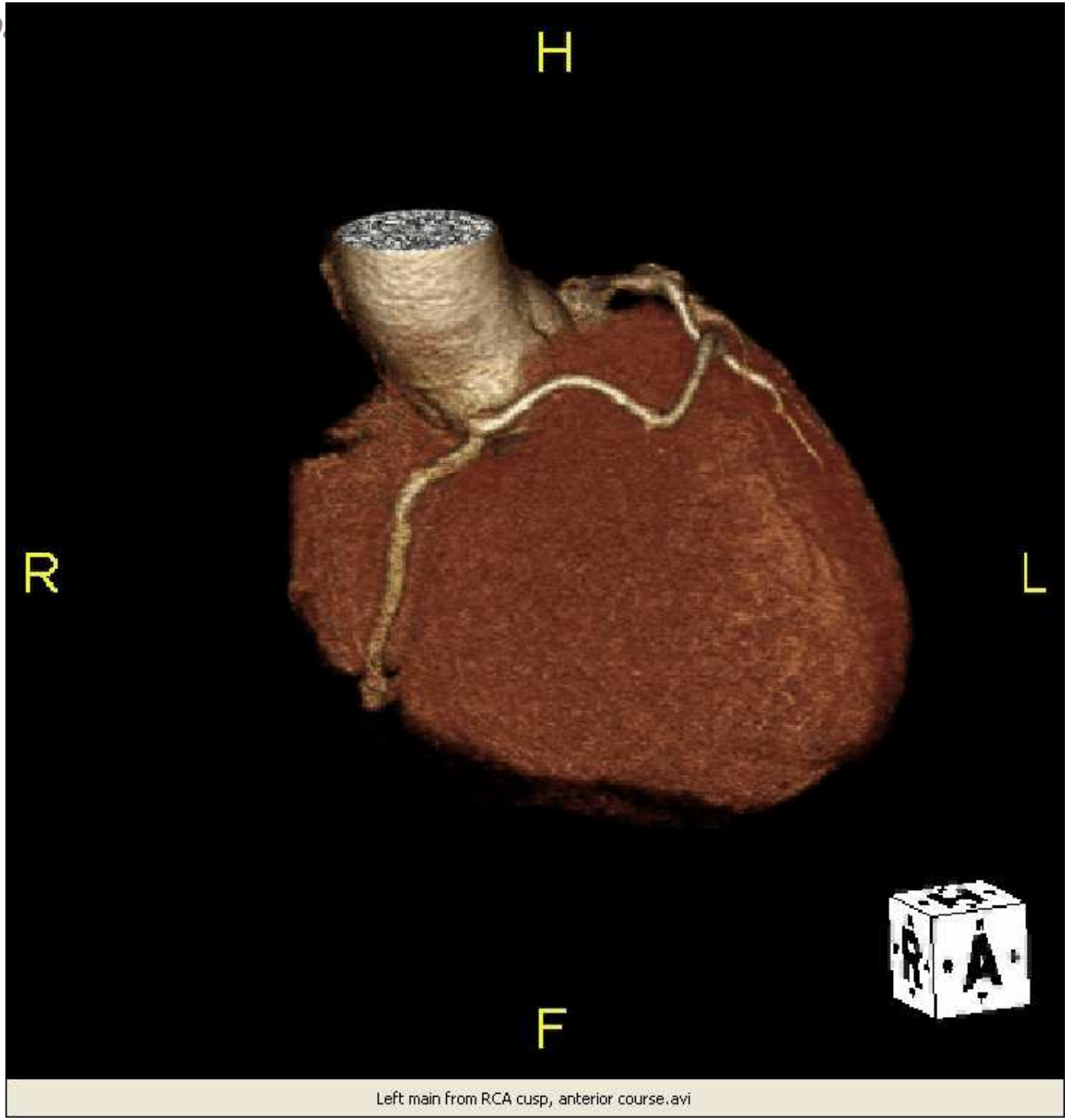


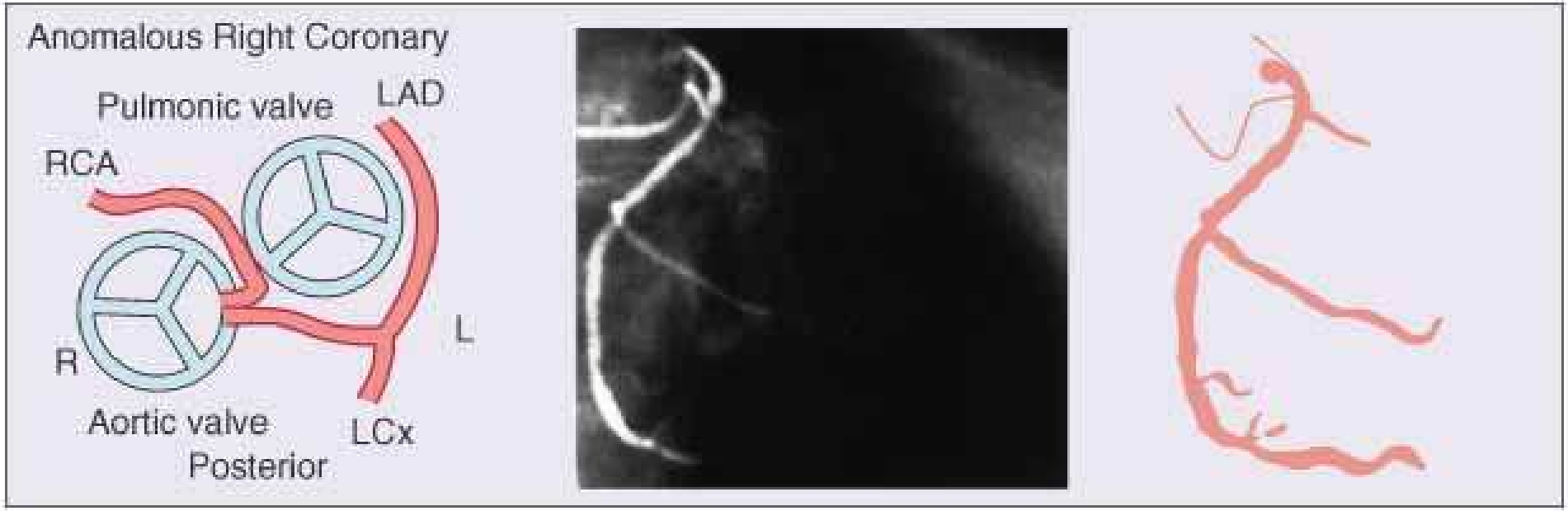
Coronary anomalies

- Occur in about 1 in 200 caths
- Most common: Cx from Right coronary cusp
- Anomalies causing ischemia:
  - Coronary artery fistulas
  - Left coronary artery origin from PA
  - Congenital stenosis or atresia
  - Origin from either coronary from the contralateral sinus
  - Single coronary artery
    - Especially when vessel courses between Ao, PA
- Benign anomalies
  - Left Cx from Right coronary sinus
  - High anterior RCA origin

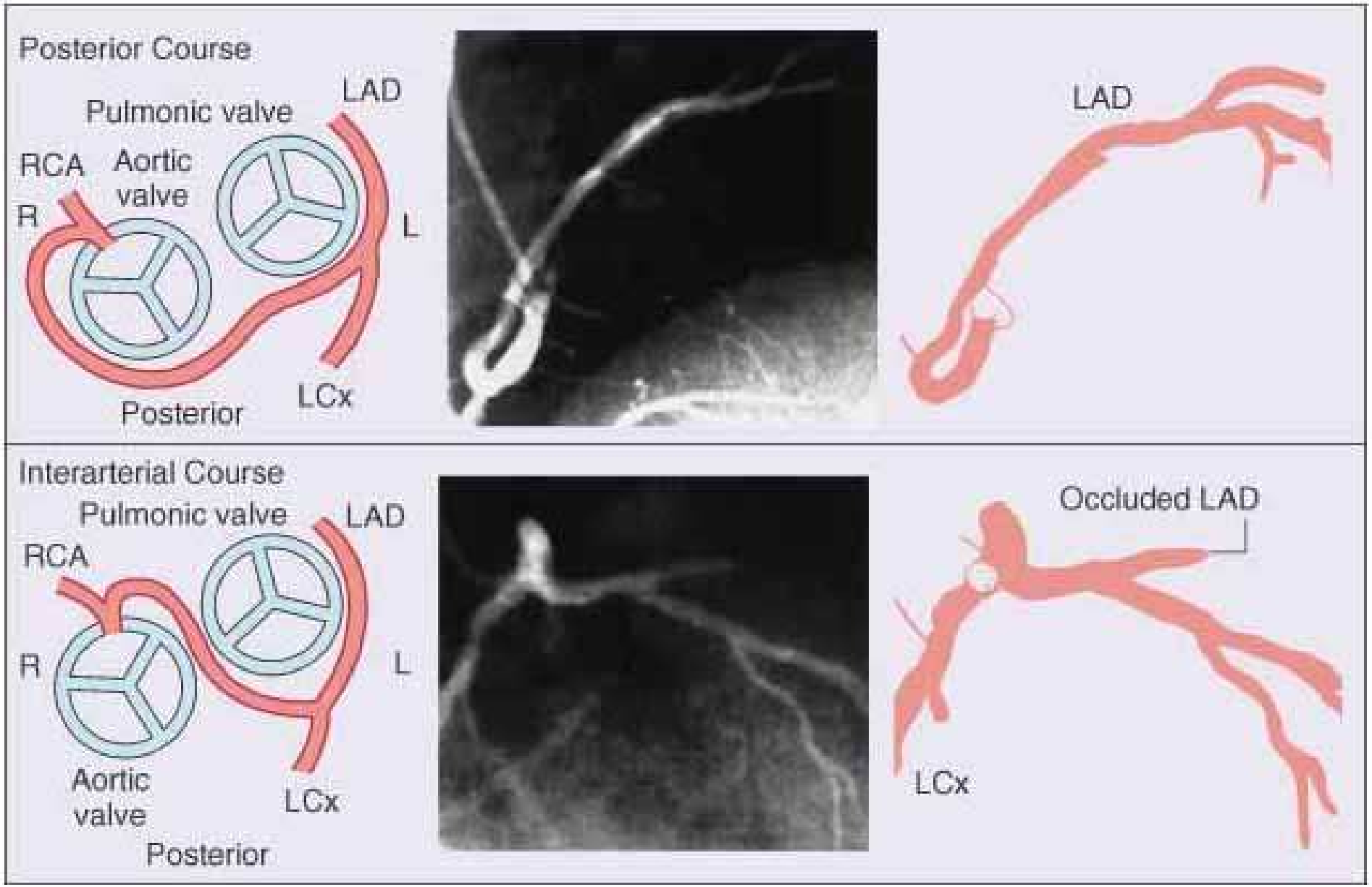
RELATIVE FREQUENCY OF CORONARY ARTERY ANOMALIES IN ANGIOGRAPHY AND PATHOLOGY SERIES			
Anomaly		Angiography series (%) (n=799)	Pathology series (%) (n=242)
Anomalous origin of one or more coronary arteries from the pulmonary trunk	Left main coronary artery or left anterior descending artery from the pulmonary trunk	1.4	15.3
	Both coronary arteries from the pulmonary trunk		1.2
	Right coronary artery from the pulmonary trunk	0.2	0.4
Anomalous origin of one or more coronary arteries from the aorta	Left main coronary artery and right coronary artery from the right aortic sinus	2.8	20.2
	Right coronary artery and left main coronary artery from the left aortic sinus	17.0	21.5
	Left circumflex artery and right coronary artery from the right aortic sinus	58.4	8.7
	Right coronary artery and/or left main coronary artery from the posterior aortic sinus	0.6	7.0
	Right coronary artery and left anterior descending artery from the right aortic sinus	4.8	0.4
Single coronary artery ostium from aorta	Single right coronary artery ostium	3.1	9.1
	Single left coronary artery ostium	3.9	9.1
Hypoplastic coronary artery			5.4
Coronary artery fistula		7.8	1.7

Crawford and DiMarco, 2nd ed

RCA from Left cusp



LAD from Right Cusp patterns



LAD from RCA, septal location

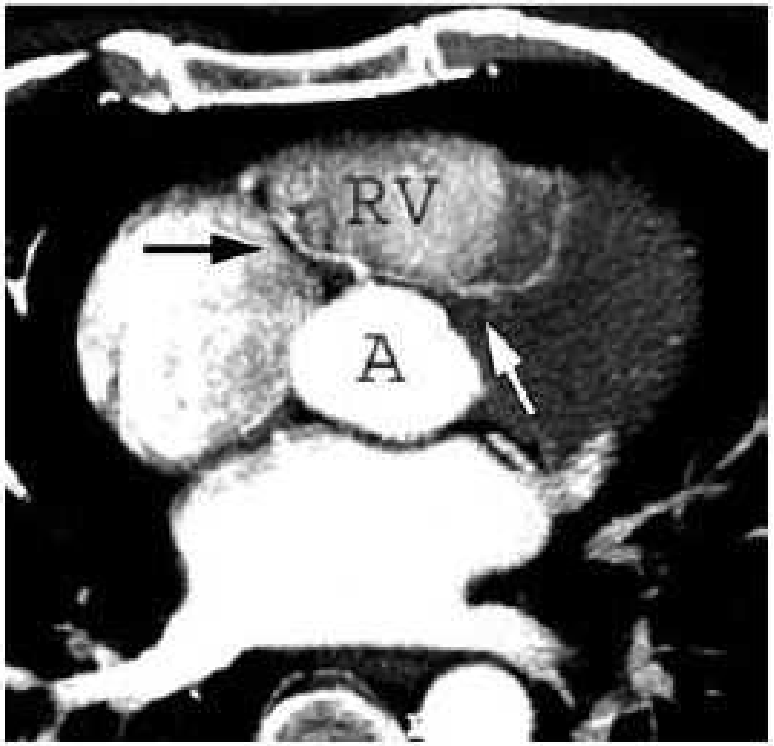


Figure 4. Patient 4. Transverse CT scan obtained in a 59-year-old man with chest discomfort. Although results of coronary angiography were suggestive of an anomalous left coronary artery, the relationship to the great vessels was uncertain. Transverse image obtained with a 16-section CT unit shows the left anterior descending coronary artery (white arrow) arising from the right coronary artery (black arrow) anterior to the aorta *(A)*. The anomalous vessel courses between the aorta and right ventricular outflow track *(RV)*. The course of the artery is more inferior than that illustrated in Figure 3. In addition, the vessel courses through muscle, which is consistent with an intraseptal location.

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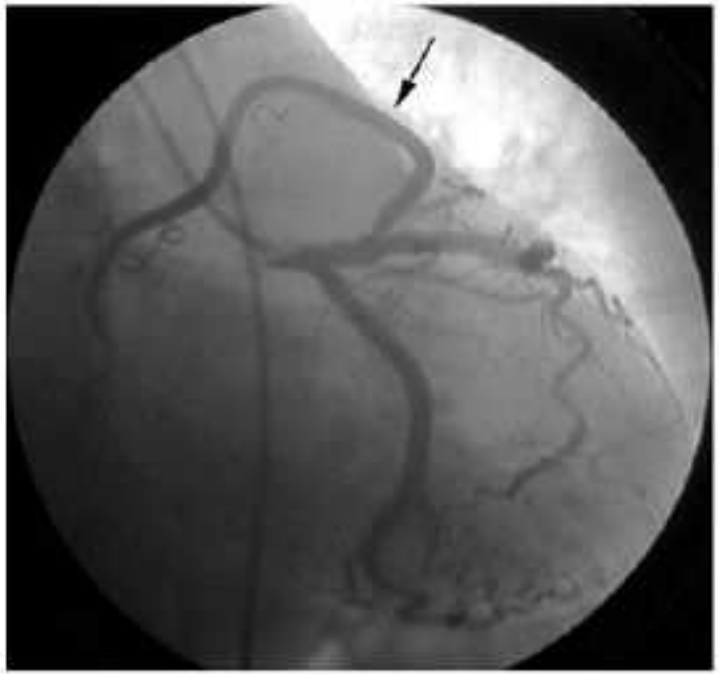
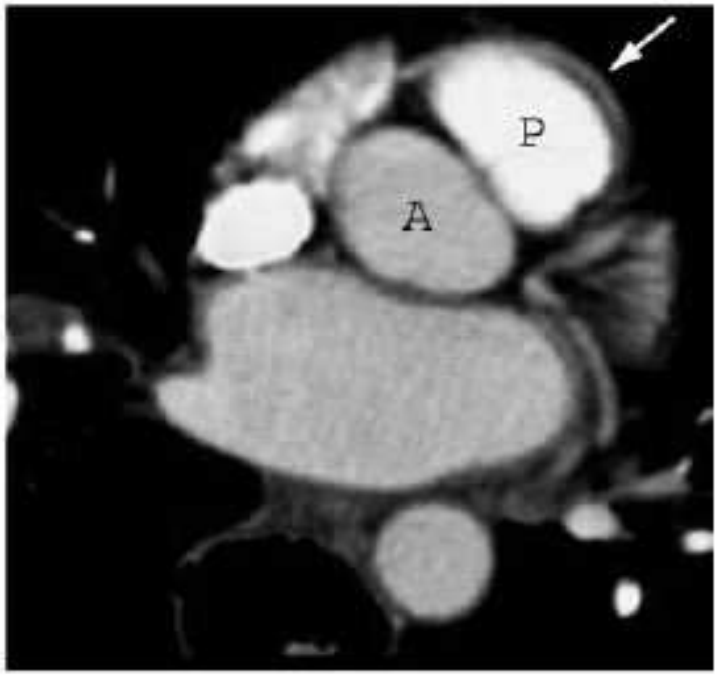
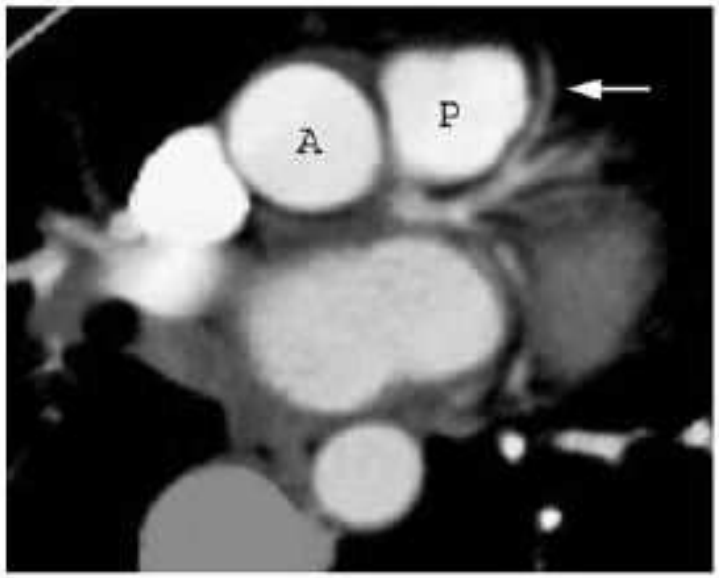
RCA from LAD, anterior to PA



a.



c.



b.

Figure 1. Images obtained in a 45-year-old woman with recurrent chest pain and palpitations. (a) Coronary angiogram obtained with a right anterior oblique projection shows an anomalous right coronary artery (arrow) originating from the proximal left anterior descending artery and extending to the anterior atrioventricular groove. (b, c) Transverse CT scans obtained with a four-section scanner show the anomalous right coronary artery (arrow) arising from the left anterior descending artery and coursing anterior to the main pulmonary artery *(P)* into the anterior atrioventricular groove. *A* = aorta.

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RCA from Left coronary Courses between Ao and PA

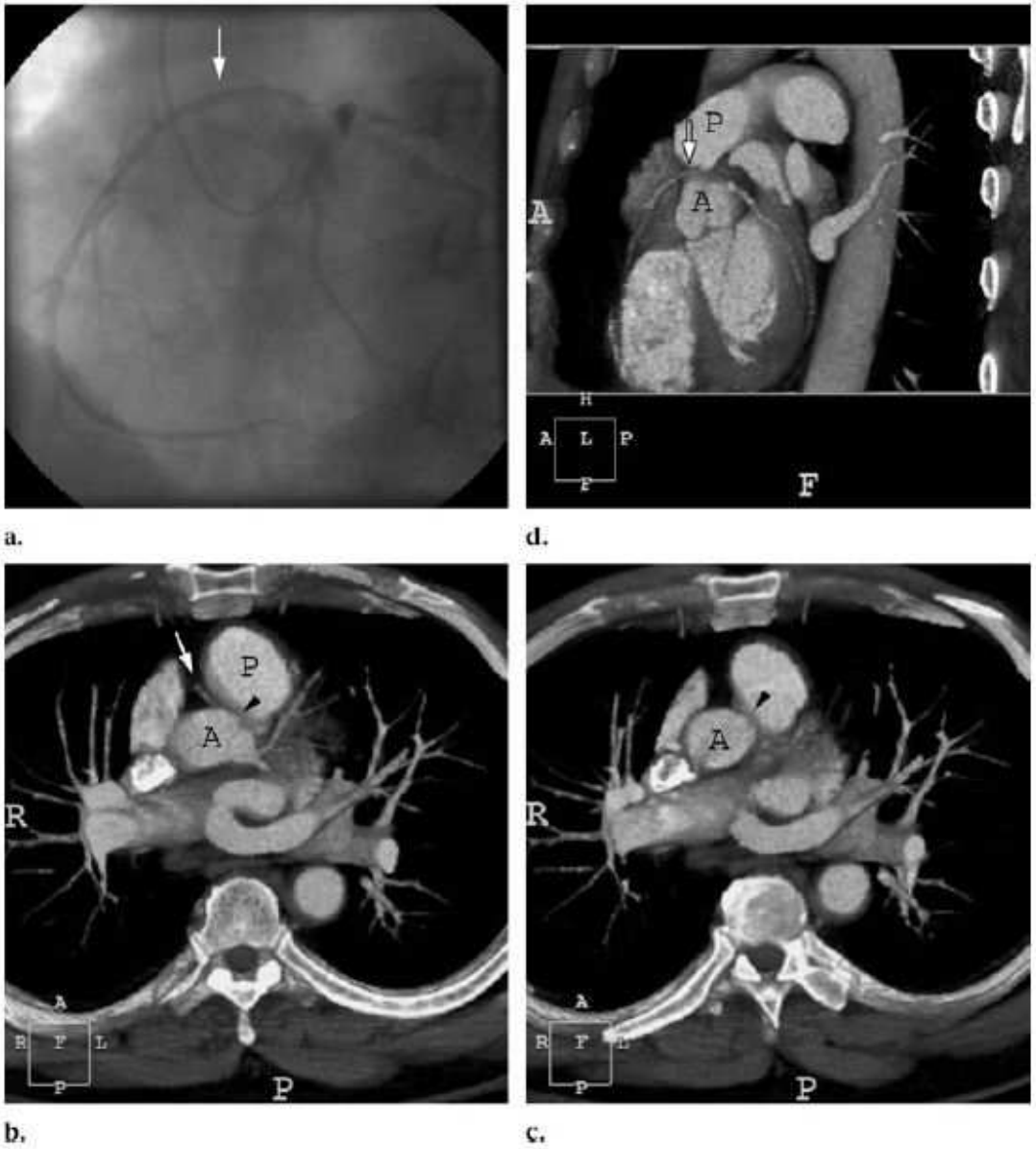

Figure 5. Images obtained in a 28-year-old man with hypertension, recurrent chest pain, and an anomalous right coronary artery. **(a)** Coronary angiogram obtained in a left anterior oblique projection shows the anomalous right coronary artery (arrow) originating from the left coronary artery. **(b, c)** Contiguous transverse thick-slab reformations (10-mm-thick section) from a four-section CT unit show the right coronary artery (arrow in **b**) originating from the left cusp (arrowhead) and extending between the aorta *(A)* and the main pulmonary artery *(P)* to reach the anterior atrioventricular groove. **(d)** Volume-rendered image demonstrates the anomalous vessel (arrow) extending from the left cusp anteriorly. *A* = aorta, *P* = main pulmonary artery.

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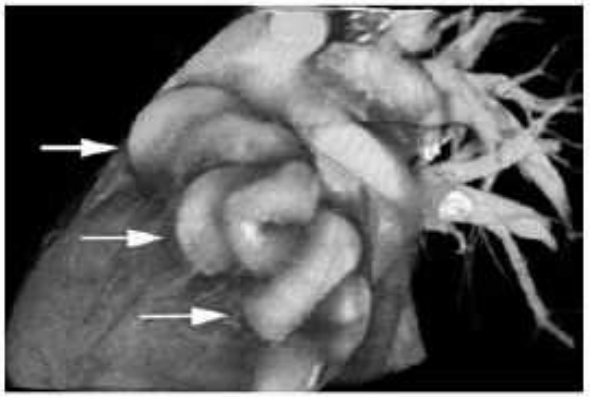
Left Circumflex (tortuous) – Great cardiac vein fistula



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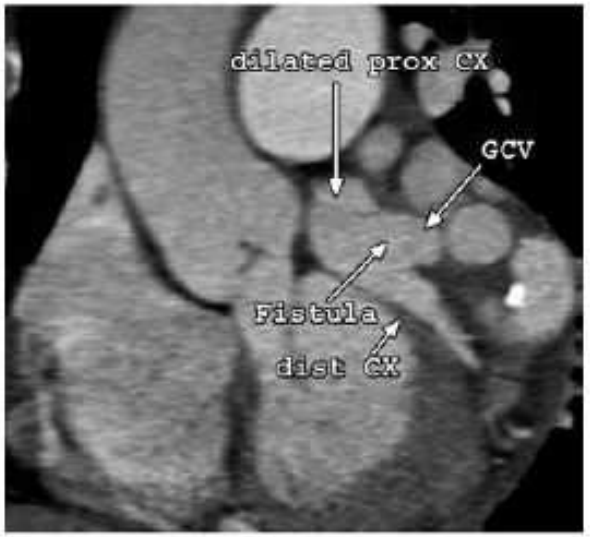

b.

Figure 6. Images obtained in a 69-year-old man with chest pain. Results of coronary angiography were suggestive of an arteriovenous fistula. **(a)** Volumetric image shows the markedly enlarged and tortuous left circumflex vessel (arrows) coursing along the posterior cardiac surface. **(b)** Parasagittal thin-slab reformatted image (5-mm-thick section) shows the junction of the left circumflex artery *(CX)* and the great cardiac vein *(GCV)*. Note the more normal-appearing distal *(dist)* circumflex artery. *prox* = proximal

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And now on to hemodynamics…

Normal arterial pulse

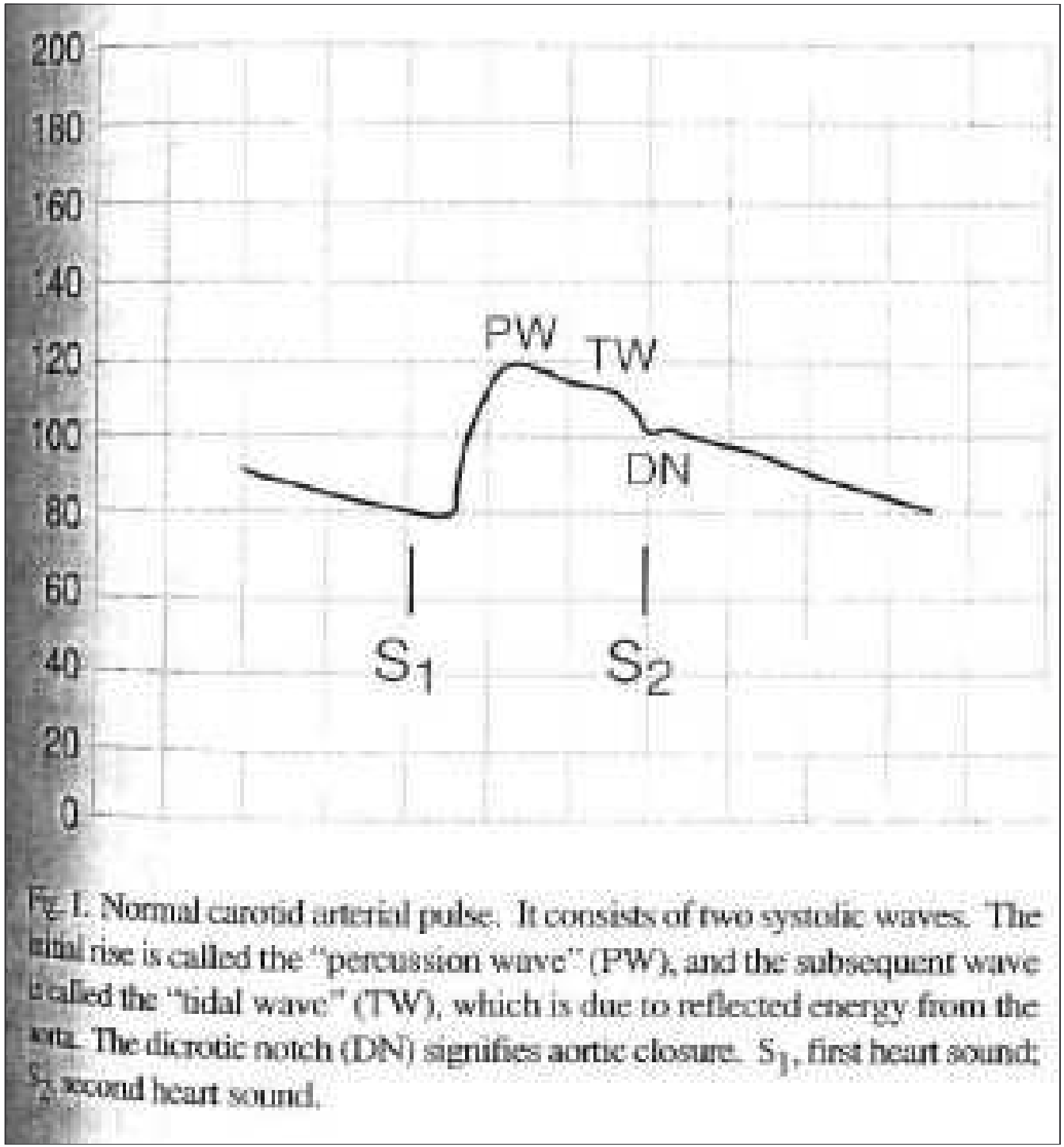

Fig. 1. Normal carotid arterial pulse. It consists of two systolic waves. The initial rise is called the "percussion wave" (PW), and the subsequent wave is called the "tidal wave" (TW), which is due to reflected energy from the aorta. The dicrotic notch (DN) signifies aortic closure. S_1, first heart sound; S_2, second heart sound.

Mayo Clnic, 2nd ed

Parvus and Tardus pulse

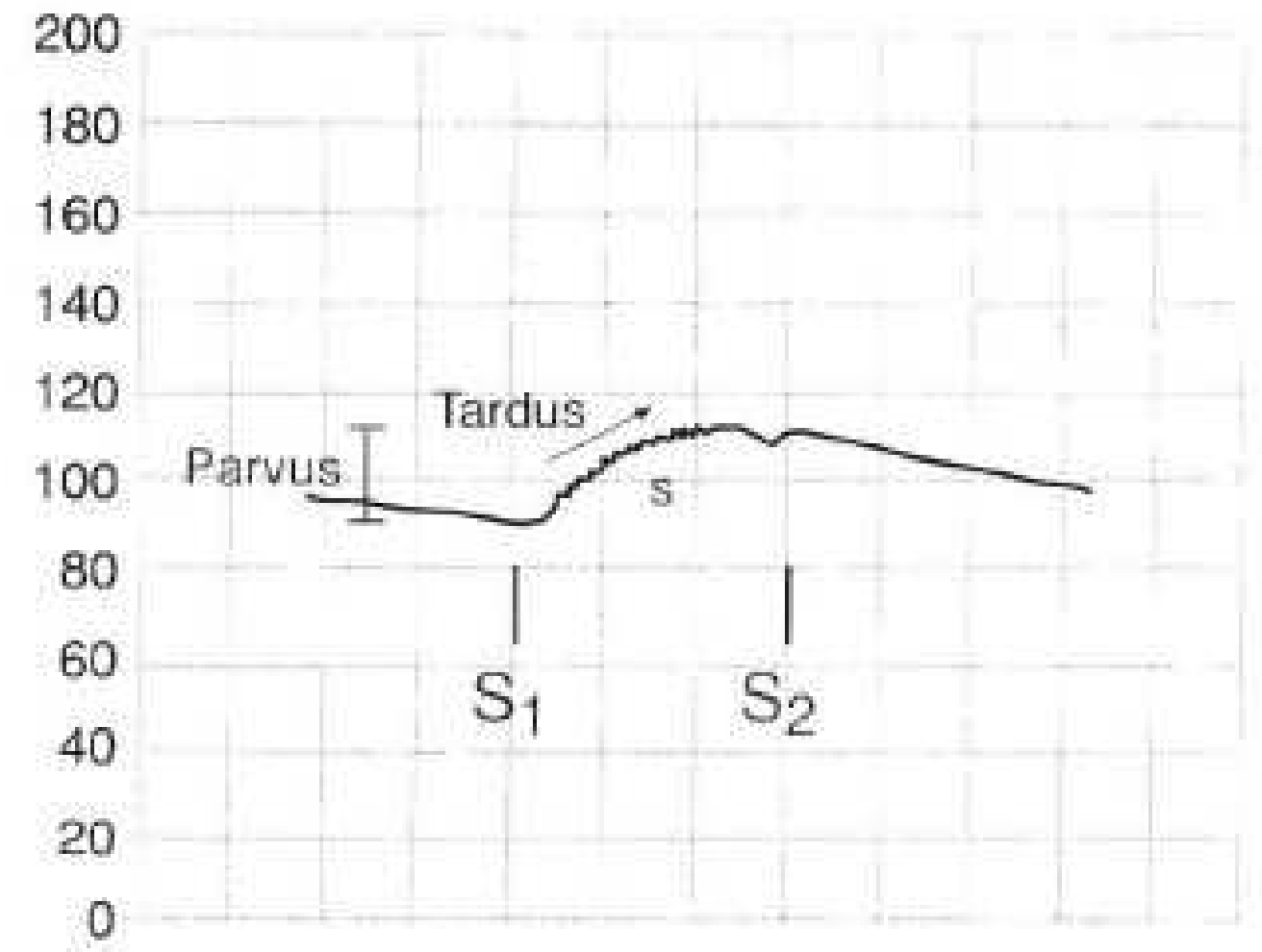

Fig. 2. The parvus (low amplitude) and tardus (slow uprising, arrow) pulse of severe calcified aortic stenosis. The irregularity in the slowly uprising pulse is from the turbulence created by the stenotic aortic valve and, on palpation, is often felt as a shudder (S) in the carotid pulse. Note that the degree to which a pulse is parvus and tardus is correlated with the severity of aortic stenosis. S_1, first heart sound; S_2, second heart sound.

Mayo Clnic, 2nd ed

Bifid pulse
Aortic stenosis and regurgitation

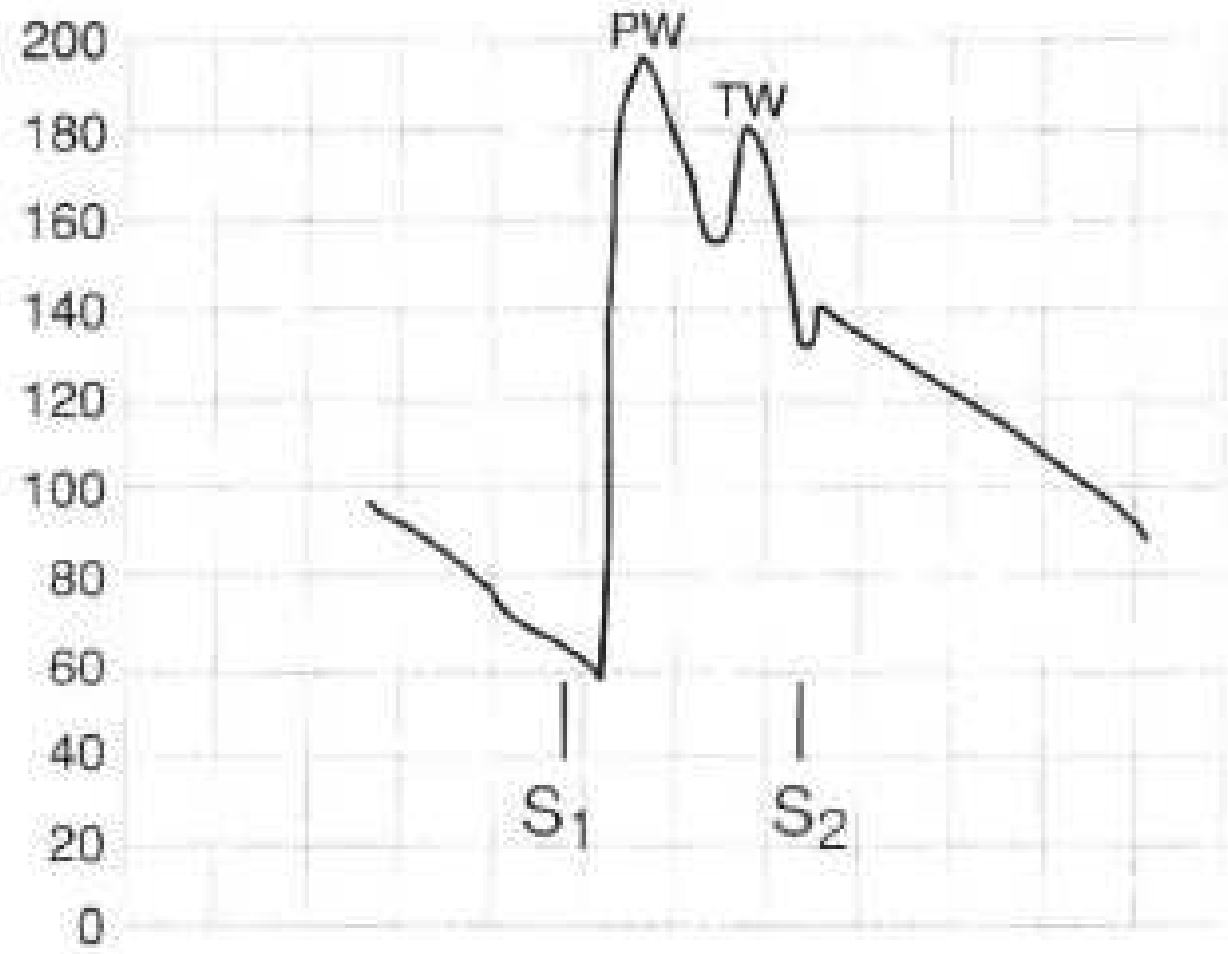

Fig. 3. A bifid pulse in combined aortic regurgitation and stenosis. Note the increased pulse pressure resulting from the lower diastolic blood pressure combined with the increased systolic pressure (blood pressure 200/60 mm Hg). The first peak is the percussion wave (PW) and the second peak is the tidal wave (TW). Note that no specific features of this pulse are related to the severity of the aortic regurgitation. S_1, first heart sound; S_2, second heart sound.

Mayo Clnic, 2nd ed

Bisferians pulse
Hypertrophic
Cardiomyopathy

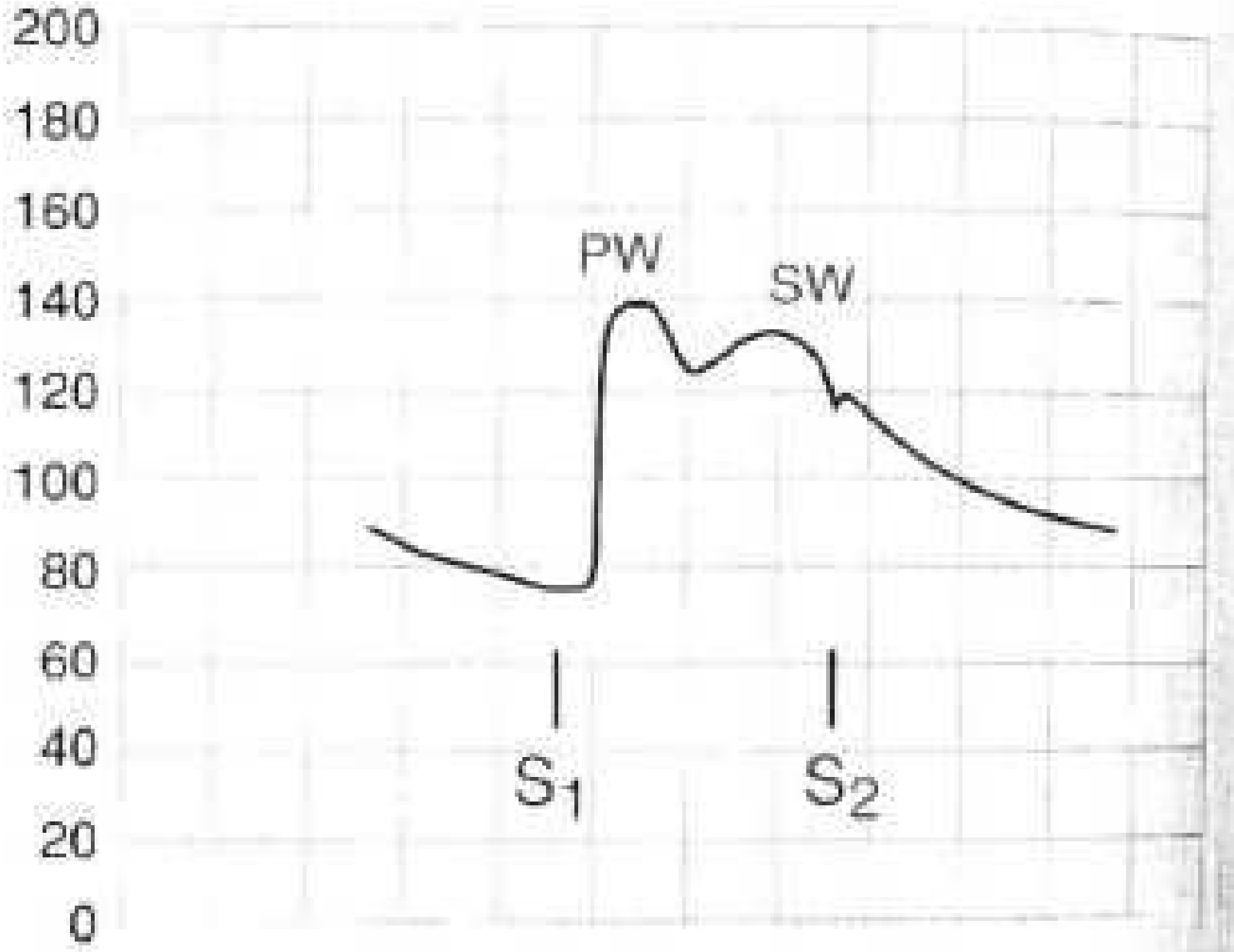

Fig. 4. Spike-and-dome pattern of hypertrophic obstructive cardiomyopathy. After the initial percussion wave (PW), a late systolic sustained secondary wave (SW) can easily be palpated because the two pulsations are frequently distinct. This may also be referred to as a "bisferiens pulse." Note, however, that not all patients with hypertrophic obstructive cardiomyopathy have such a pulse. S_1, first heart sound; S_2, second heart sound.

Mayo Clnic, 2nd ed

Dicrotic
pulse
LV
dysfunction

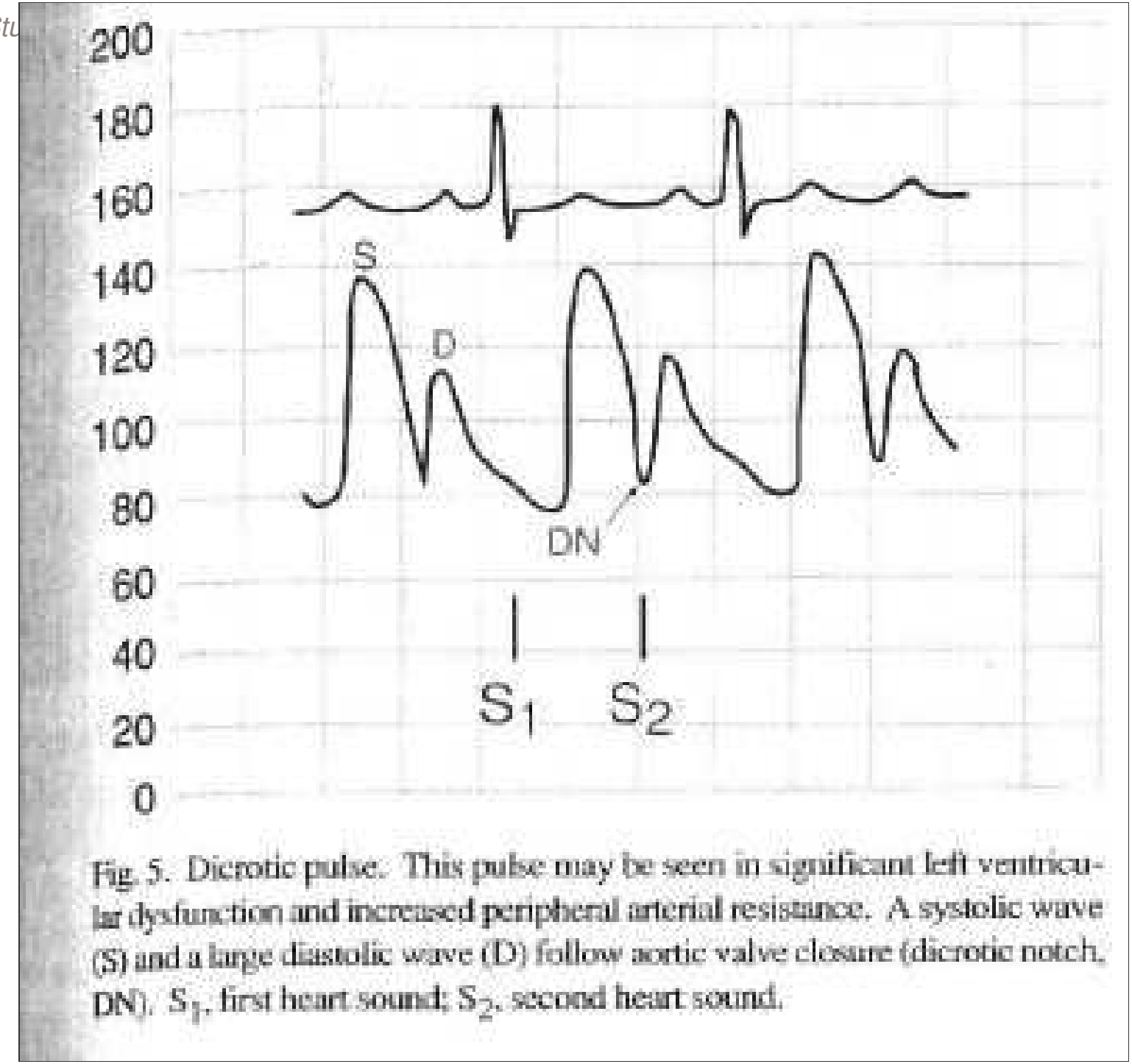

Fig. 5. Dicrotic pulse. This pulse may be seen in significant left ventricular dysfunction and increased peripheral arterial resistance. A systolic wave (S) and a large diastolic wave (D) follow aortic valve closure (dicrotic notch, DN). S_1, first heart sound; S_2, second heart sound.

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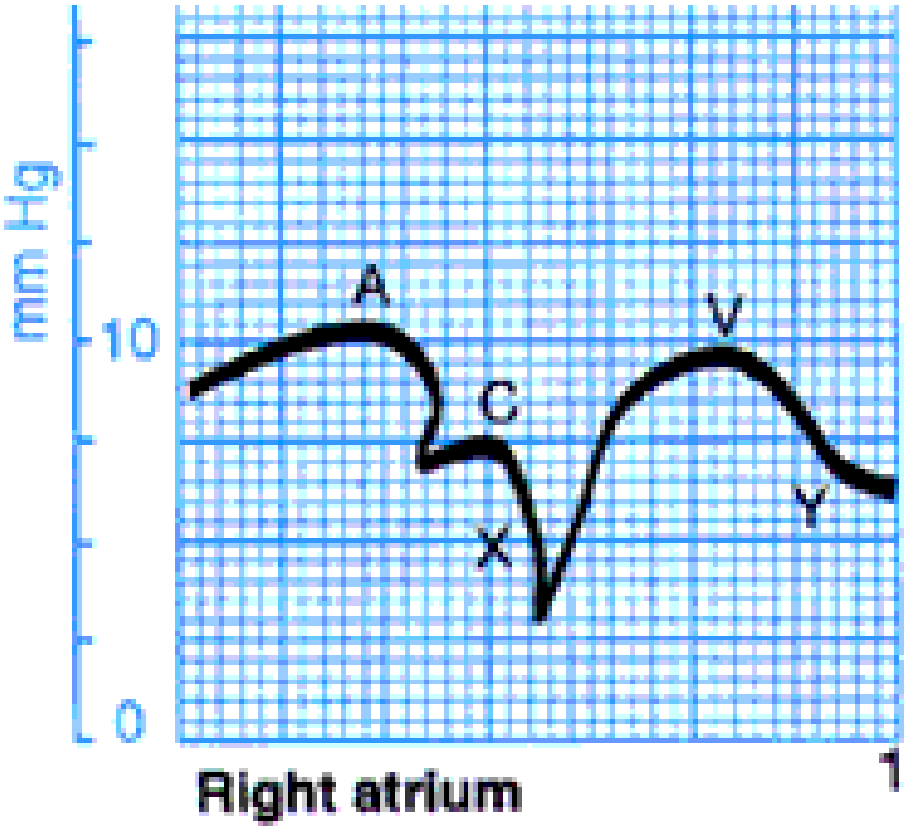
Right Atrial Waveform



a wave - RA contraction
elevated in RV failure
c wave - tricuspid closure
v wave - passive filling of RA during ventricular systole = T wave on ECG
elevated in tricuspid regurgitation
x descent - atrial diastole
y descent - atrial emptying

http://www.staff.vu.edu.au/CriticalCare/Critical%20Care/lecture2_notes.htm

Venous Pulses

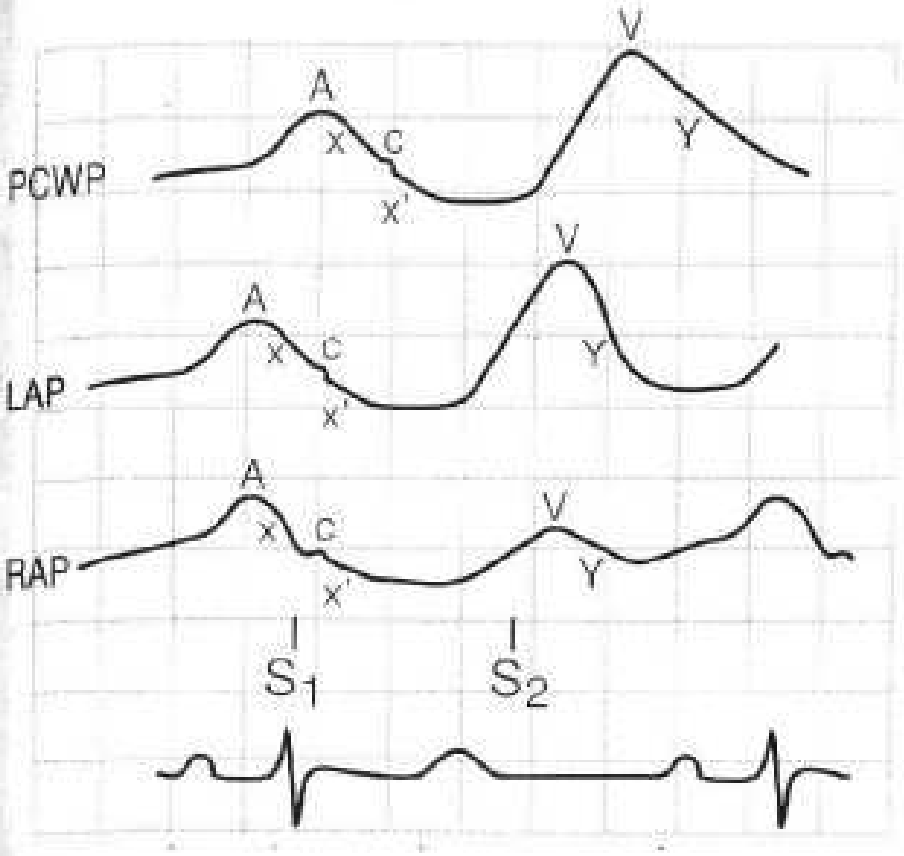


Fig. 6. Top to bottom, tracings of normal pulmonary capillary wedge pressure (PCWP), left atrial pressure (LAP), and right atrial pressure (RAP). The A wave is due to atrial contraction and the downward X descent, to atrial relaxation. The brief outward C wave is caused by a cephalad motion of the closing atrioventricular valve. The downward X' descent is a continuation of the original X descent after atrioventricular valve closure. The V wave occurs with passive atrial filling. The Y descent denotes atrial emptying into the ventricle after opening of the atrioventricular valve. This figure shows important differences in the three pressure tracings. The A wave is the first upward deflection on the hemodynamic tracing after the start of the P wave in the ECG and is within the PR segment of the ECG tracing for the right and left atrial pressure tracings. Because of the reflection of the left atrial pressure across the pulmonary vasculature, there is a time delay in PCWP tracings. Therefore, the A wave of PCWP is toward the end of the PR segment, as shown here. Similarly, the V wave is located in the TP segment of the ECG for the LAP and RAP tracings. In PCWP, the V wave may be in the latter half of the TP segment, occasionally extending into the PR segment. Another difference between the right- and left-sided pressures is that the A wave is the dominant wave of the LAP tracing, whereas the V wave is the larger of the two upward waves in the LAP and PCWP tracings. Finally, the major difference between the LAP and PCWP is the rapidity of the Y descent. The LAP tracing usually has a distinct sharp Y descent, whereas that of the PCWP tracing is more gentle. This difference can be important clinically when estimating the pressure gradient across the mitral valve (mitral stenosis), in which the use of PCWP as a surrogate for LAP may tend to overestimate the pressure gradient. S_1, first heart sound; S_2, second heart sound.

Mayo Clnic, 2nd ed

Giant A wave
During systole

RA
Tricuspid Stenosis
RV noncompliance
RV Failure
Pulmonary Stenosis
Pulmonary HTN

LA
Mitral Stenosis
LV noncompliance

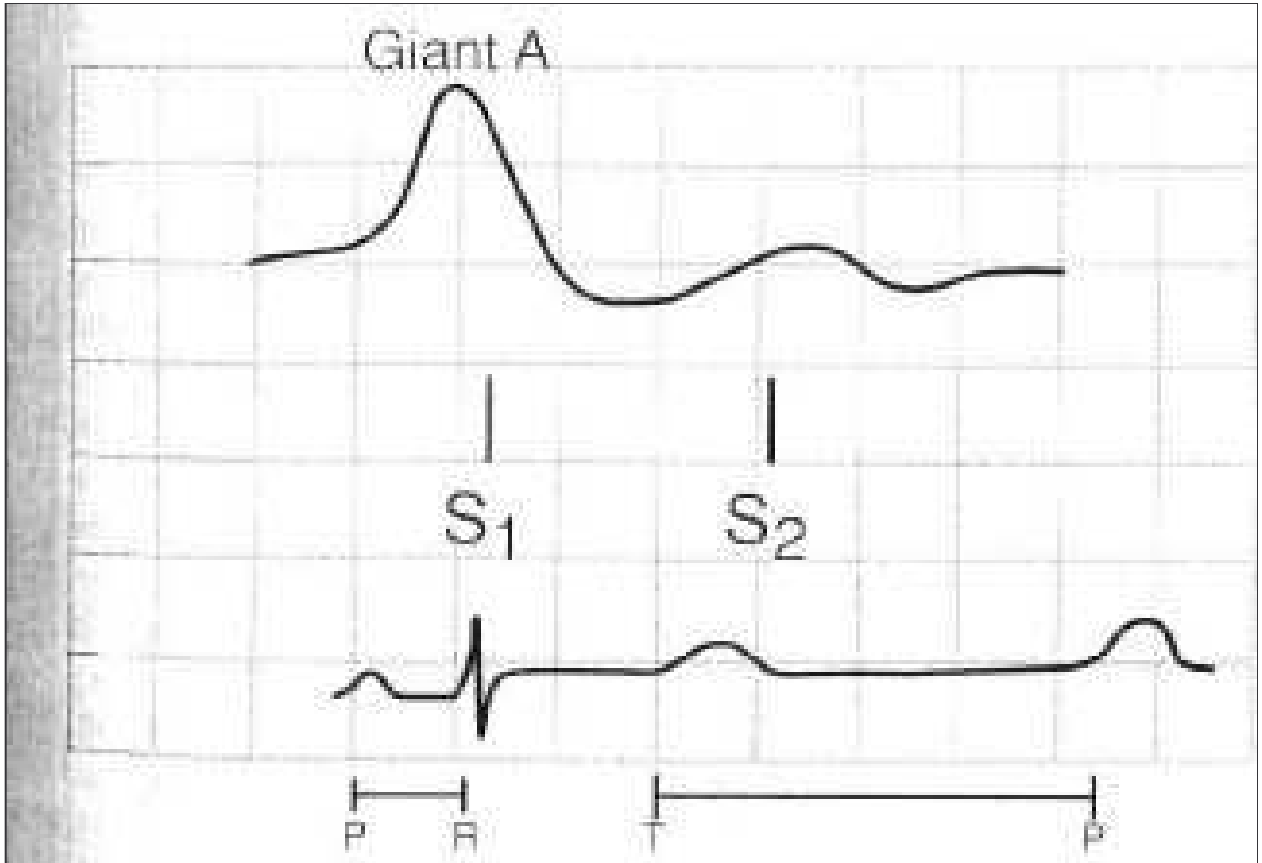


Fig. 7. Right atrial pressure tracing showing a giant A wave. This is usually associated with a poorly compliant ventricle. Note: the *giant* A wave is different from the *cannon* A wave, which refers to the pressure generated when the right atrium contracts against a closed tricuspid valve (not shown here) and is usually seen in patients with complete heart block. Cannon A waves are an intermittent phenomenon, whereas giant A waves are seen with every sinus beat. S_1, first heart sound; S_2, second heart sound.

Mayo Clnic, 2nd ed

Elevated V wave
During diastole

RA
Tricuspid
Regurgitation
RV failure
Restrictive
cardiomyopathy

LA
Mitral
Regurgitation
LV failure
VSD

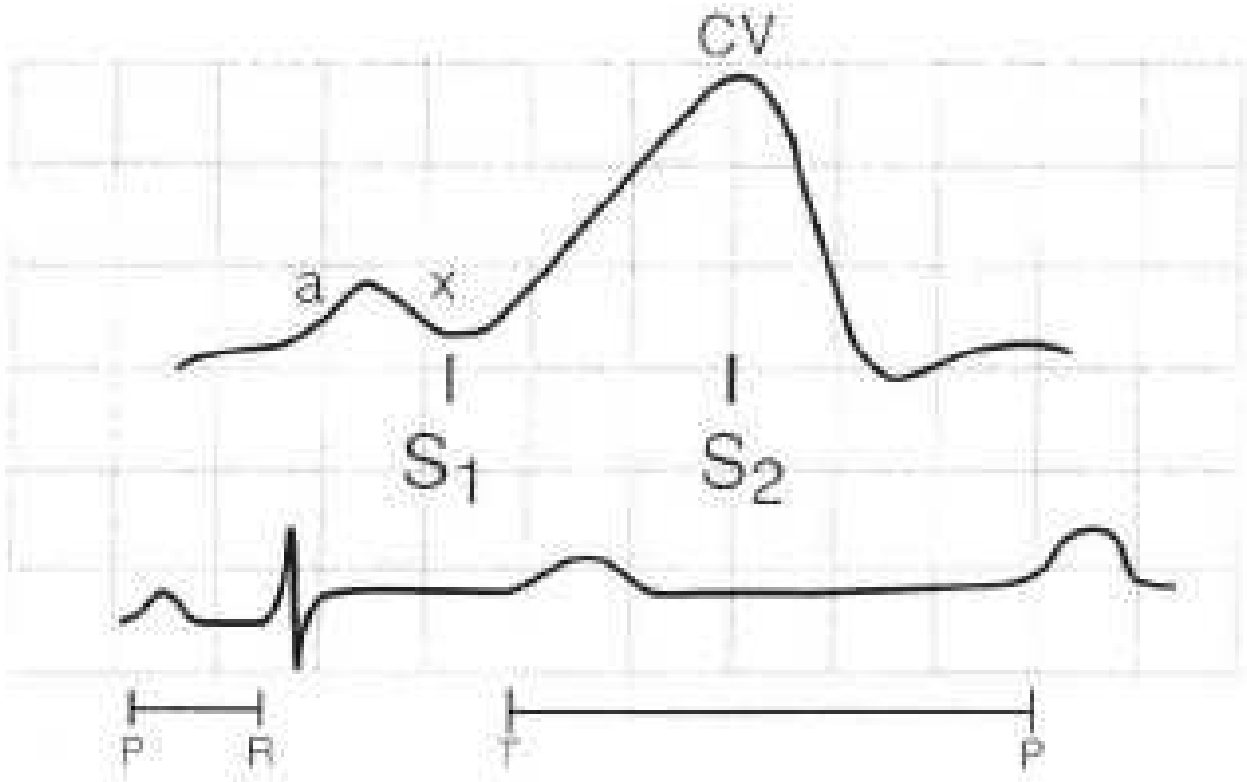


Fig. 8. A CV wave in a pulmonary capillary wedge pressure tracing. This is frequently seen in patients with significant mitral regurgitation. Note that although the A wave is characteristically delayed (end of the PR segment) for a pulmonary capillary wedge pressure tracing, the upward deflection of the CV wave is much earlier (beginning or slightly before the TP segment) than would be expected. Because of the incompetent valve, the CV wave is not only larger, it also starts early in systole. S_1, first heart sound; S_2, second heart sound.

Mayo Clnic, 2nd ed

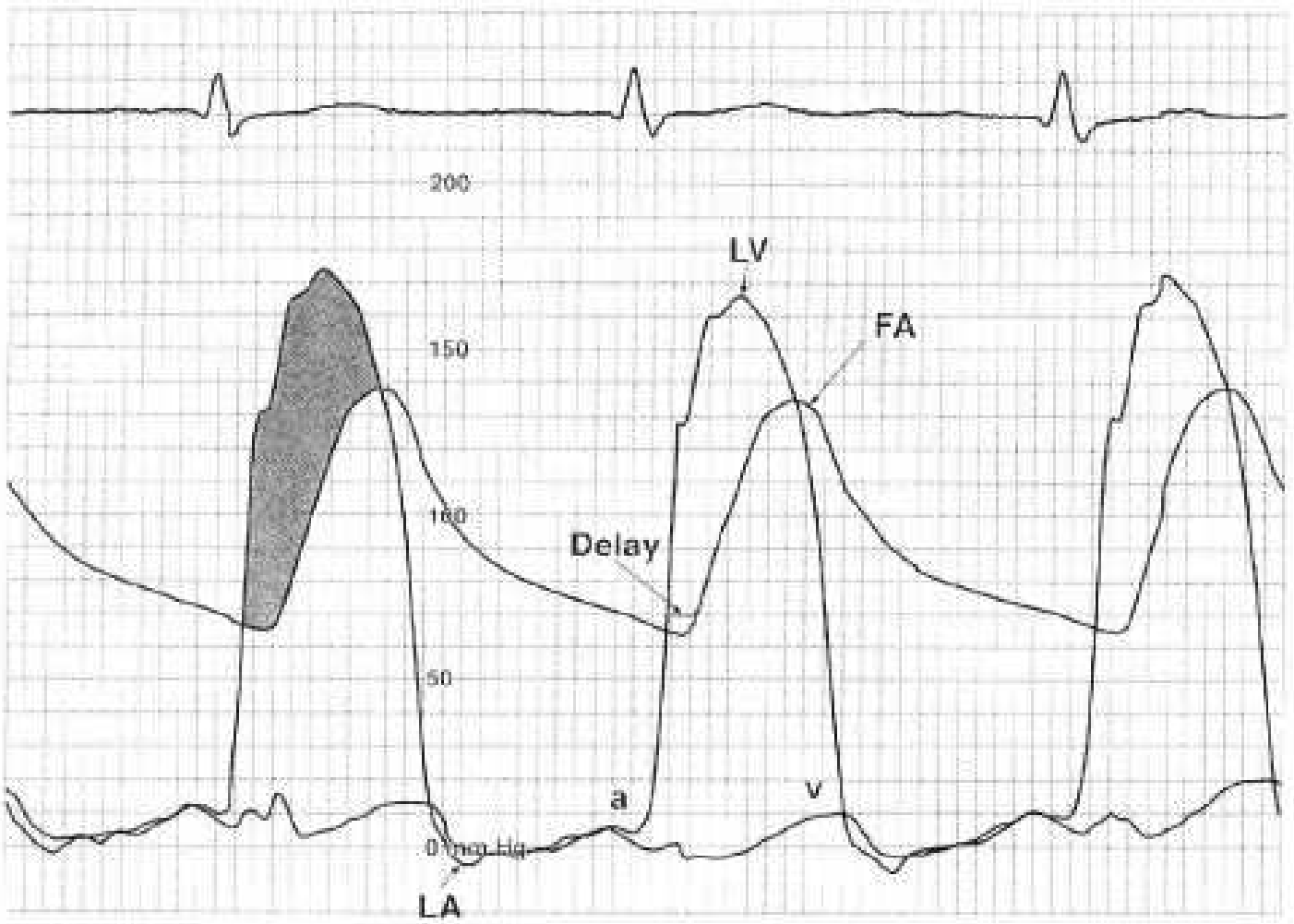


Fig. 22. Aortic stenosis. Pressure tracings from the left ventricle (LV), femoral artery (FA), and left atrium (LA). The shaded area represents the gradient between the LV and FA. Because of the transmission of pressure to a peripheral artery, there is a delay (*arrow*) in the upstroke of the FA. If this is not taken into account, the gradient across the aortic valve can be over- or underestimated.

Mayo Clnic, 2nd ed

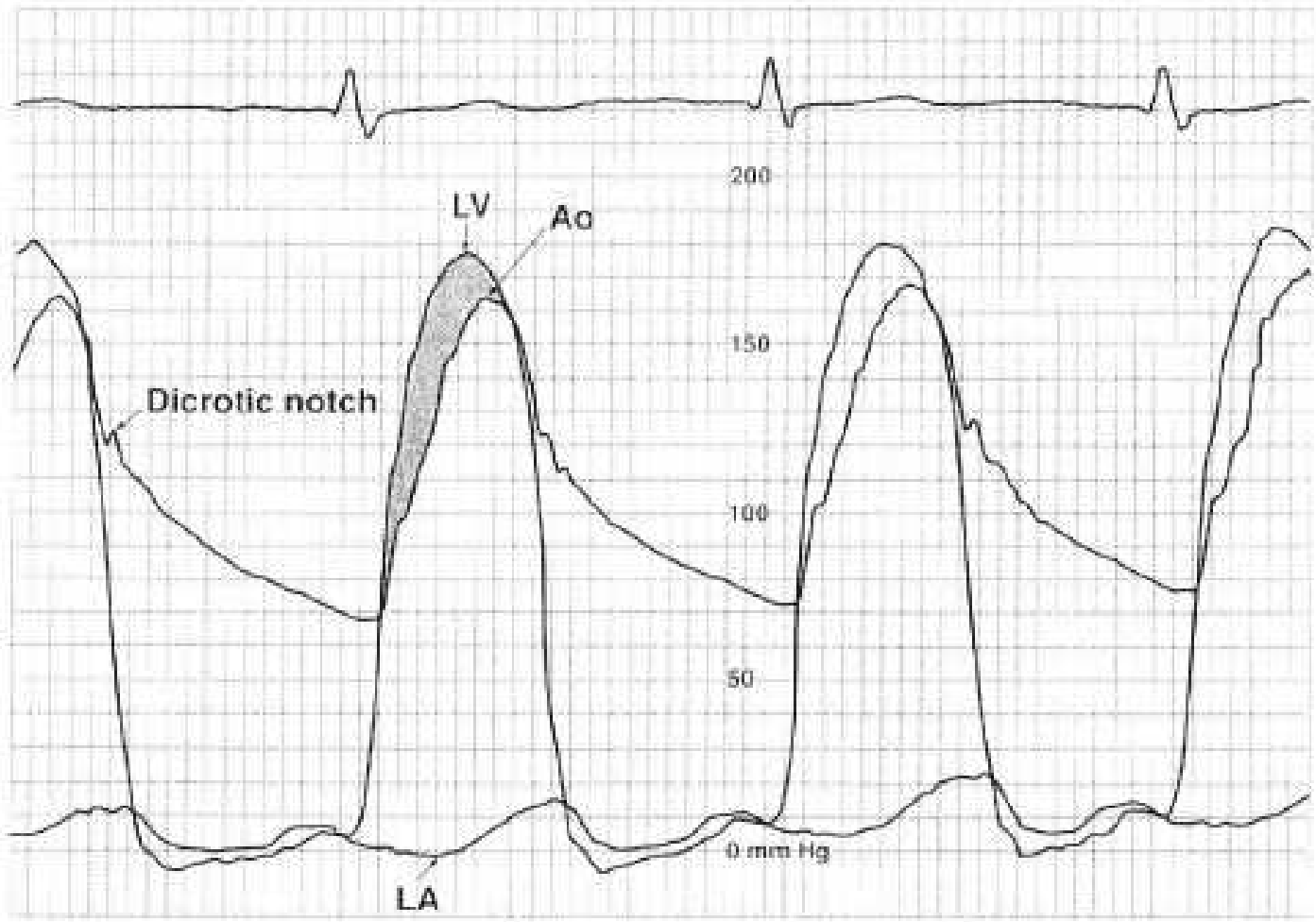


Fig. 23. Aortic stenosis. This is from the same patient as in Figure 22; however, instead of the femoral artery, aortic pressure (Ao) is used to measure the gradient (shaded area) across the aortic valve. Compare this figure with Figure 22, and note the obvious difference in measured gradients.

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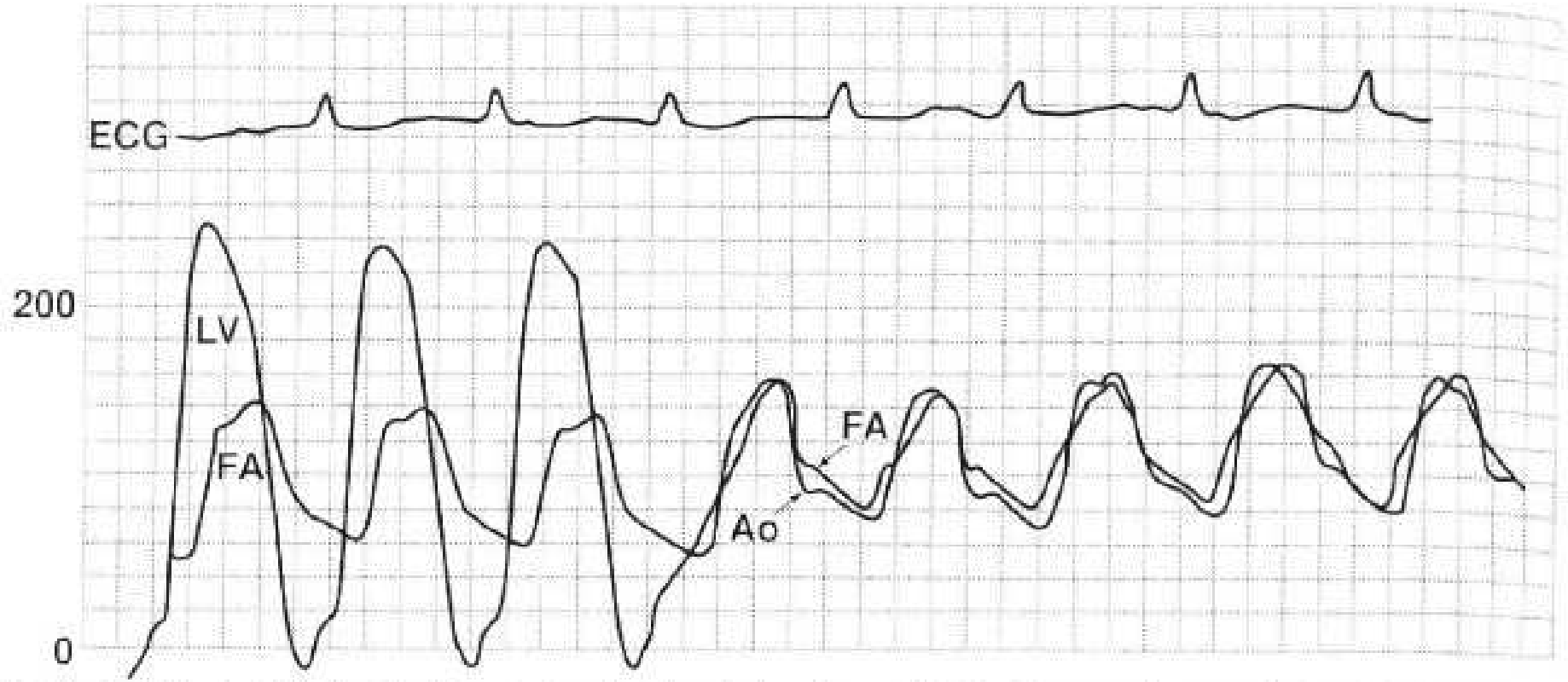

Fig. 24. Carabello sign. Pullback of the left ventricular catheter into the femoral artery (FA). The FA pressure increases when the catheter is withdrawn from across the critically stenosed aortic valve (Ao).

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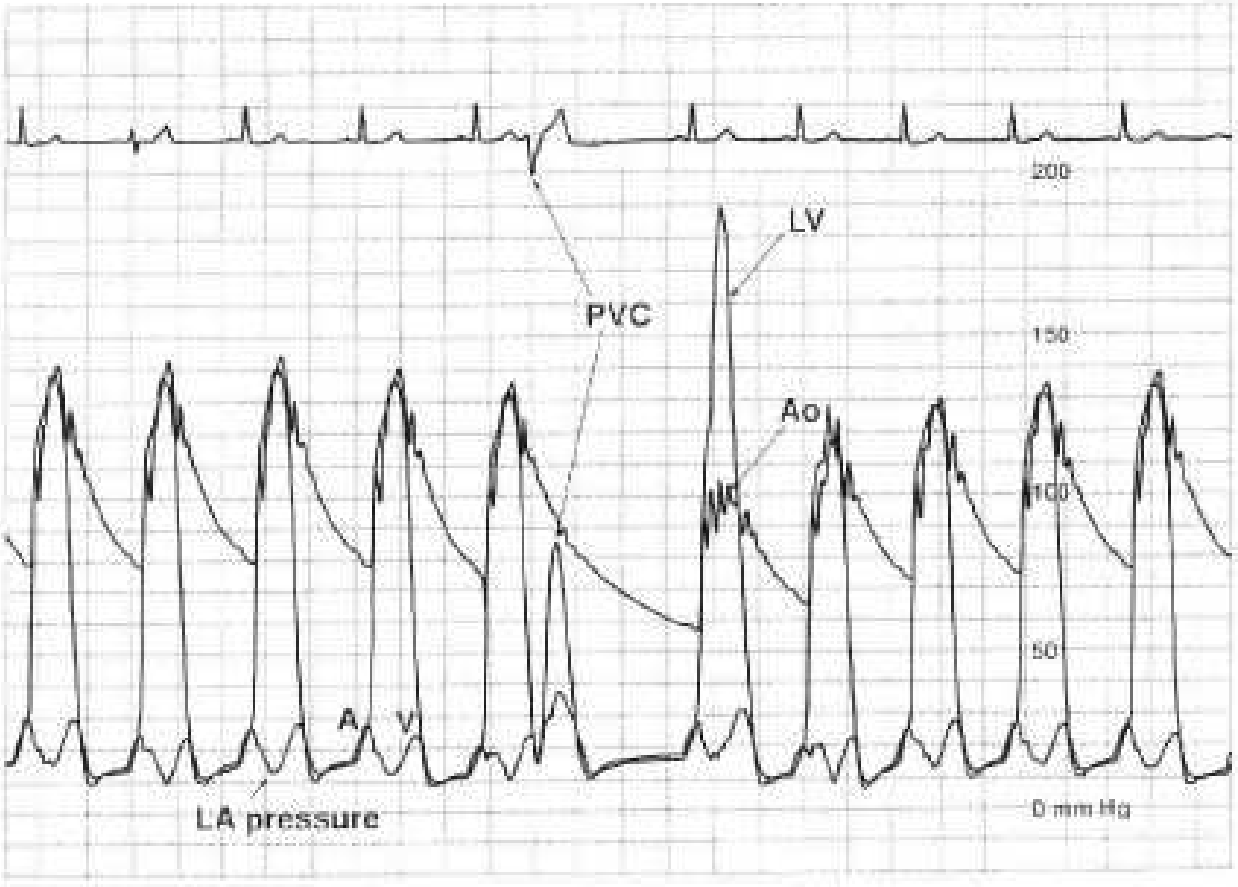

Fig. 32. Brockenbrough sign. The post-extrasystolic behavior of a gradient across the aortic/outflow tract can differentiate between a fixed and a dynamic obstruction. In a patient with hypertrophic obstructive cardiomyopathy, the post-extrasystolic beat demonstrates an increased gradient, resulting in decreased aortic pressure (Ao) even though left ventricular (LV) systolic pressure has increased significantly. This feature is characteristic of dynamic LV outflow tract obstruction and is called the "Brockenbrough sign." In fixed obstruction like aortic stenosis (in the presence of normal LV function), the gradient should not change significantly and the aortic pulse pressure should remain the same or increase slightly. This patient demonstrates an increased gradient for several beats after a premature ventricular contraction (PVC) before it returns to baseline. Also, the dynamic nature of the gradient with beat-to-beat variation should be noted. Note the decrease in the aortic pulse pressure in the post-extrasystolic beat along with the increase in the LV systolic pressure. The increase in left atrial (LA) pressure during the post-extrasystolic beat should also be appreciated. This patient had typical left ventricular outflow type hypertrophic obstructive cardiomyopathy. The Brockenbrough sign can be of immense help in occasional patients who may not have a significant gradient at baseline, especially in the slightly sedated state in the catheterization laboratory.

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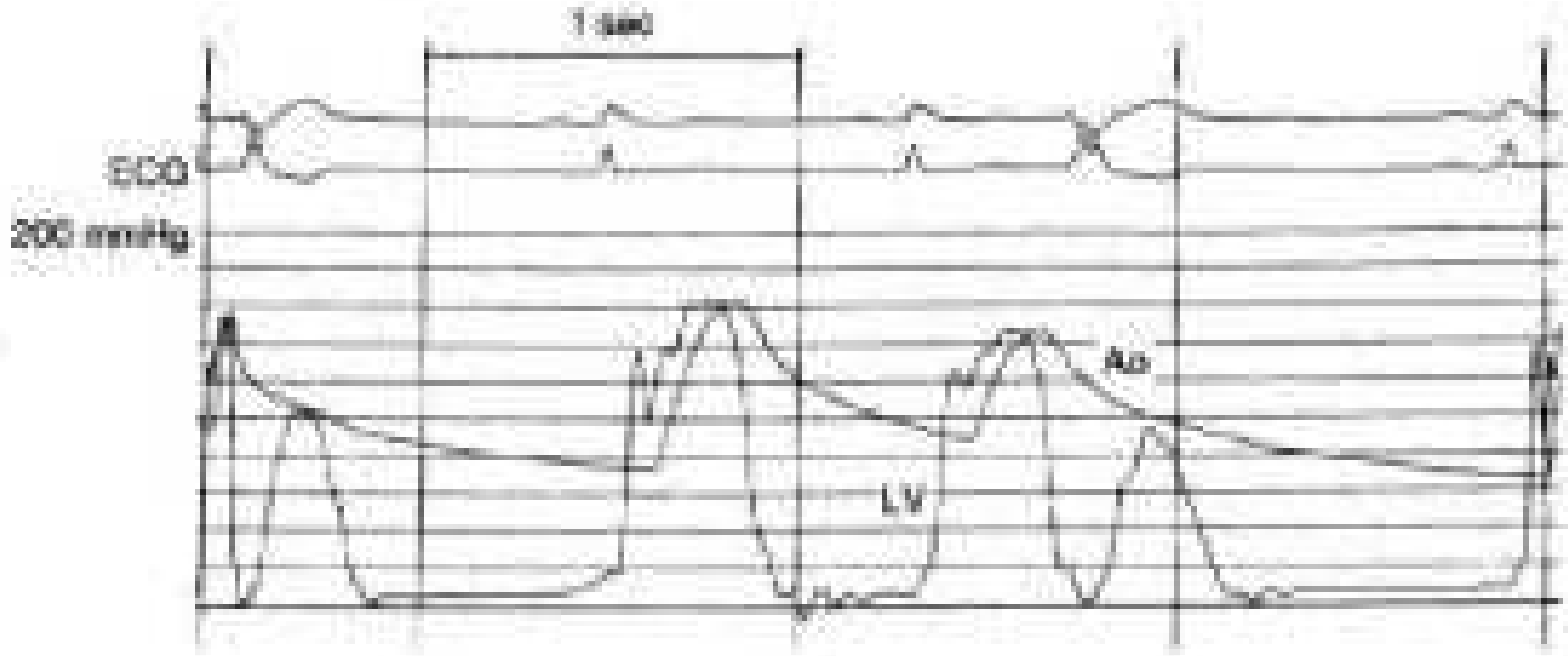


Fig. 1. Left ventricular (LV) and aortic (Ao) pressures during a premature ventricular contraction. Note the minimal change in aortic pressure and large post-PVC arterial pressure.

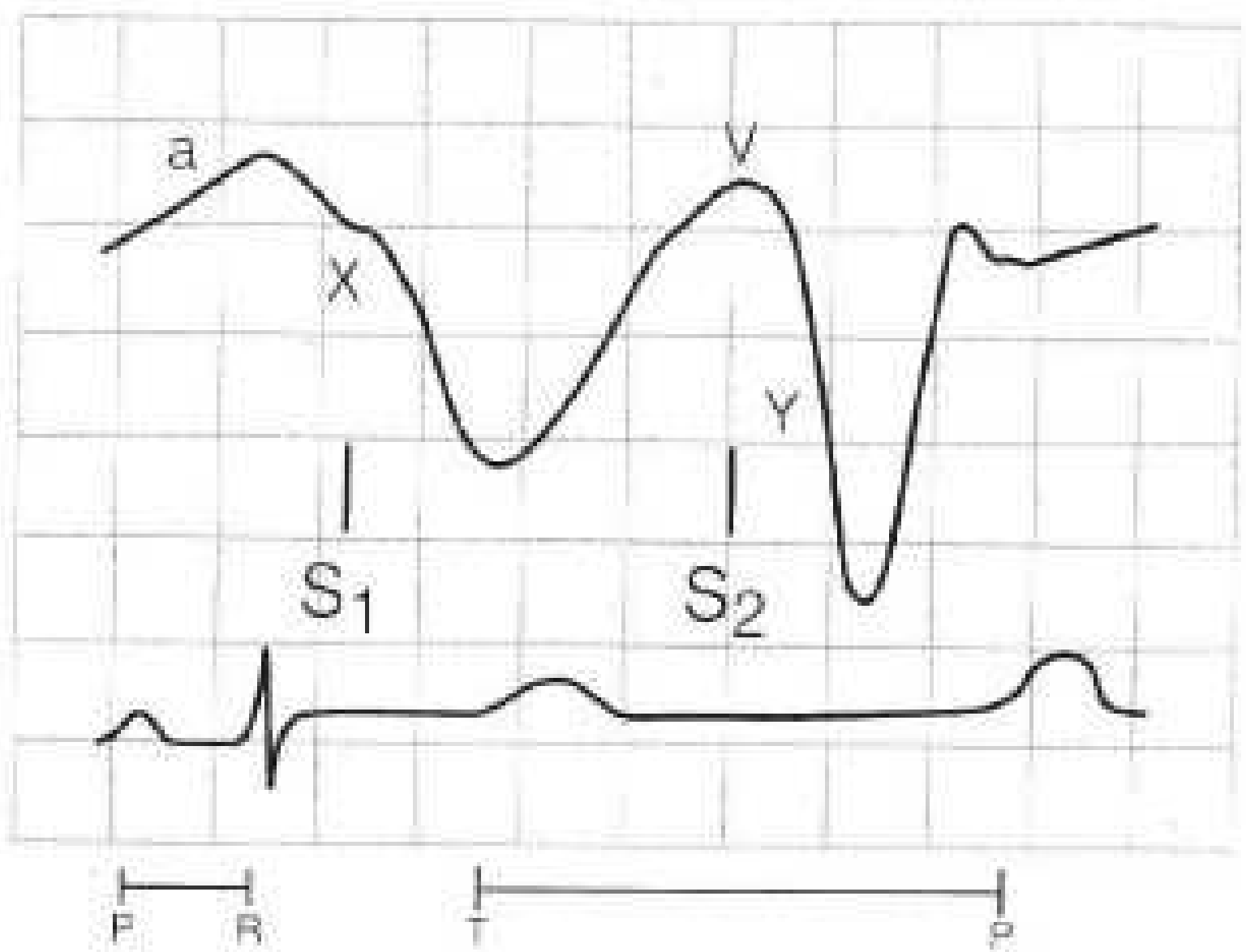


Fig. 9. Right atrial pressure tracing in constrictive pericarditis. Note the sharp X and Y descents. S_1, first heart sound; S_2, second heart sound.

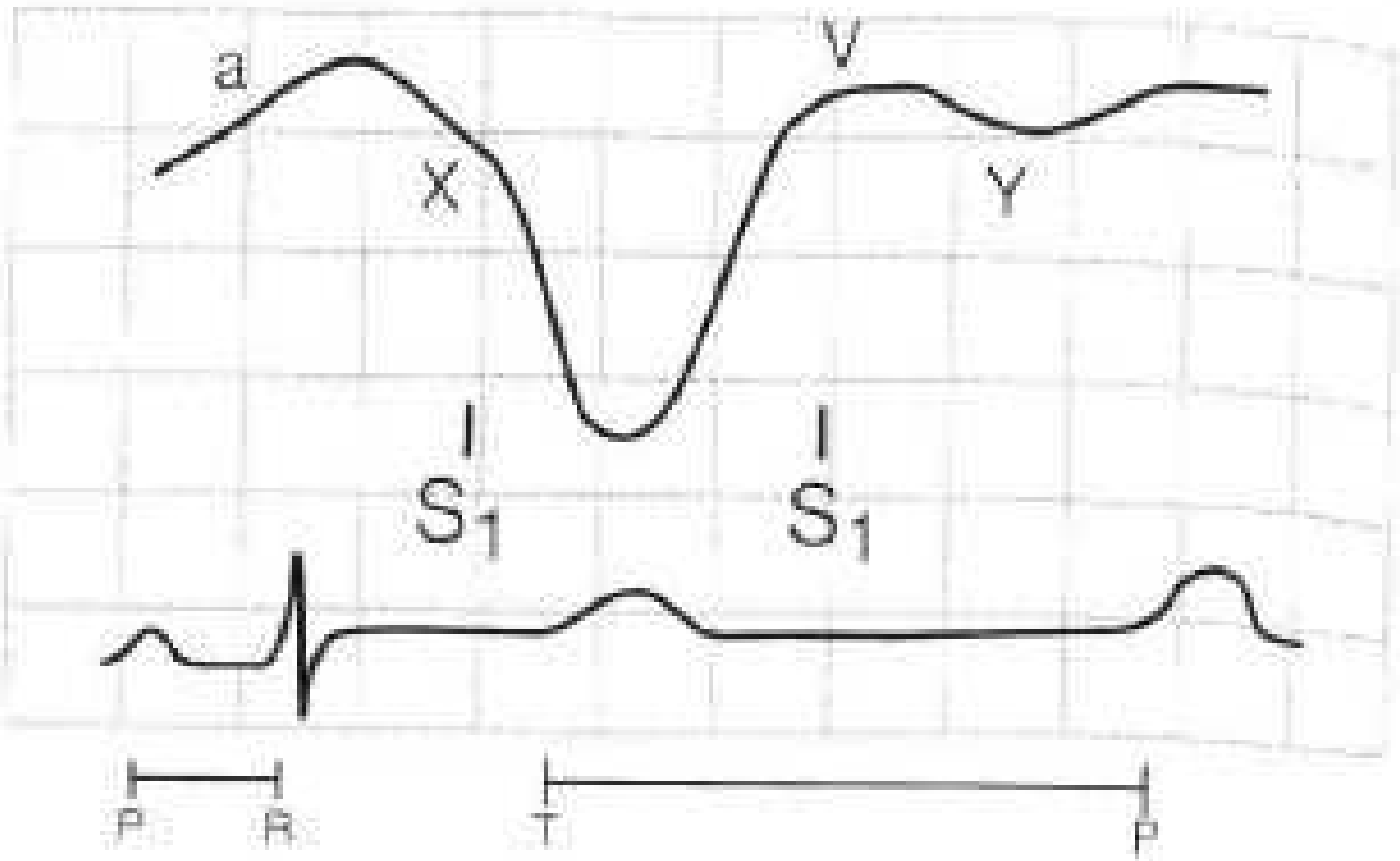


Fig. 10. Right atrial pressure tracing in pericardial tamponade. Note the sharp X descent, but a minimal or absent Y descent, consistent with minimal passive atrial emptying. S_1, first heart sound; S_2, second heart sound.

Apex Impulses in Disease

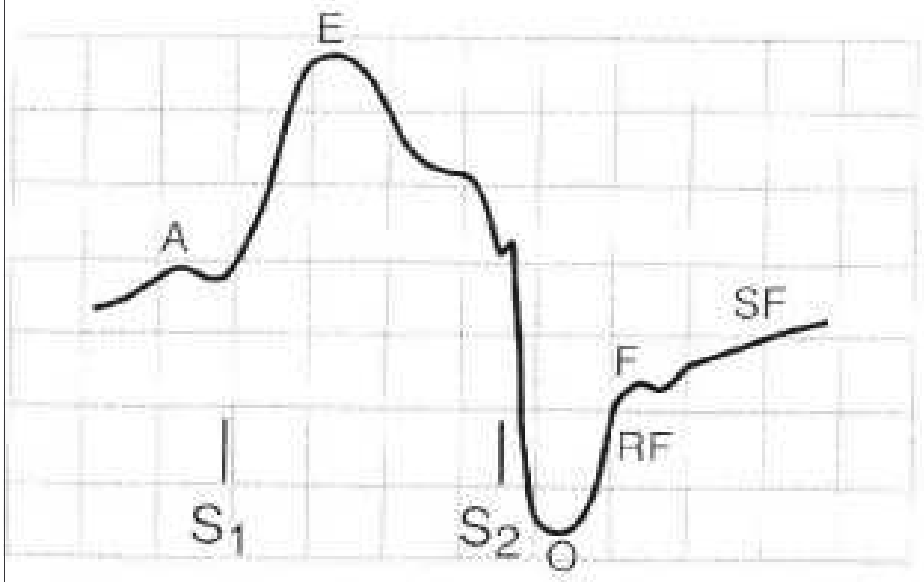


Fig. 11. Normal apex pulse (apex beat cardiogram). The normal apex pulse has several waves. The initial A wave is the outward expansion, "upward deflection on the apex cardiogram," of the apical area due to left atrial contraction. E represents maximal ejection. Note that the upward deflection has occurred entirely in the first half of systole; therefore, a normal apical impulse should be palpable only during the first half of systole. The notch in the downward slope coincides with the second heart sound (S_2) and the closure of the aortic valve, signifying the end of systole. As systole ends, the left ventricle relaxes, accelerating its retraction from the chest wall (downward slope on the apex cardiogram), which ends at O. The O point marks mitral valve opening. As the left ventricle dilates because of blood flowing through the open mitral valve, an upward deflection is noted, the rapid filling wave (RF). The F point is the peak of this rapid filling and is synchronous with the timing of the third heart sound. The slow filling wave (SF) signifies slow ventricular filling during mid-diastole, before atrial contraction. Note: in a normal heart, only the E point during the early part of systole is palpable. S_1, first heart sound.

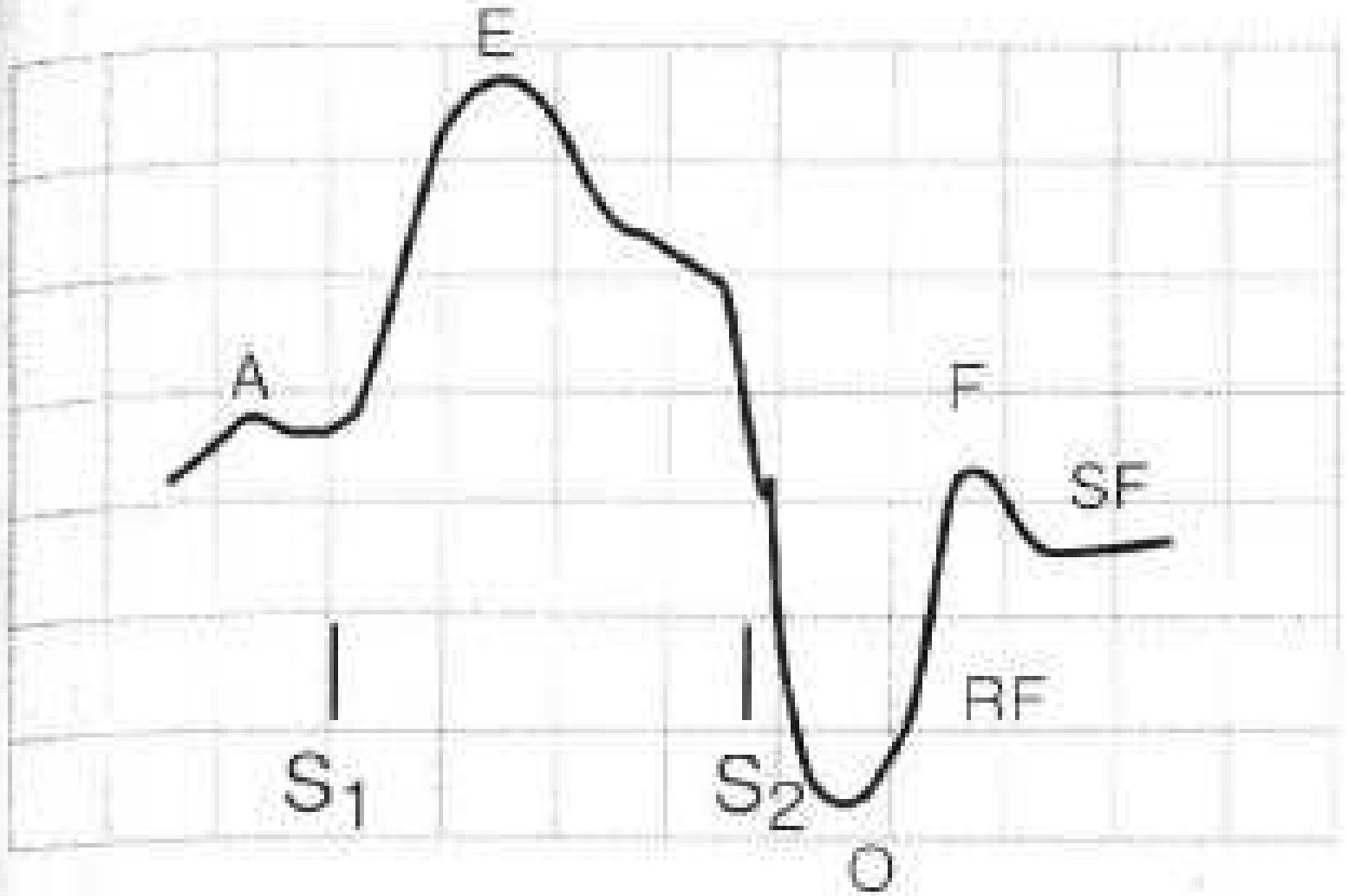


Fig. 12. In conditions in which the the rapid filling wave (RF) is steep and tall (e.g., increased filling due to severe mitral regurgitation or restrictive filling pattern), the F point is more pronounced. This may be appreciated as an audible or palpable third heart sound.

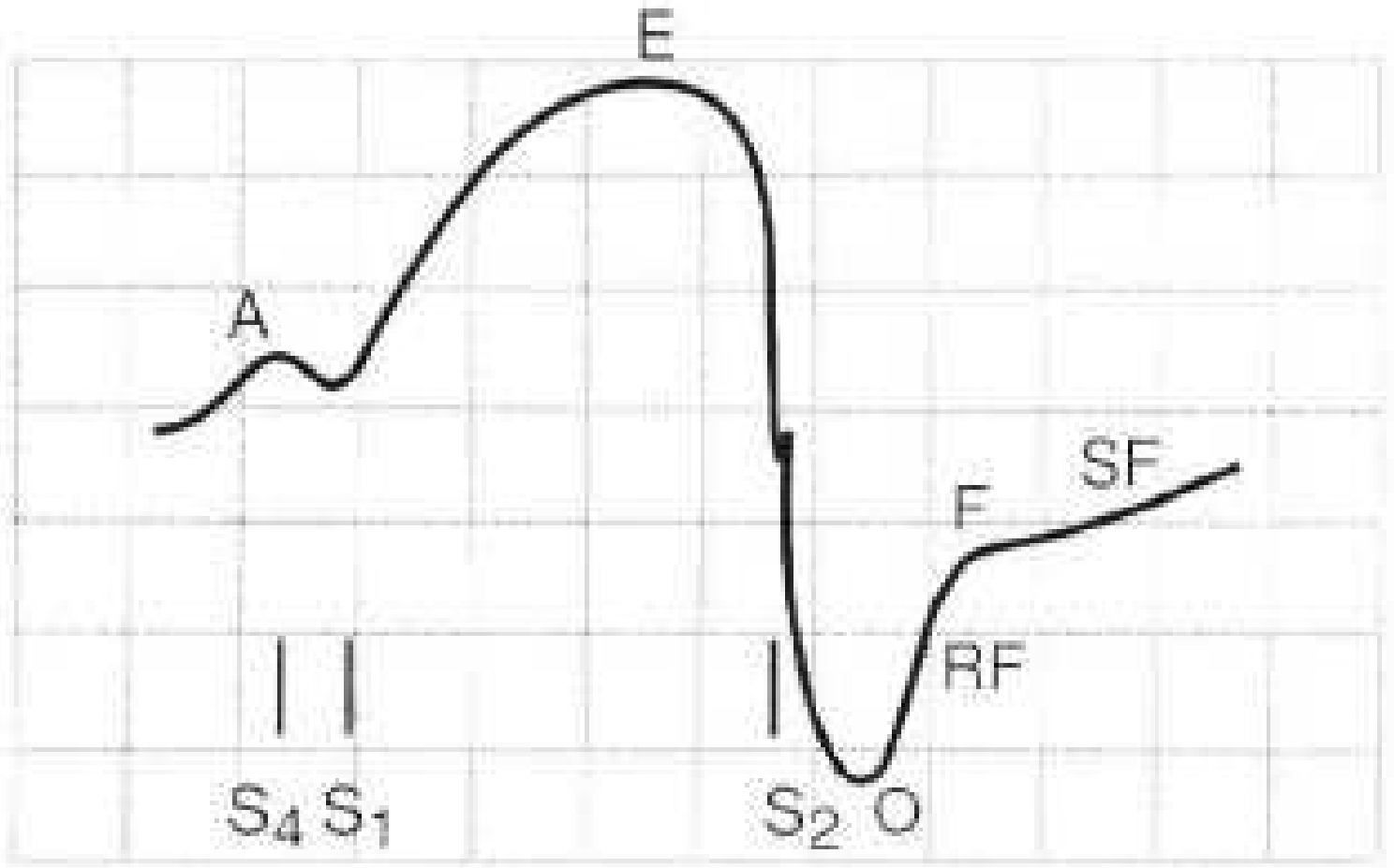


Fig. 13. In aortic stenosis with normal left ventricular function, the apex impulse is strong, prolonged, and reaches a sustained peak, E, in late systole. Contrast this with a normal apex in which peak E is reached in early systole. Also, the amplitude of the A wave may be increased, thereby making it palpable. This would coincide with the fourth heart sound (S_4). S_1, first heart sound; S_2, second heart sound.

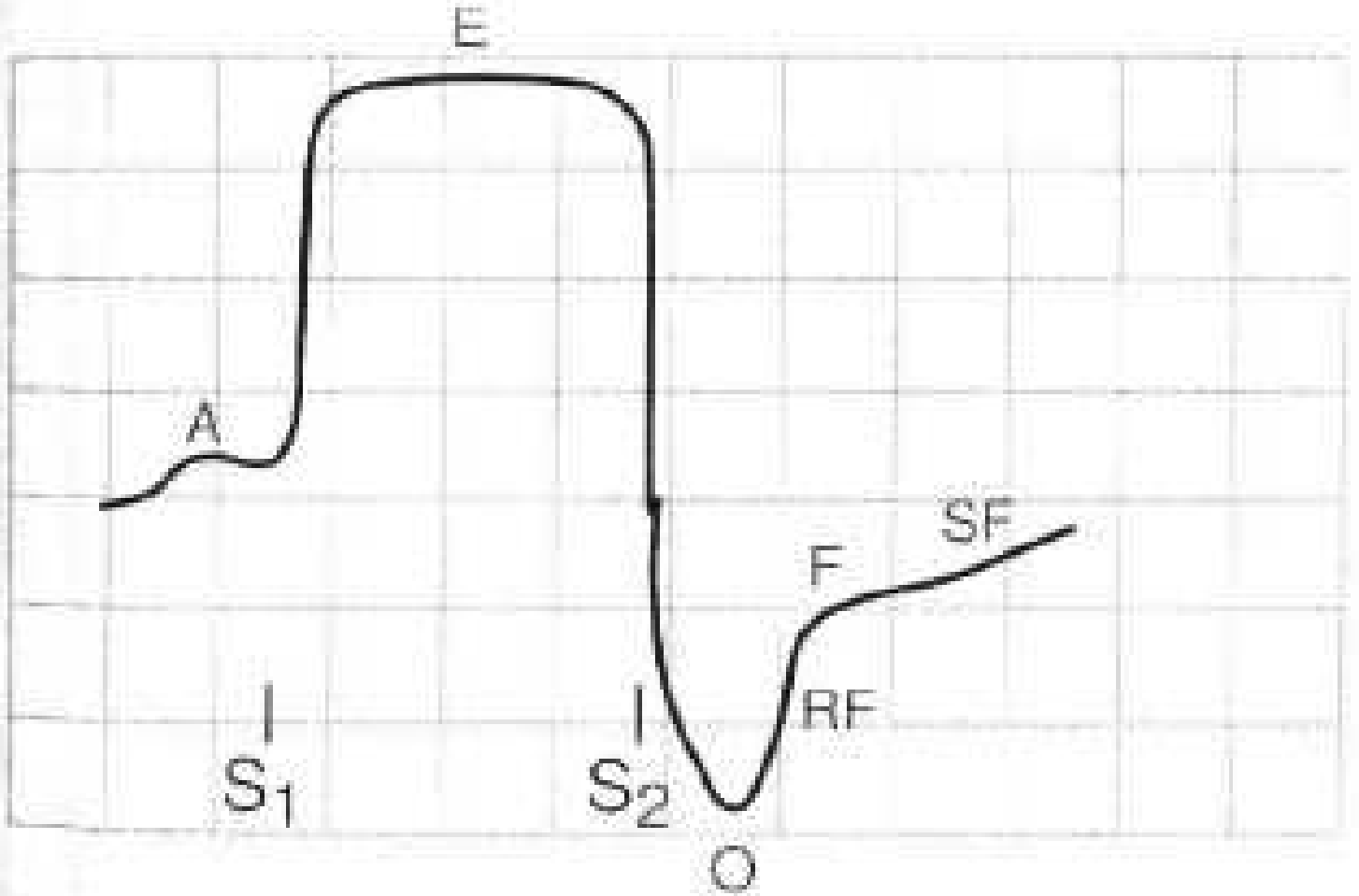


Fig. 14. There is significant dyskinesis of the apical impulse in patients with a left ventricular aneurysm. In contrast to left ventricular hypertrophy, the peak is reached early in systole and, in contrast to the normal impulse, remains sustained throughout systole. Additionally, the apical impulse extends over a wider area corresponding to the ventricular aneurysm. S_1, first heart sound; S_2, second heart sound.

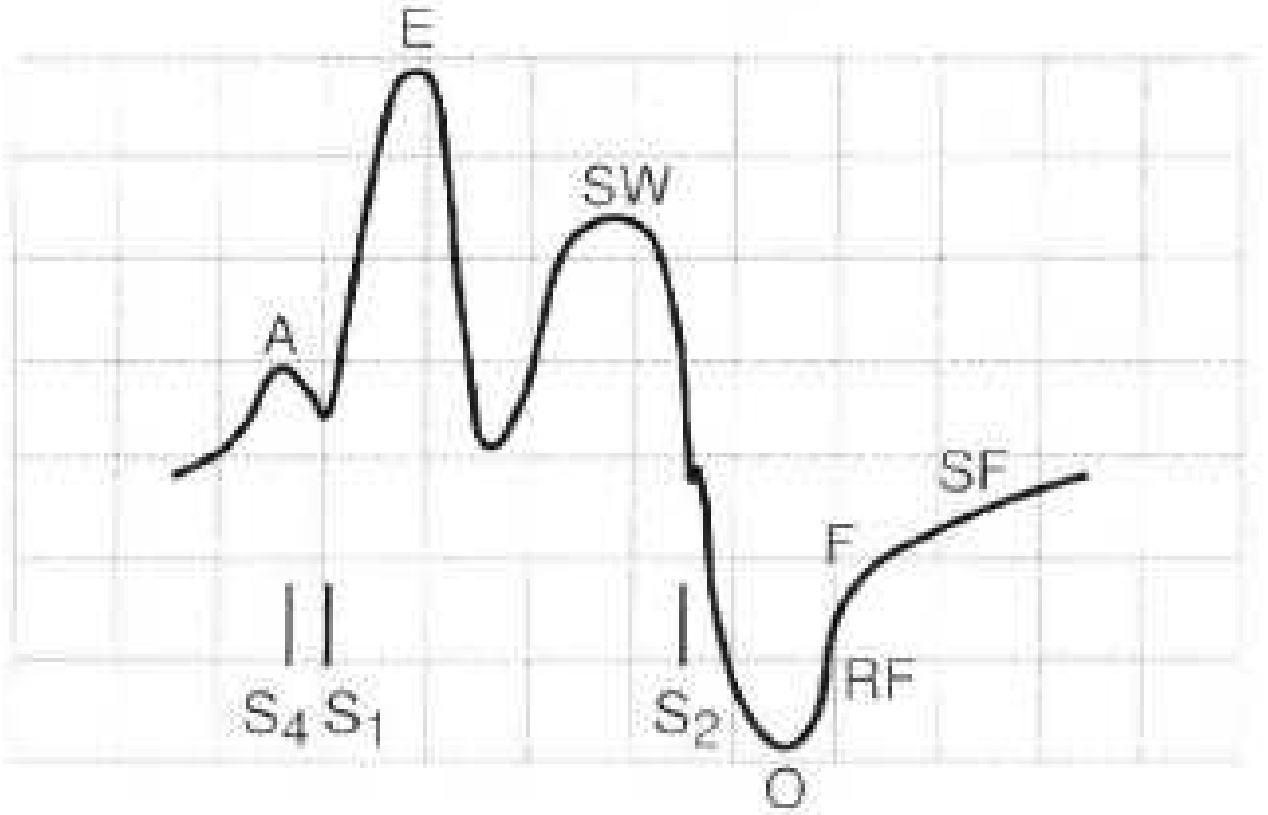


Fig. 15. The classic "triple-ripple" apical impulse of hypertrophic obstructive cardiomyopathy. There is a rapid and early rise to the E point, after which there is sudden cessation and even withdrawal of the apical impulse (corresponds to dynamic outflow obstruction, which peaks in mid-systole) until mid-systole, when a more sustained secondary wave (SW) may be palpable. Additionally, the A wave amplitude may also be increased and, thus, palpable. This corresponds to an audible fourth heart sound (S_4) from the left ventricle. These three peaks are frequently referred to as the "triple-ripple apical impulse of hypertrophic obstructive cardiomyopathy." Note, however, that this classic representation is not universally found in hypertrophic obstructive cardiomyopathy.

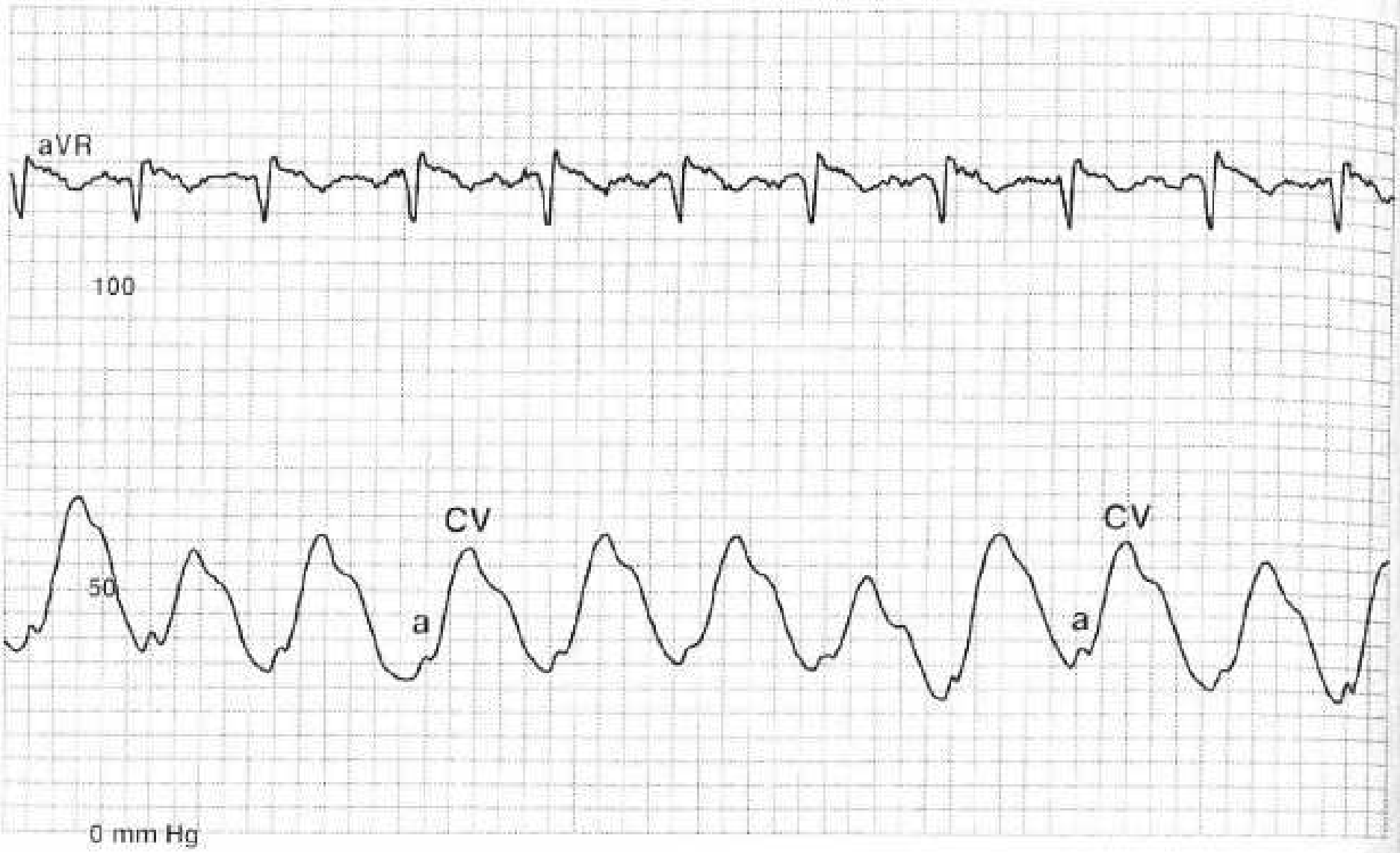


Fig. 16. Pulmonary capillary wedge pressure tracing in a patient with severe mitral regurgitation due to ruptured capillary muscle in association with acute myocardial infarction. The large CV waves measured on this tracing averaged 60 mm Hg. Note that the start of the CV wave in this tracing (before the TP segment of the ECG) is earlier than would be expected in a normal V wave.

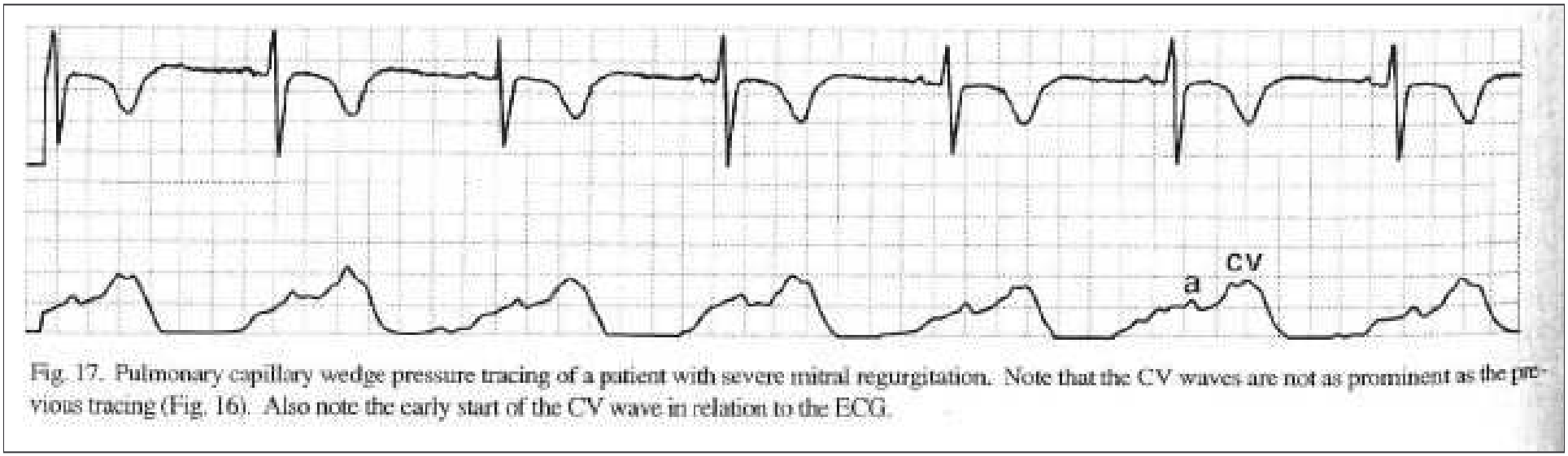


Fig. 17. Pulmonary capillary wedge pressure tracing of a patient with severe mitral regurgitation. Note that the CV waves are not as prominent as the previous tracing (Fig. 16). Also note the early start of the CV wave in relation to the ECG.

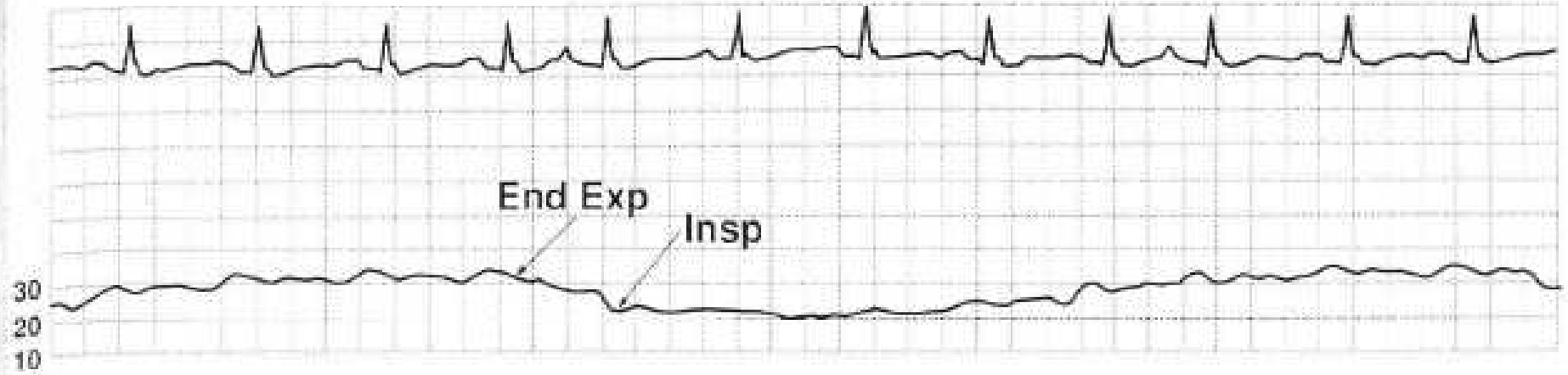


Fig. 18. Variations of pulmonary capillary wedge pressure during spontaneous breathing. The negative intrathoracic pressure generated during the inspiratory phase (Insp) of spontaneous respiration results in an artificial lowering of the pulmonary capillary wedge pressure. During the end-expiratory phase (End Exp), there is relative apnea and the pulmonary capillary wedge pressure here best approximates the left ventricular end-diastolic pressure. As shown in the figure, this point occurs just before the negative dip in the pulmonary capillary wedge pressure. During positive pressure ventilation, end expiration remains the optimal time to measure pulmonary capillary wedge pressure. However, because of positive pressure ventilation, pulmonary capillary wedge pressure is artificially increased during the inspiratory phase and end-expiration occurs just before the upward (positive) shift in the pressure. Therefore, it is important to remember respiratory variations/modes when measuring pulmonary capillary wedge pressure. Note that in most catheterization laboratories, multiple cardiac cycles across respiratory phases are averaged to obtain the mean wedge pressure.

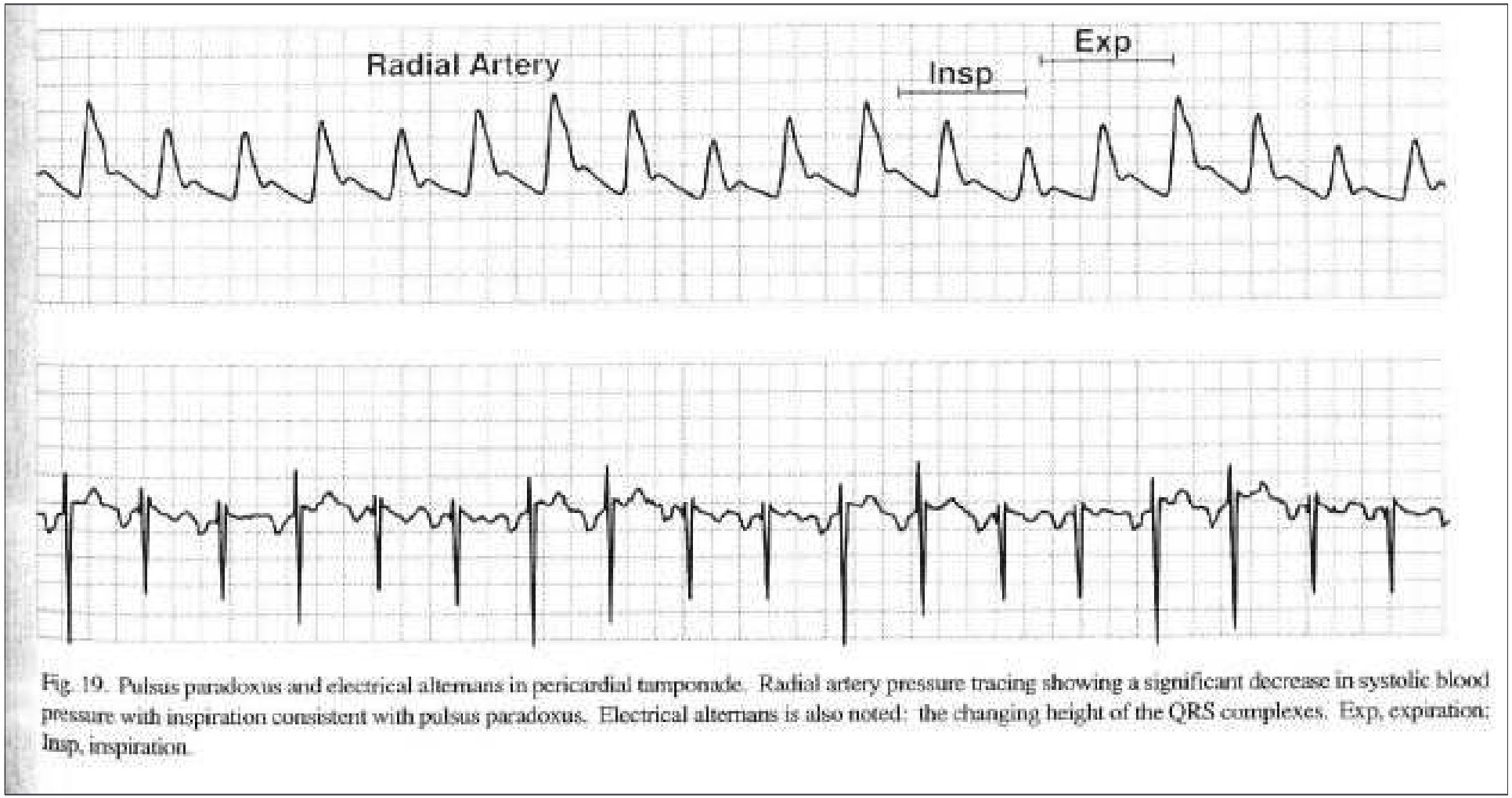


Fig. 19. Pulsus paradoxus and electrical alternans in pericardial tamponade. Radial artery pressure tracing showing a significant decrease in systolic blood pressure with inspiration consistent with pulsus paradoxus. Electrical alternans is also noted: the changing height of the QRS complexes. Exp, expiration; Insp, inspiration.

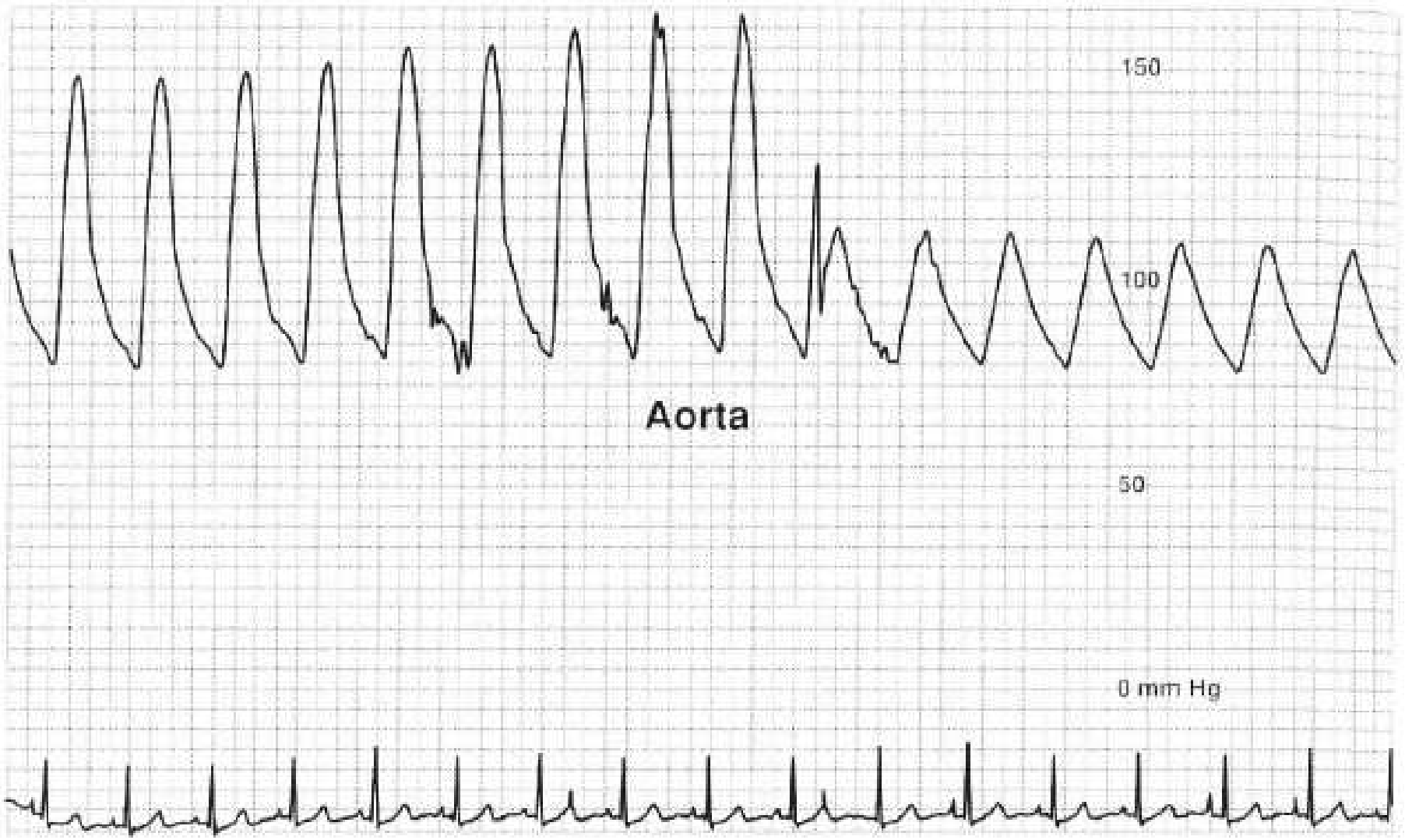


Fig. 20. Coarctation of the aorta. Pullback from the ascending aorta to below the subclavian artery, showing the pressure difference across the coarctation.

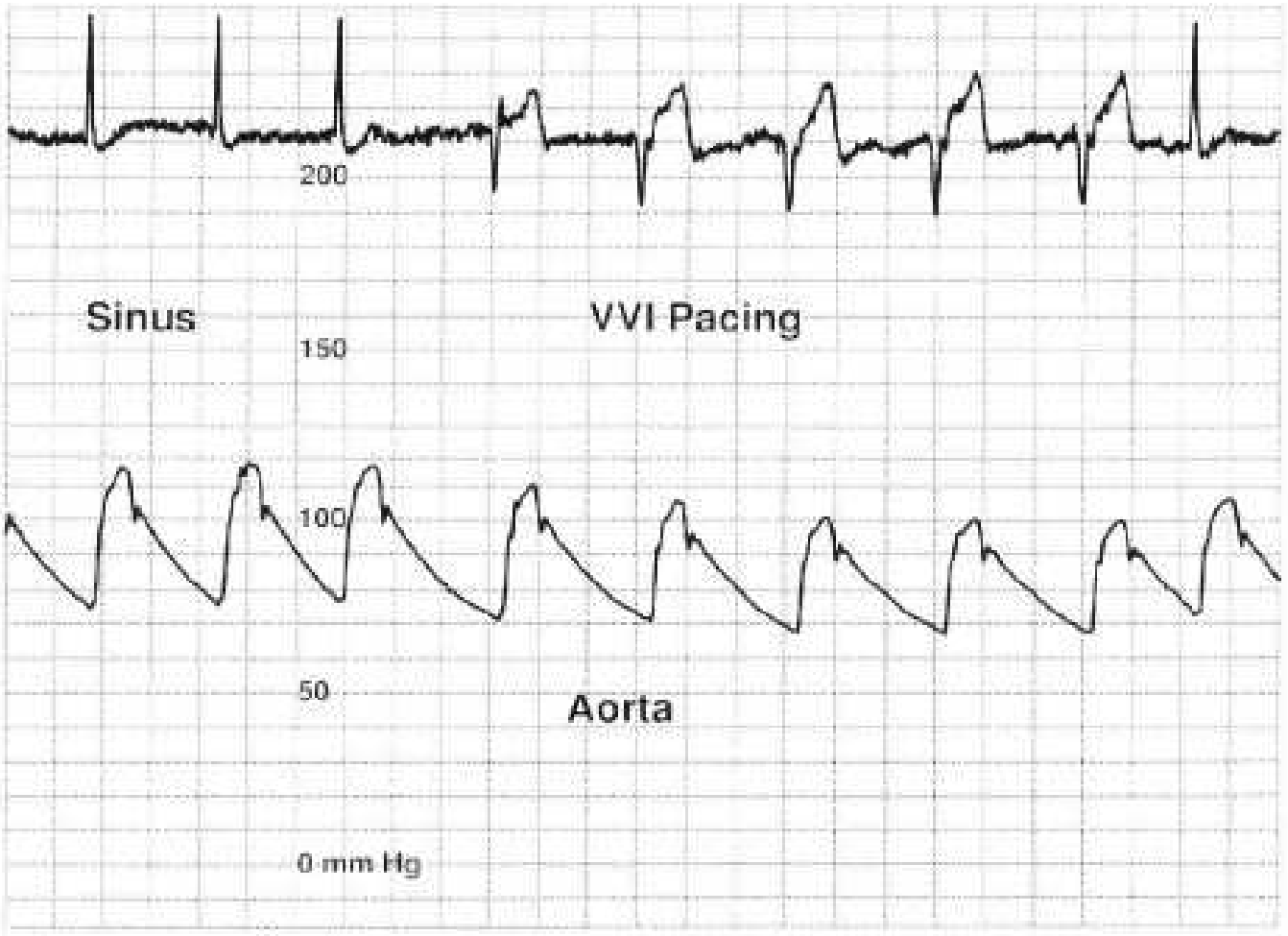


Fig. 21. Pacemaker syndrome. The first 3 beats are sinus-mediated and the next 4 are paced by a VVI permanent pacemaker. Note the decrease in systolic blood pressure while being paced.

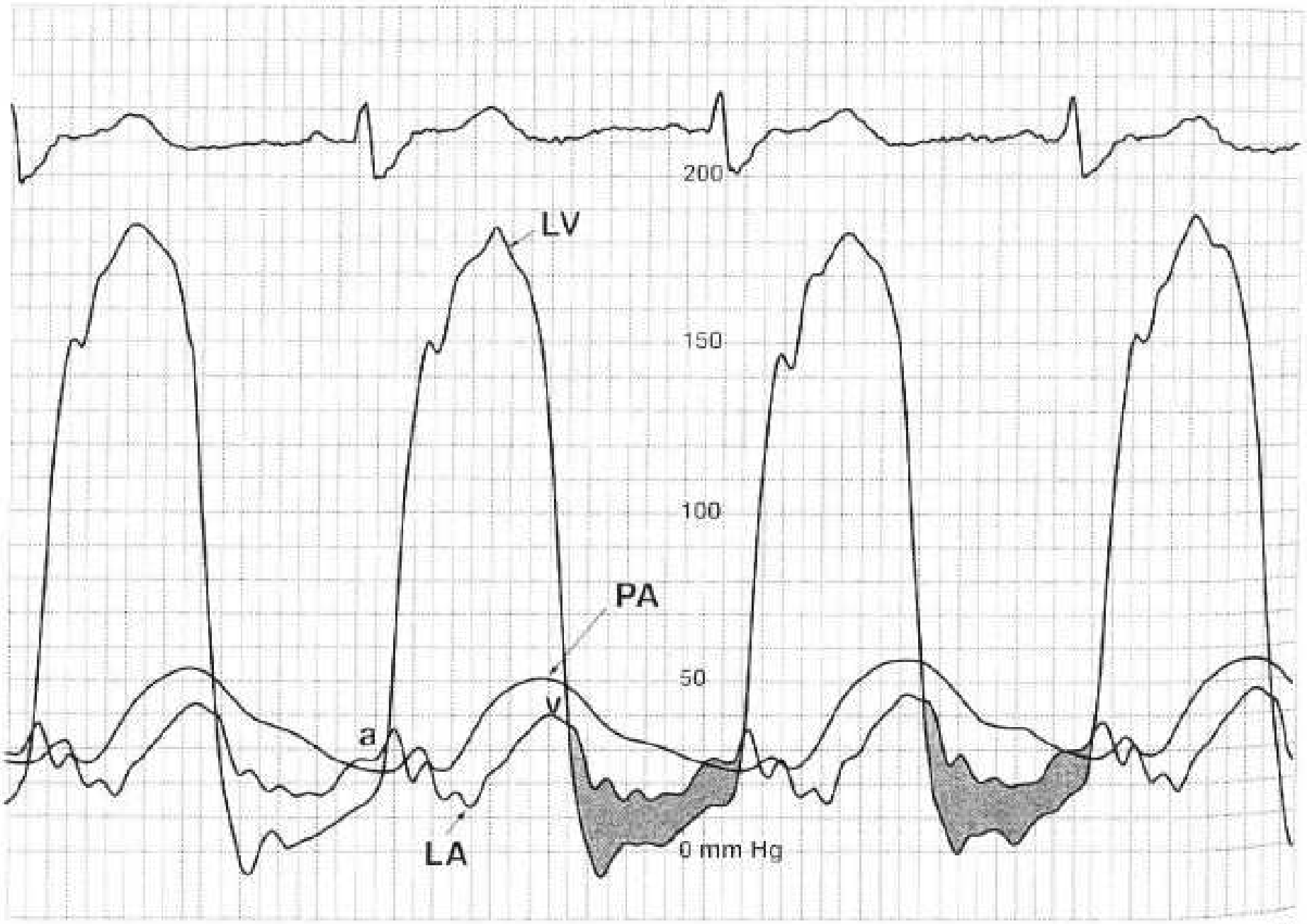


Fig. 25. Mitral stenosis. The shaded area represents the pressure gradient across the mitral valve. LV, left ventricular pressure; PA, pulmonary artery pressure; LA, left atrial pressure; a, left atrial A wave; v, left atrial V wave.

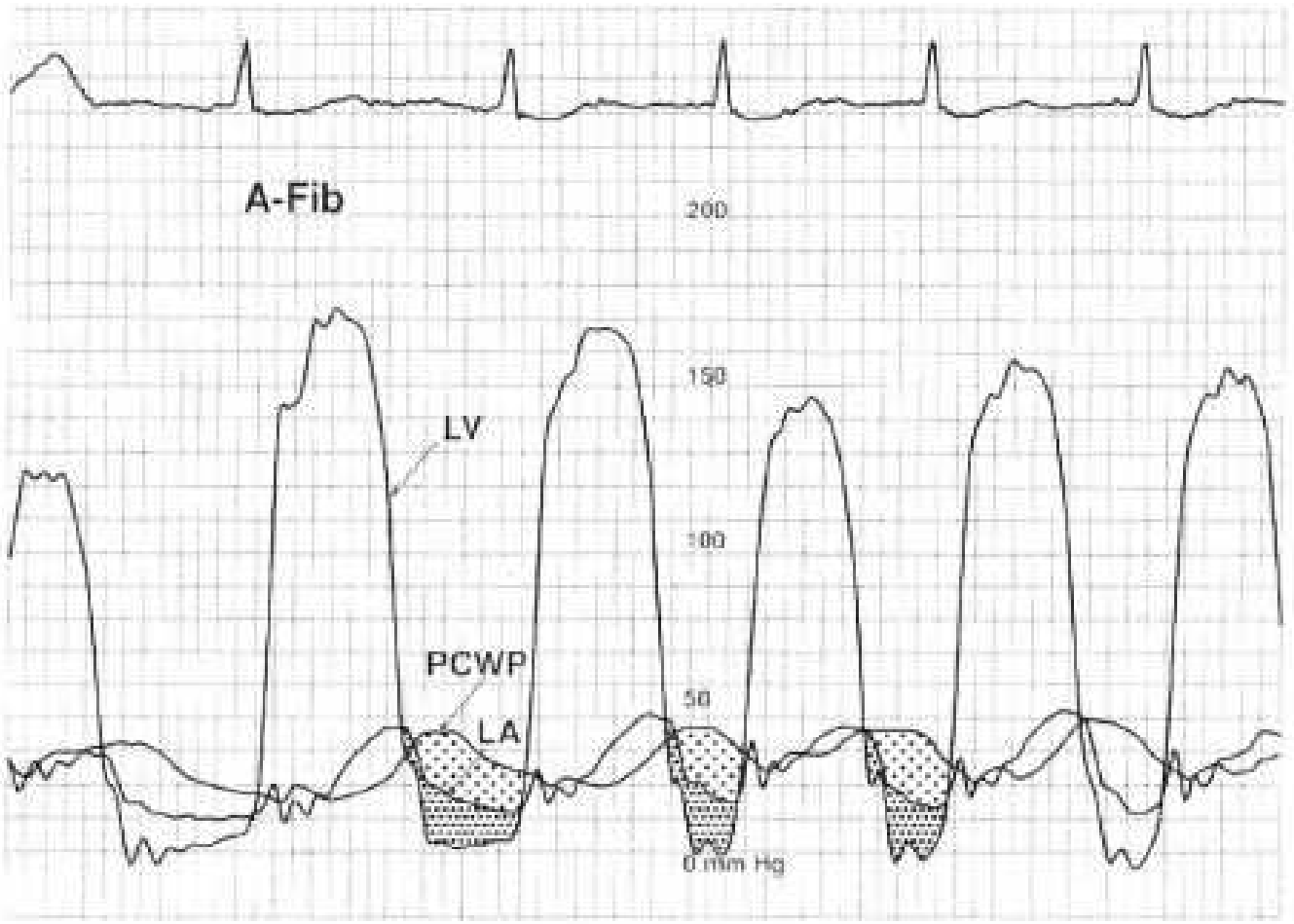


Fig. 26. Severe mitral stenosis. Simultaneous left ventricular (LV), pulmonary capillary wedge pressure (PCWP), and left atrial (LA) pressure tracings. This case illustrates the possibility of overestimating the mitral valve gradient if the PCWP rather than the true LA pressure is measured. Large stiple, gradient between PCWP and LV (false gradient across the mitral valve); small stiple, gradient between LA and LV (true gradient across the mitral valve).

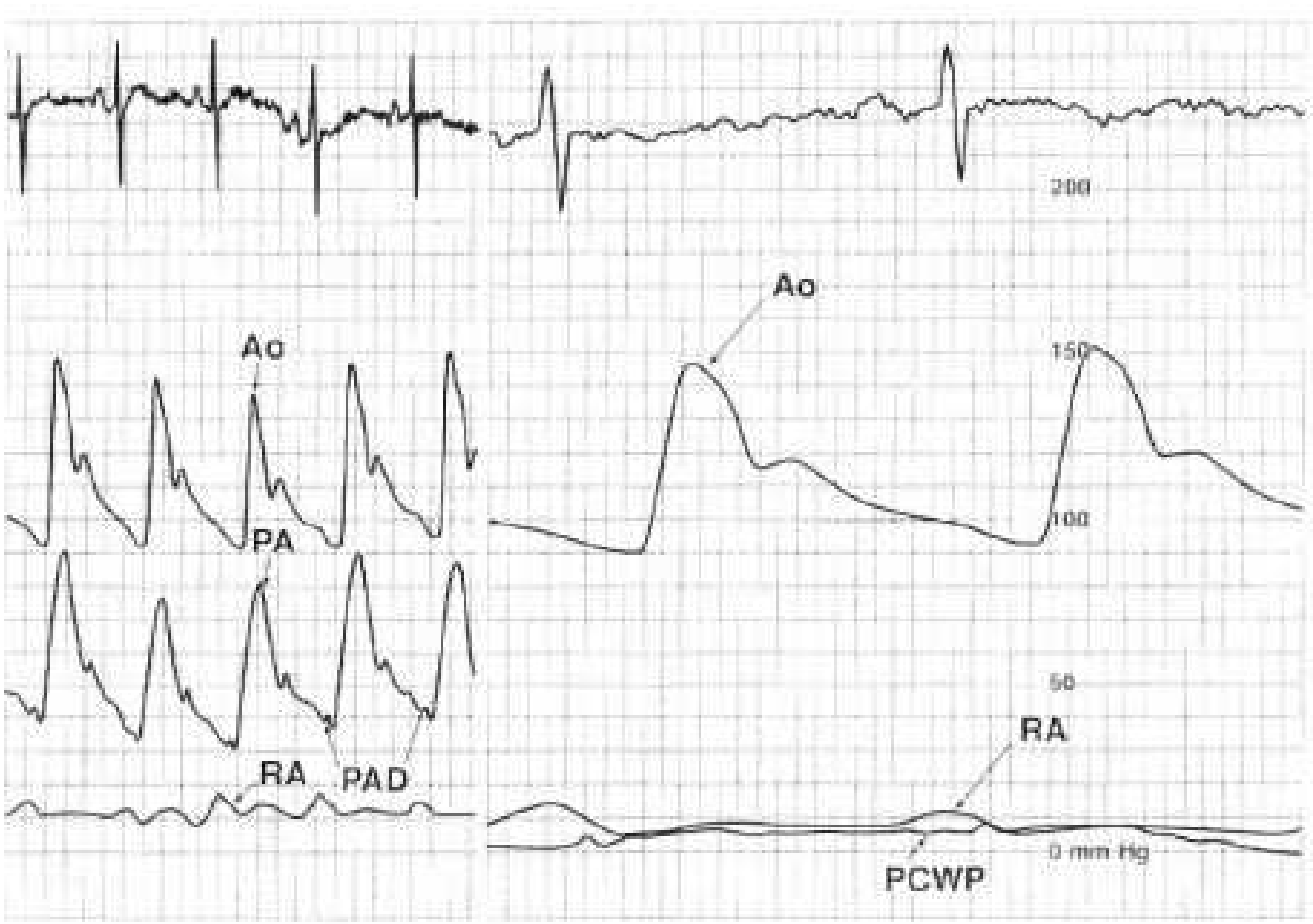


Fig. 27. Pulmonary hypertension. Right heart catheterization is frequently used to identify the cause of pulmonary hypertension, that is, cardiac vs. pulmonary cause. This can be readily appreciated while assessing the relationship of pulmonary artery diastolic pressure (PAD) and pulmonary capillary wedge pressure (PCWP). Normally, and in cases in which pulmonary hypertension is due to a cardiac cause (e.g., left ventricular dysfunction, mitral stenosis), PWCP is 2 to 3 mm less than PAD. However, when the hypertension has a pulmonary cause (e.g., primary pulmonary hypertension, secondary pulmonary hypertension from thromboemboli, pulmonary fibrosis), the PAD may be significantly higher, while the pulmonary artery wedge pressure remains normal, producing a significant difference between these two pressures. In this figure, the patient had primary pulmonary hypertension with a mean PAD of 35 mm Hg and a PCWP of only 7 mm Hg. Note that the right atrial pressure, RA, is slightly higher than the PCWP. Ao, aorta.

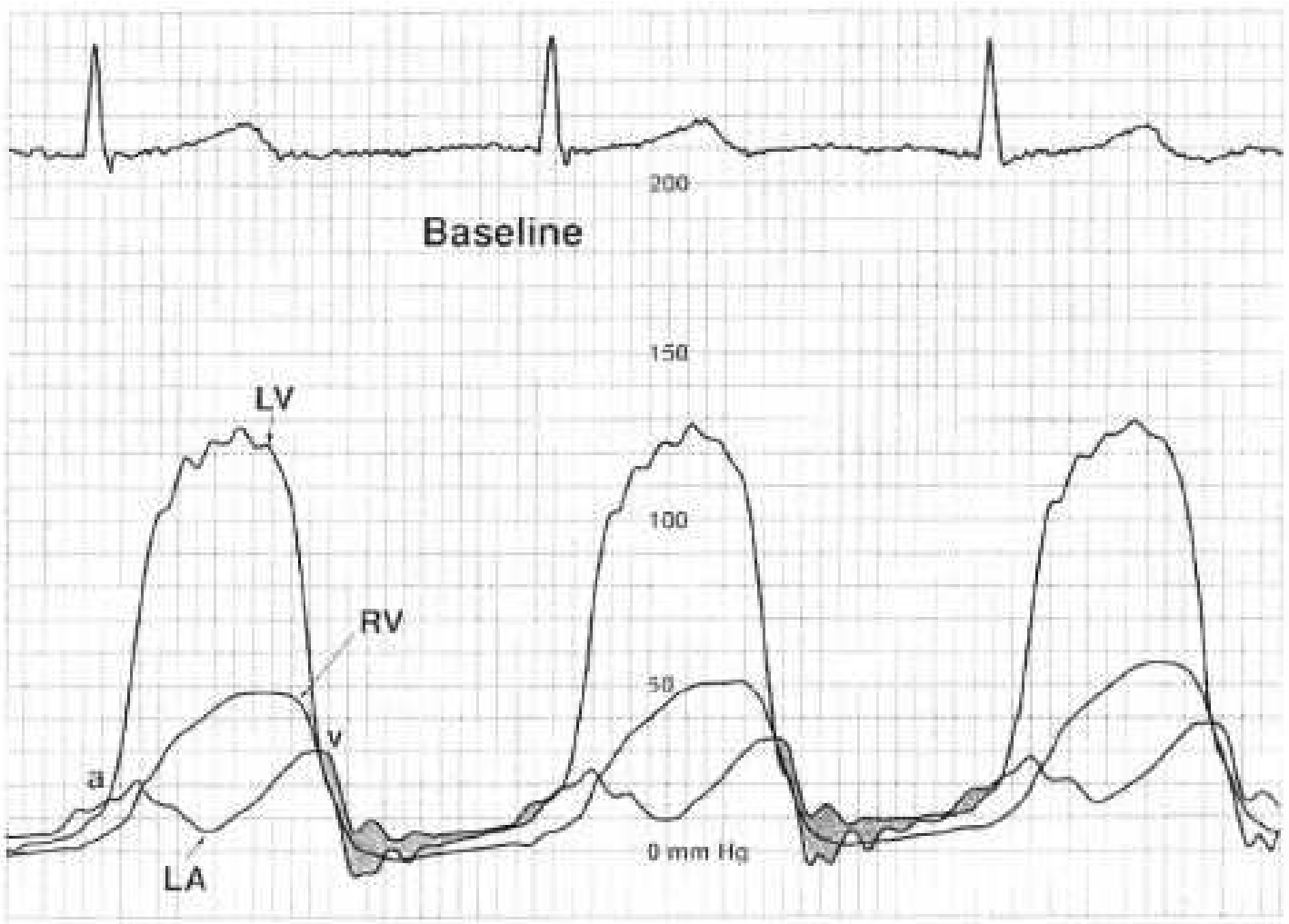


Fig. 28. Mitral stenosis. Pressure tracings in a patient with significant exercise intolerance. The patient was found to have mild mitral stenosis. Rest tracing with a heart rate of approximately 70 beats/min. LV, left ventricular pressure; RV, right ventricular pressure; LA, left atrial pressure. The shaded area represents the pressure gradient across the mitral valve.

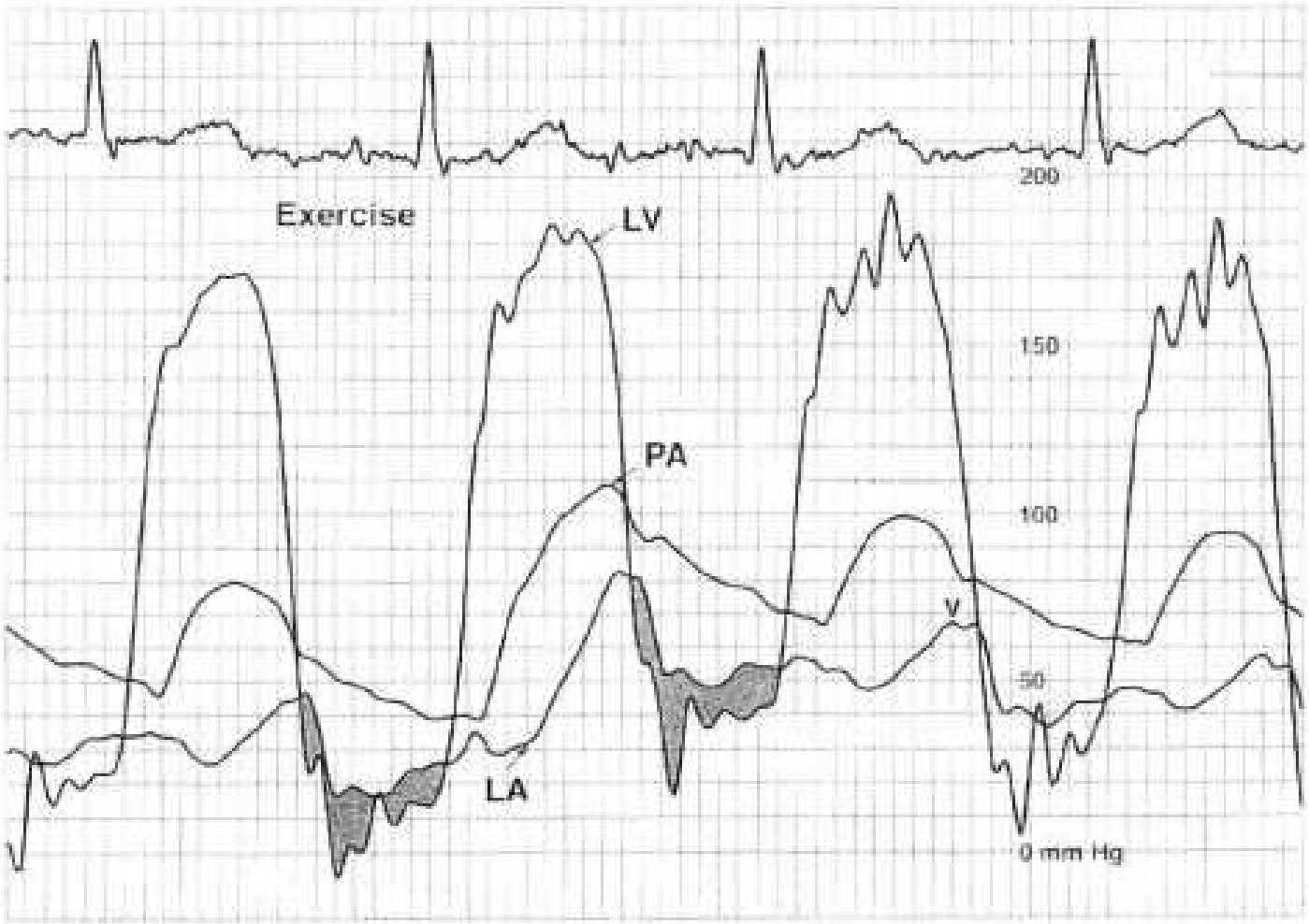


Fig. 28. Effect of exercise. This tracing is from the same patient as in Figure 27. After 4 minutes of exercise, a significant increase in the gradient across the mitral valve was noted. Observe the marked increase in right heart pressures (catheter now in pulmonary artery [PA]) from 50 mm Hg to about 100 mm Hg and the increase in left atrial pressure (LA) (about 30 mm Hg to about 80 mm Hg at the maximal height of the LA "v" wave). The exercise heart rate was approximately 110 beats/min. The shaded area represents the pressure gradient across the mitral valve.

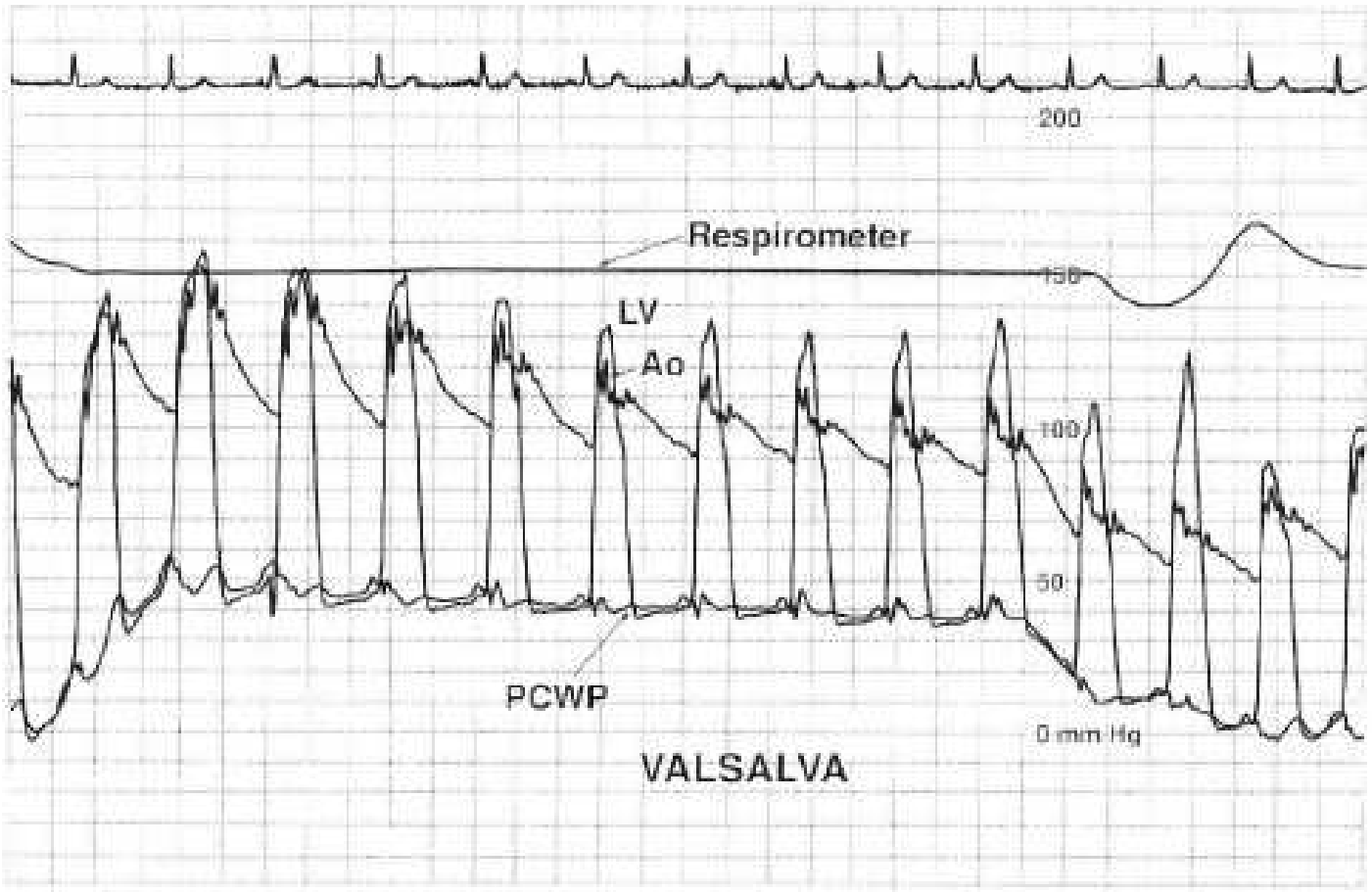


Fig. 30. Hypertrophic obstructive cardiomyopathy. Dynamic left ventricular outflow tract obstruction during phase 2 of the Valsalva maneuver. Note the significant increase in left ventricular (LV) end-diastolic pressure and steady decrease in LV systolic pressure during phase 2 of the Valsalva maneuver along with an increase in the outflow tract gradient. Ao, aorta; PCWP, pulmonary capillary wedge pressure.

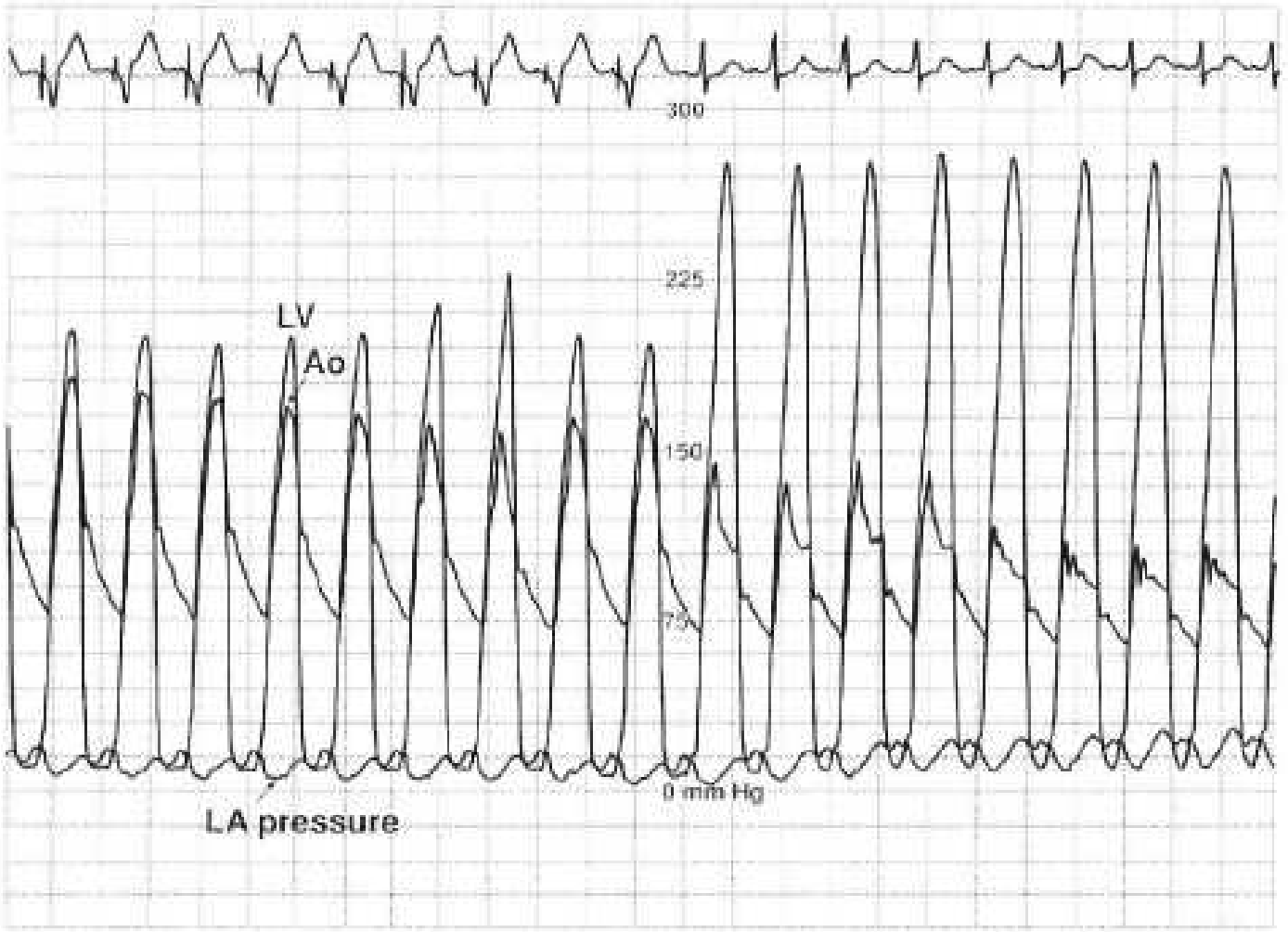


Fig. 31. Effect of pacing on dynamic left ventricular outflow tract gradient. This tracing is from a patient with severe hypertrophic obstructive cardiomyopathy who was evaluated in the catheterization laboratory to assess whether pacing would be beneficial in decreasing the outflow gradient. Both chambers were paced with varying intervals, and the effect of each pacing regimen was assessed. In the first half of the figure, the patient is being paced in a P-synchronized mode with an atrioventricular interval of 100 ms. In the second half of the figure, the pacing is discontinued and the patient is in sinus rhythm. Note the significant worsening of the outflow gradient after the pacing is discontinued. Also note the increase in mean left atrial pressure with discontinuation of pacing secondary to worsening of the outflow gradient. LV, left ventricular pressure; LA, left atrial pressure; Ao, aortic pressure.

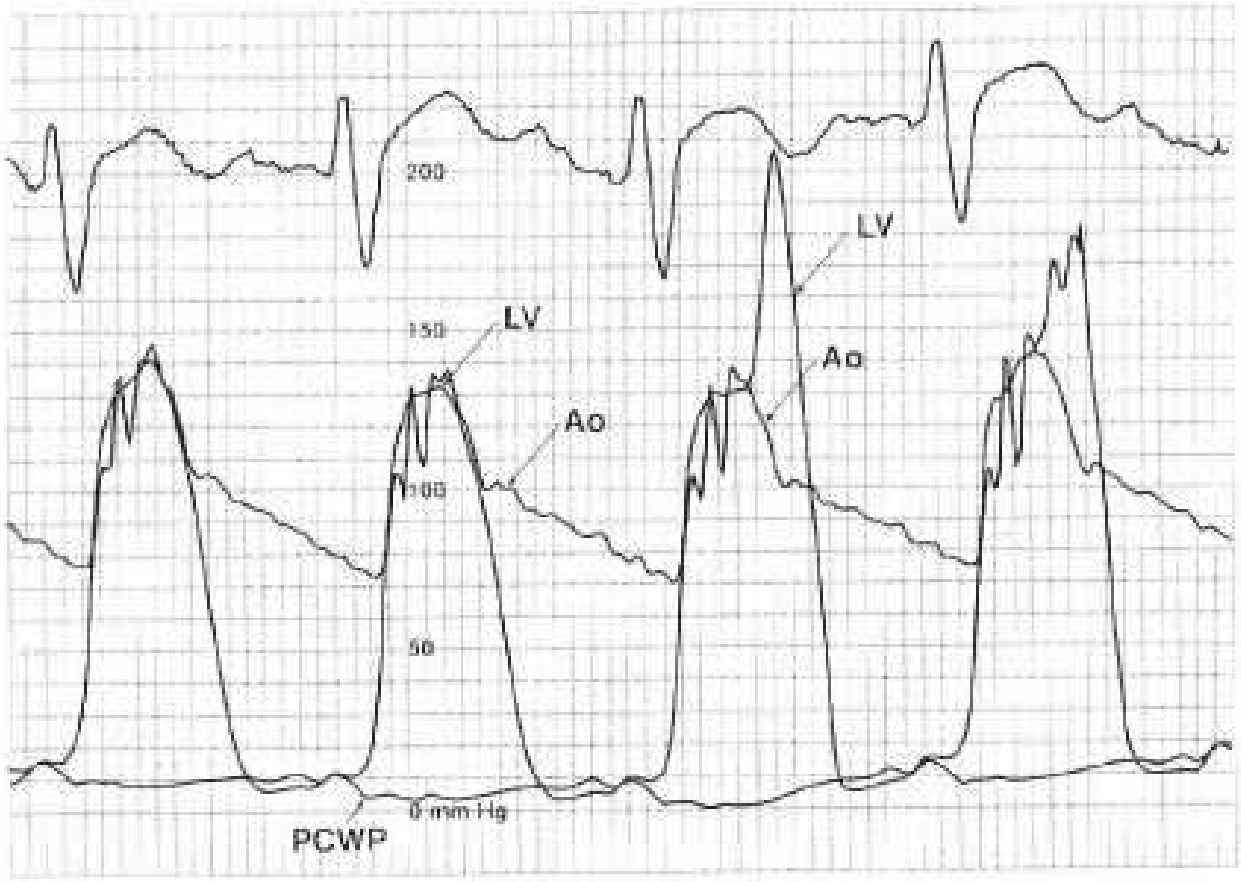
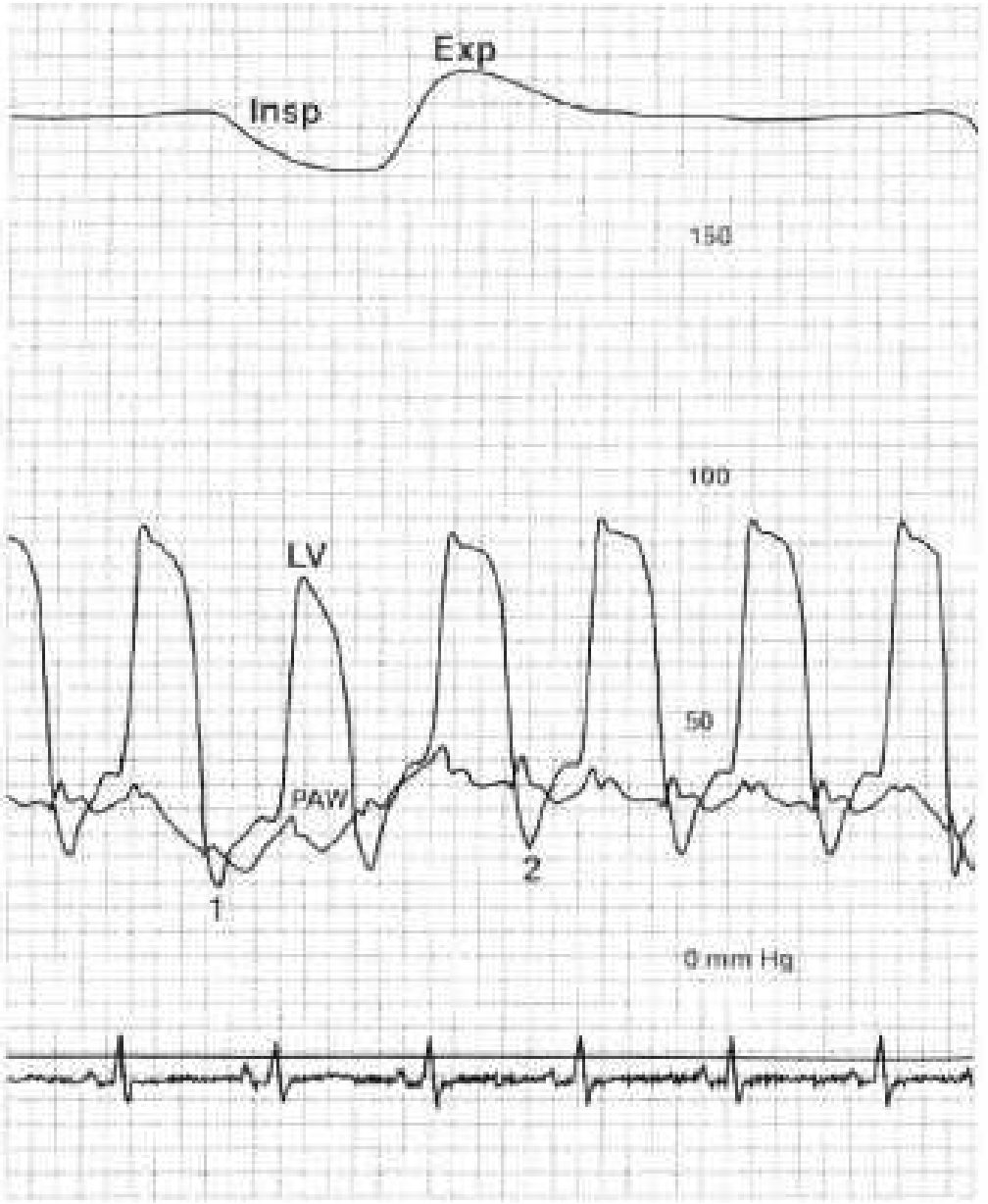


Fig. 33. Artifact. This is a tracing of catheter entrapment. Beat no. 3 is an artifact from left ventricular (LV) catheter entrapment in the small hyperdynamic cavity. The key points that differentiate this from Brockenbrough sign are the absence of premature ventricular contraction and the fact that the aortic pulse pressure (Ao) did not decrease with the apparent increase in the LV systolic pressure.

Fig. 34. Dissociation of intrathoracic and intracardiac pressures in constrictive pericarditis. The following 3 tracings are from a patient with surgically proven constrictive pericarditis. Simultaneous recordings of left ventricular and pulmonary capillary wedge pressures demonstrating dissociation of intrathoracic and intracardiac pressures. Note the decrease in early diastolic gradient with inspiration (Insp) (beat marked "1") and the increase with expiration (Exp) (beat marked "2"). Also note the dip-and-plateau morphology of left ventricular (LV) diastolic pressures. The nasal respirometer tracing is also shown. PAW, pulmonary artery wedge.



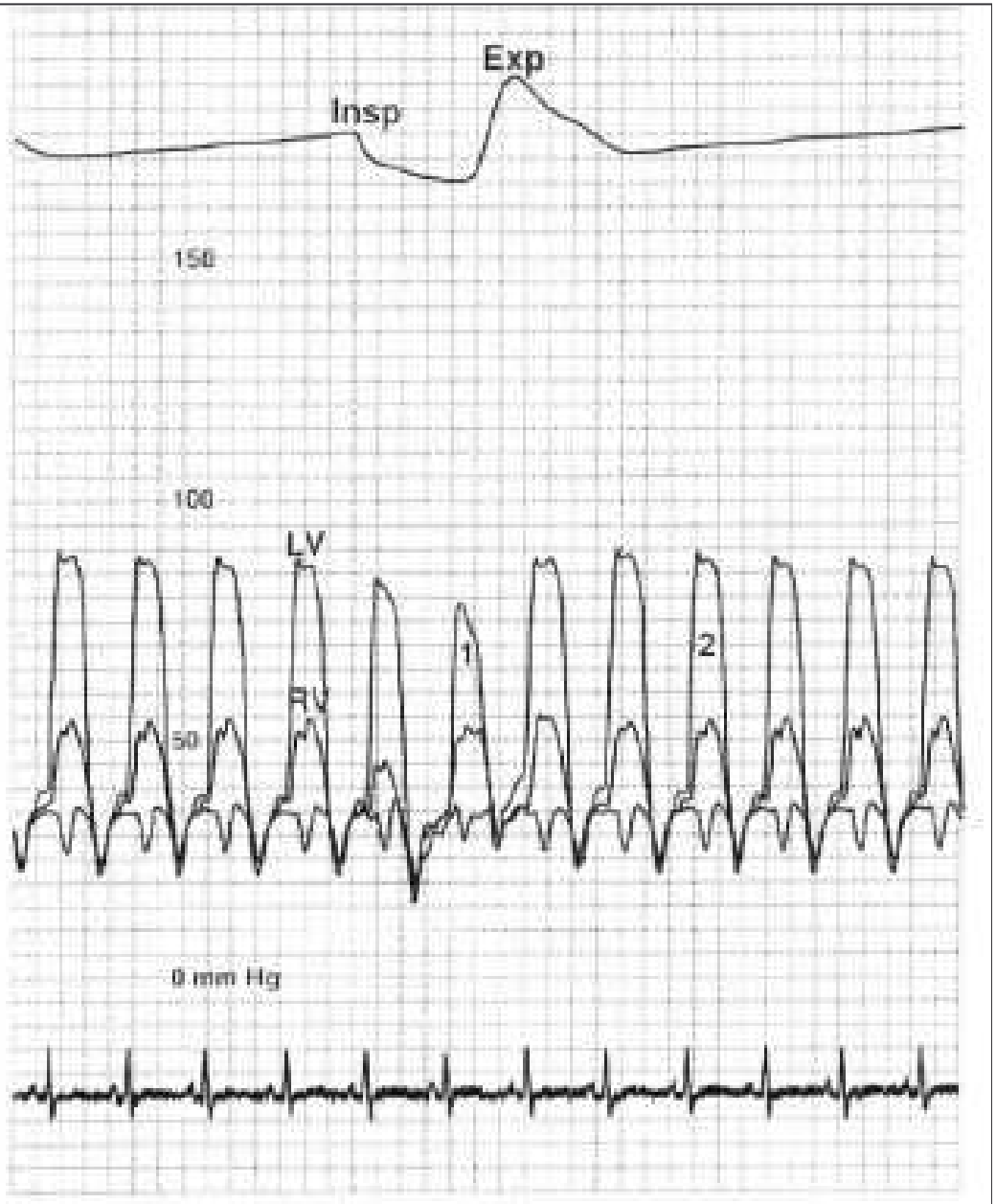
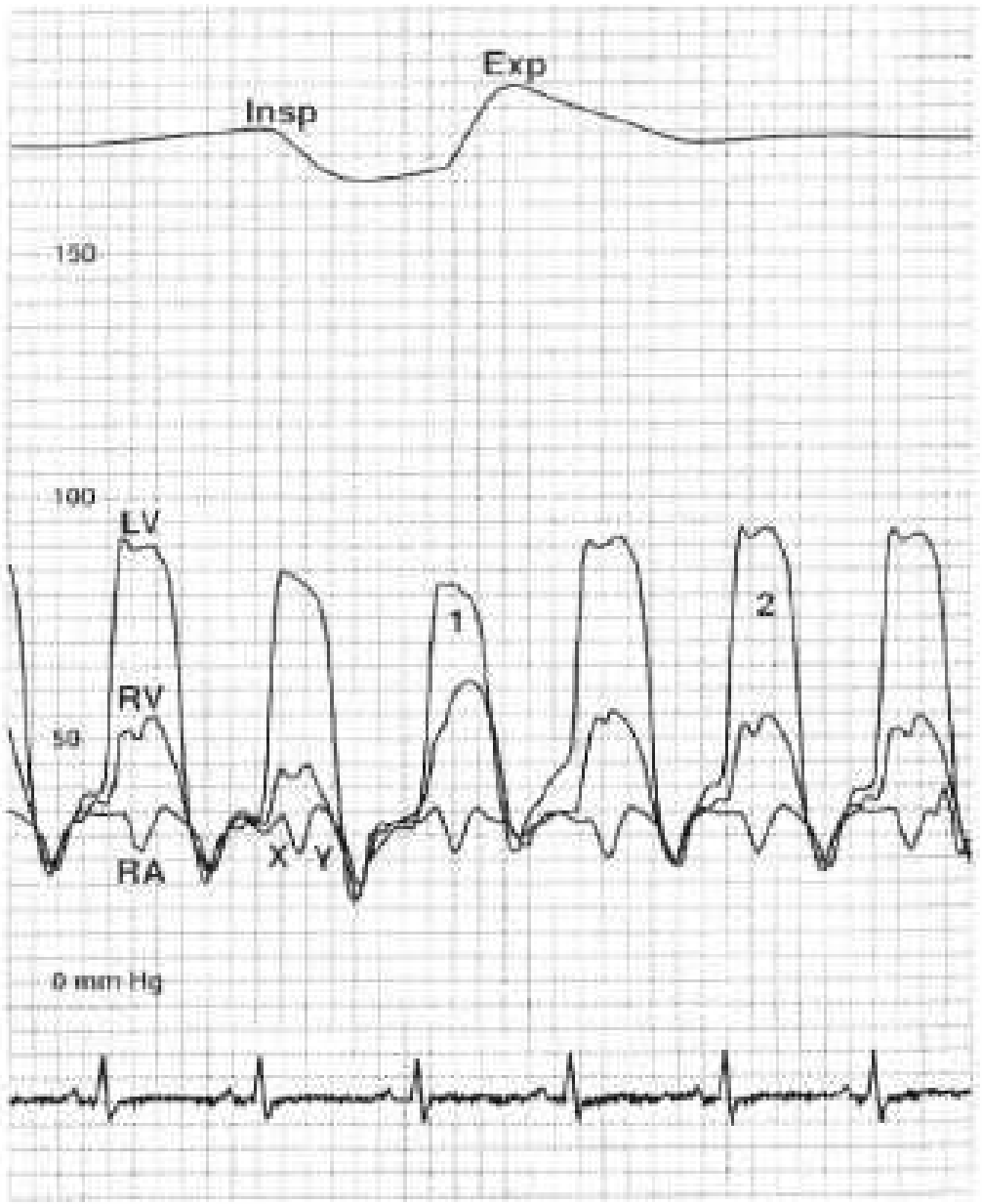


Fig. 35. Ventricular interdependence in constrictive pericarditis. Simultaneous recordings of left ventricular (LV), right ventricular (RV), and right atrial pressures demonstrating ventricular interdependence. Note the discordance in LV and RV systolic pressures with respiration (beats 1 and 2). Other criteria of constrictive pericarditis are also seen, e.g., a marked "W" or "M" pattern in the right atrial pressure tracing, absence of decrease in right atrial pressure with inspiration (Kussmaul sign), right ventricular end-diastolic pressure (RVEDP) > 1/3 of right ventricular systolic pressure (RVSP), and equalization of pressures (< 5 mm difference in left ventricular end-diastolic pressure and RVEDP). However, the RVSP (and, therefore, pulmonary artery systolic pressure, in the absence of RV outflow gradient) is slightly above 55 mm Hg. The nasal respirometer tracing is also shown. Exp, expiration; Insp, inspiration.

Fig. 36. Hemodynamic tracings in constrictive pericarditis. Higher paper speed (100 mm/s) simultaneous recordings of left ventricular (LV), right ventricular (RV), and right atrial (RA) pressures demonstrating ventricular interdependence. Note the decrease in RV systolic pressure during the first beat and a marked rise in the next ejection at peak inspiration (beat 1) while LV systolic pressure decreases. Note the rapid x and y descents in the RA tracing. The nasal respirometer tracing is shown at the top. Exp, expiration; Insp, inspiration.



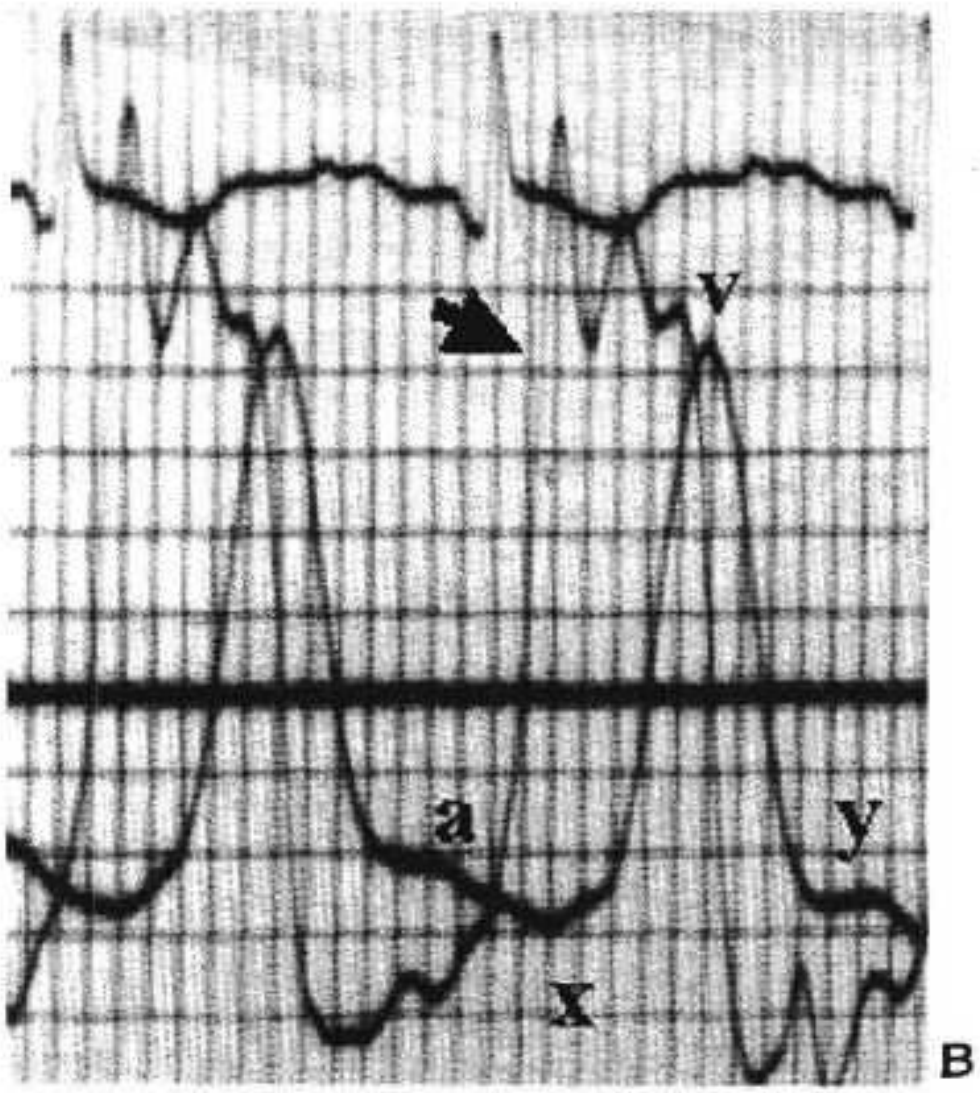


FIGURE 1. *A*, Chronic severe (4+) MR. Complete left atrium (LA) opacification was observed on the first beat after contrast injection, and the contrast material did not clear from the LA, but became more intense with every beat. Note the substantial LA dilatation. *B*, V wave in MR. Pulmonary capillary wedge pressure in a patient with severe MR. Note the significant increase in v wave amplitude, as a result of blood regurgitation from the LV into the LA, with consequent increase in left atrial and pulmonary capillary wedge pressure. a = *atrial systole*; v = *ventricular systole*; *x* = *x* descent; *y* = y descent; arrow = LV pressure tracing.

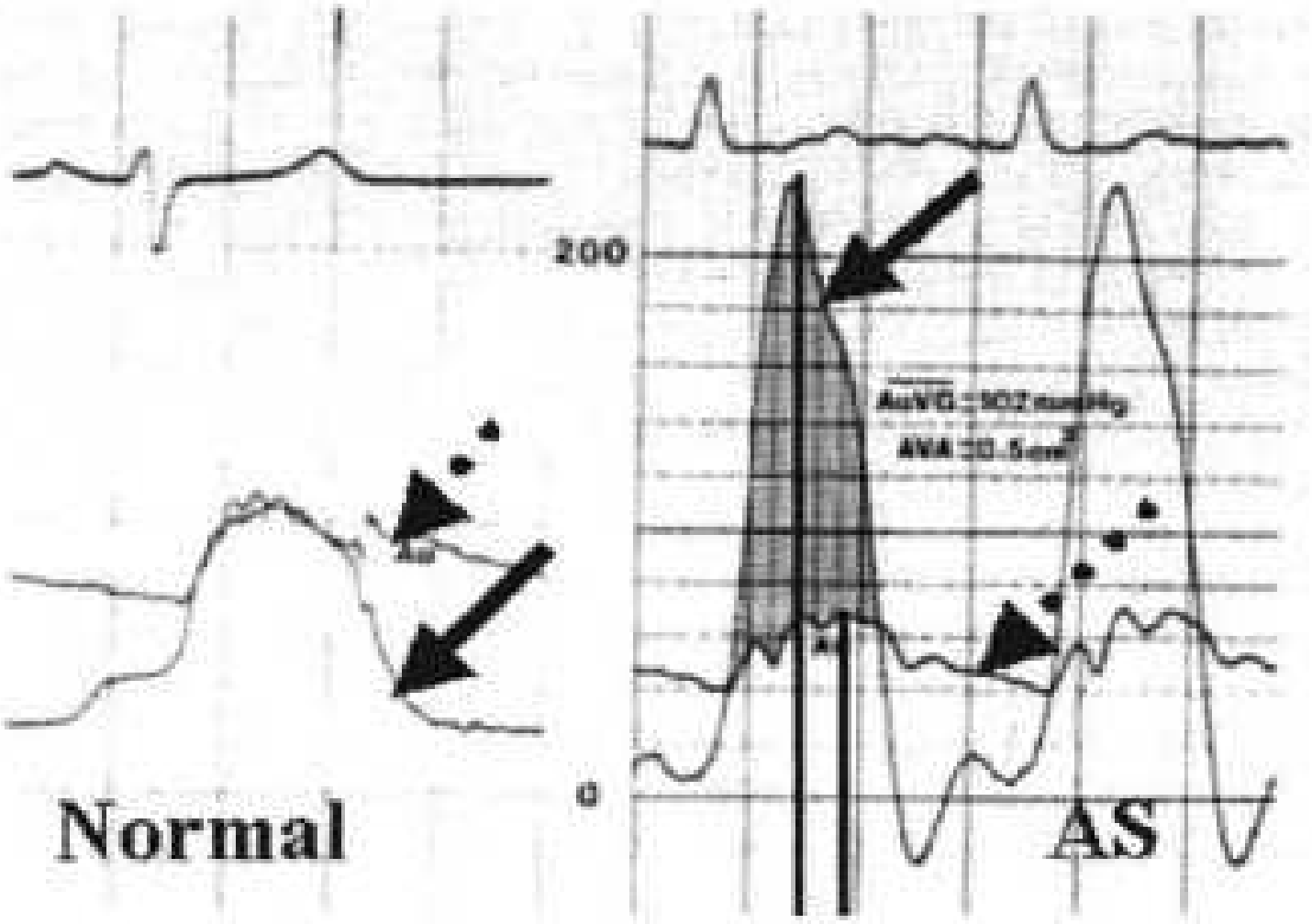


FIGURE 3. Invasive assessment of valvular gradient in a patient with aortic stenosis. The catheter-derived transvalvular aortic gradient is calculated off-line as the difference between the peak pressure in the LV (*continuous arrows*) and the aorta (*dotted arrows*). Note the systolic LV–aortic gradient (*shaded area*) and the time lag between the two peak pressures (between the two vertical lines).

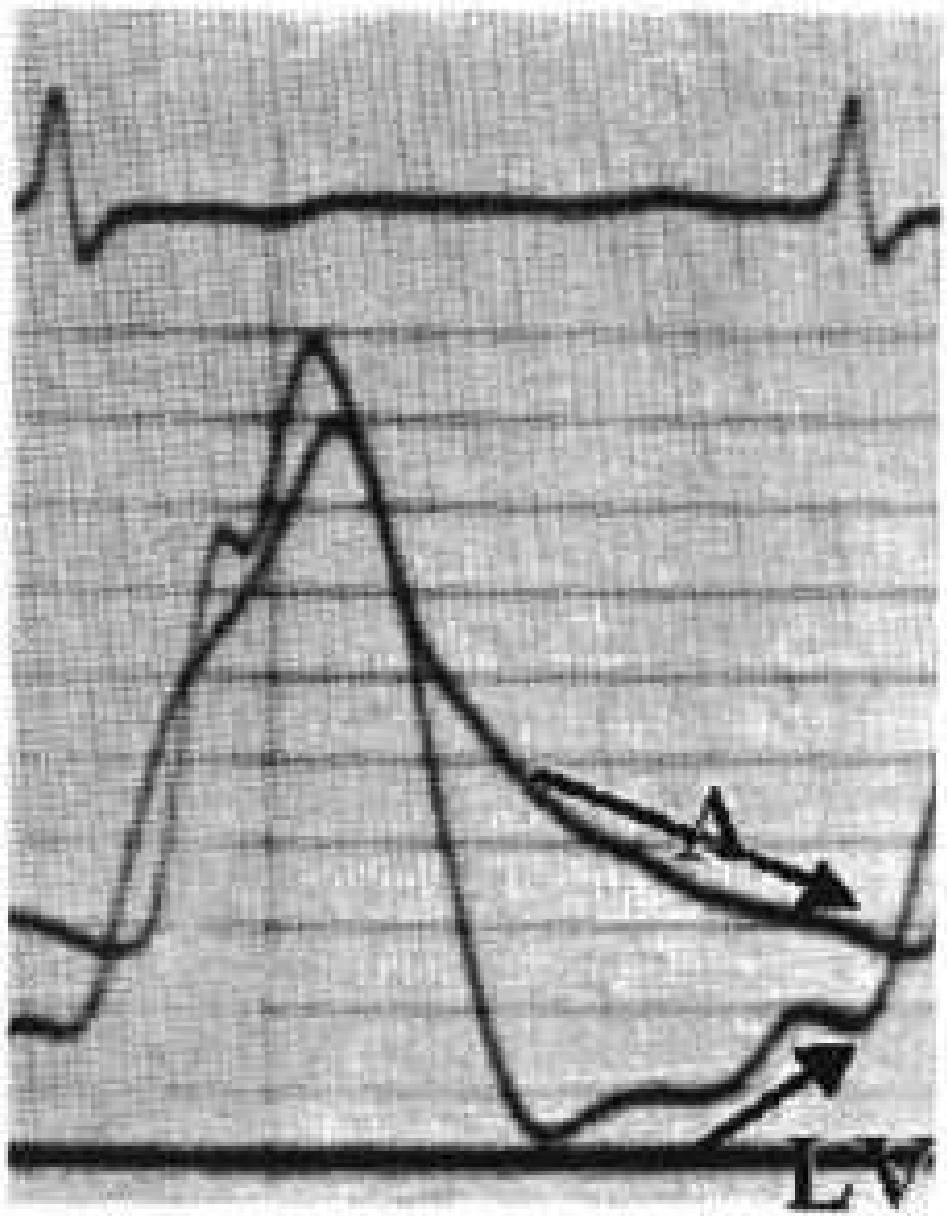


FIGURE 4. AI pressure curves. In aortic regurgitation, there is a rapid decrease in diastolic aortic pressure and a parallel rapid increase in the left ventricular pressure). The arrows indicate the evolution of the diastolic pressures (A, LV).

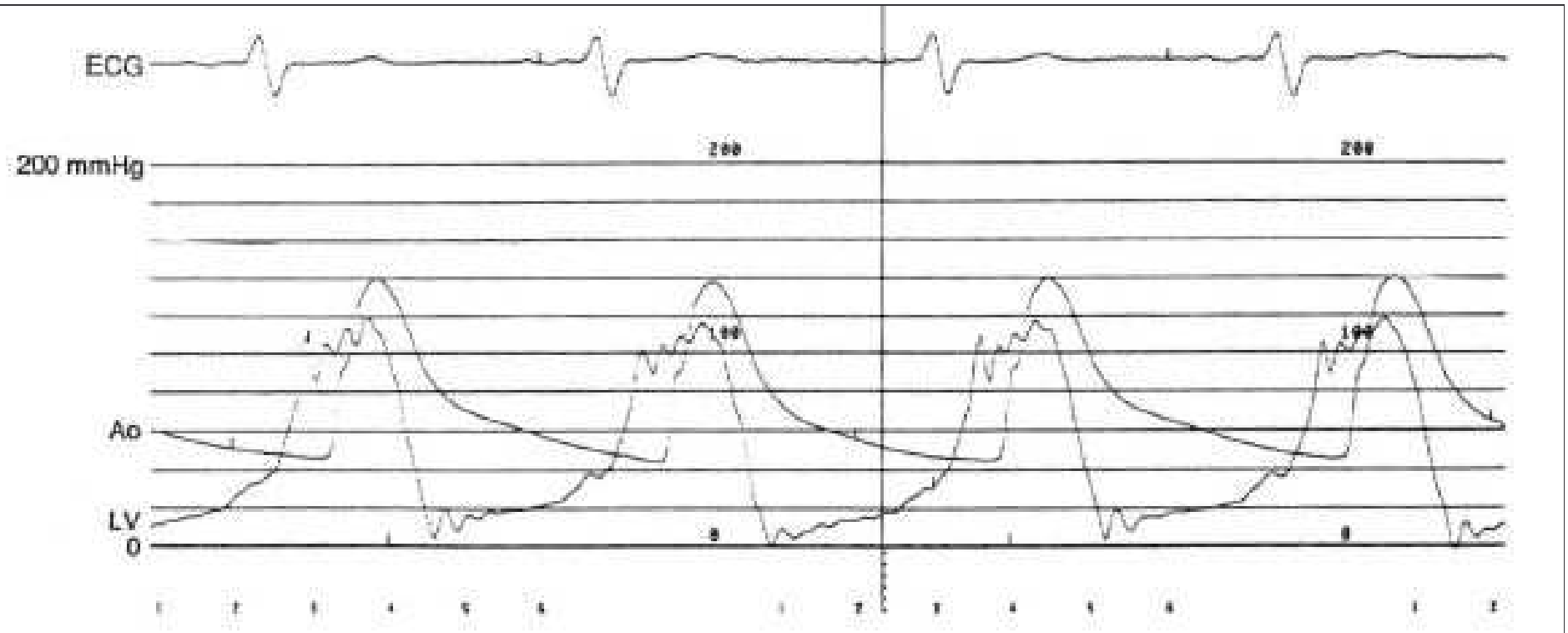


Fig. 1. Left ventricular (LV) and aortic (Ao) pressures using an 8 French femoral sheath and 7 French pigtail catheter with fluid-filled transducers in a patient with a diastolic murmur. See text for details.

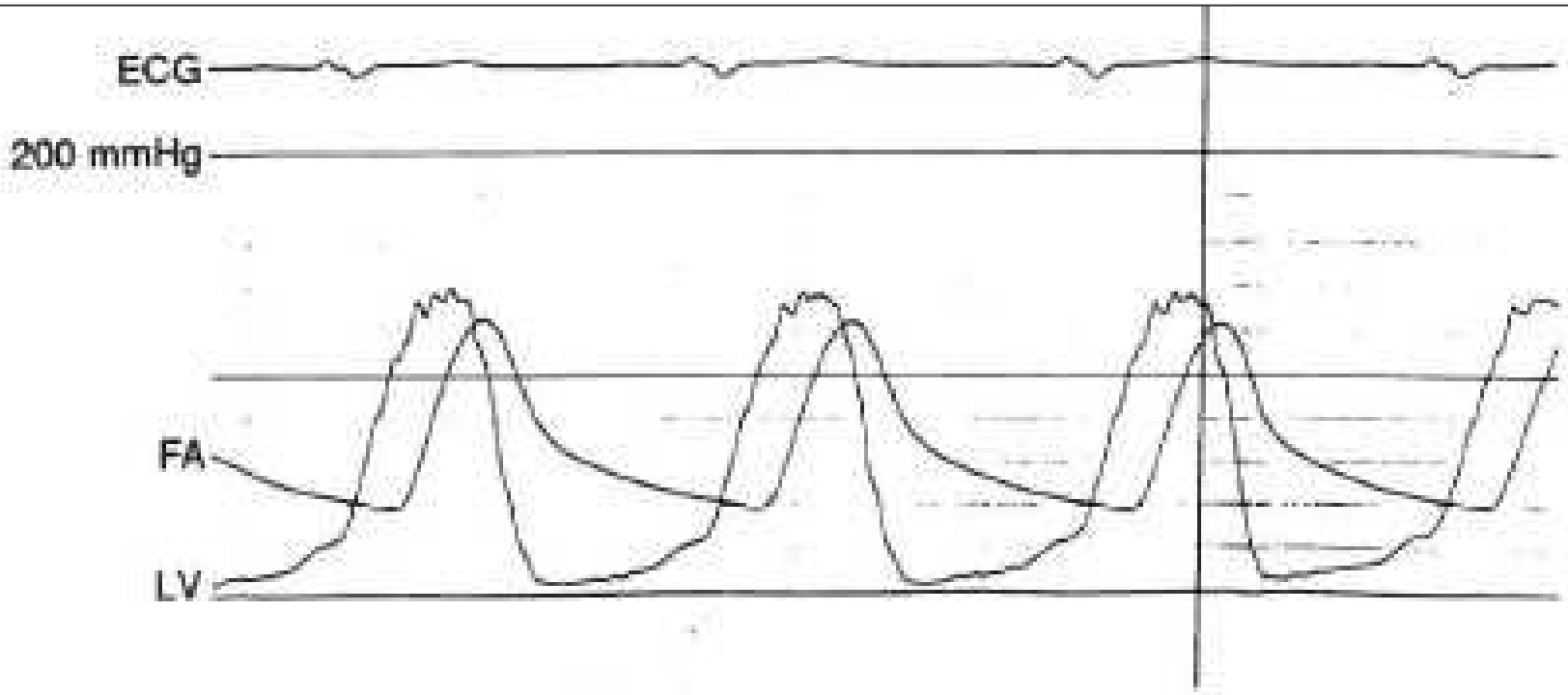


Fig. 2. Femoral artery (FA) and left ventricular (LV) pressures in a patient with systolic and diastolic murmurs. See text for details.

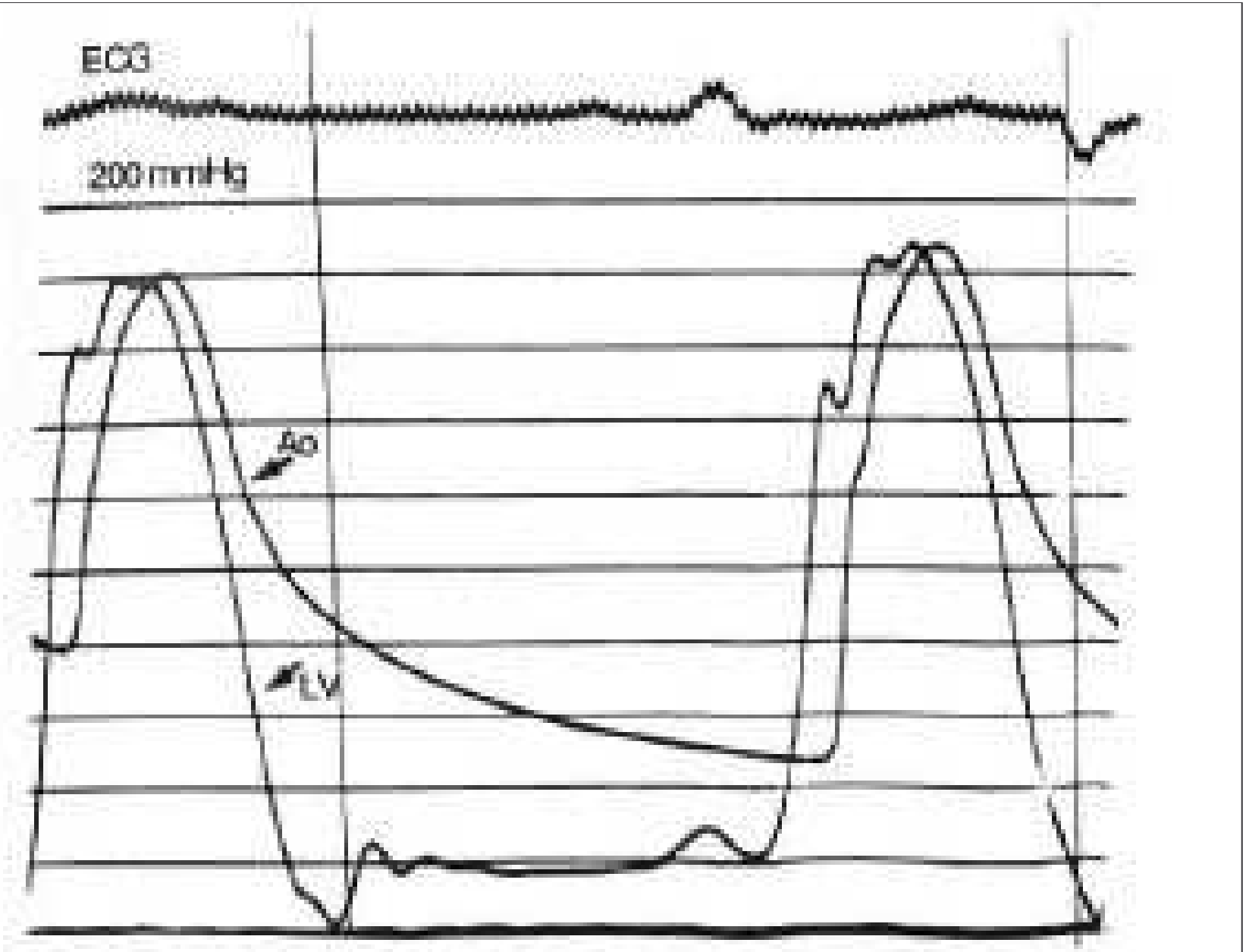


Fig. 3. Hemodynamics in a patient with hypertension. LV = left ventricular pressure; Ao = aortic pressure. See text for details.

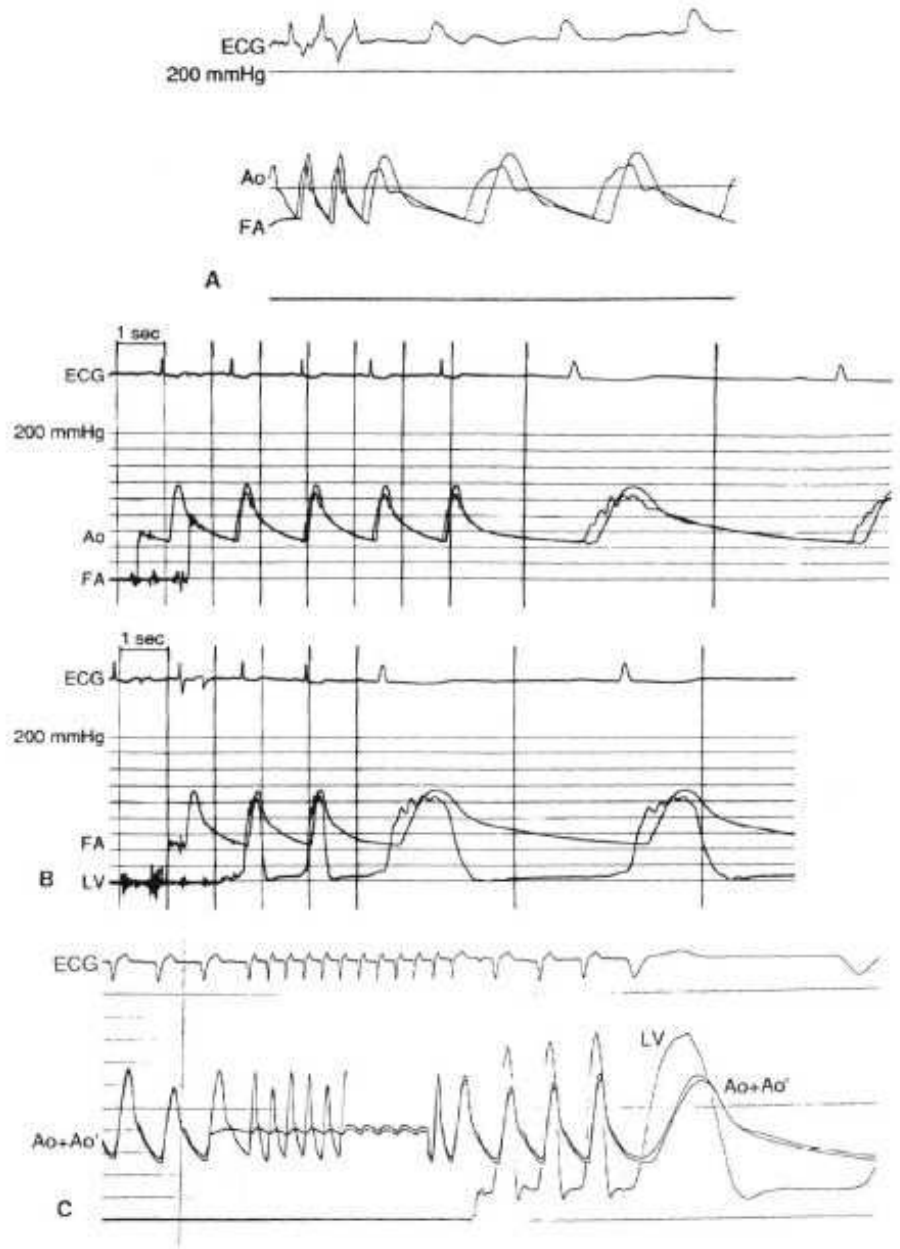


Fig. 4A: Central aortic (Ao) and femoral artery (FA) pressures in a normal subject. A 15 mm Hg femoral pressure overshoot is acceptable. See text for details.

Fig. 4B: Central aortic (Ao) and peripheral (FA) pressures are matched before recording left ventricular (LV)-aortic pressures. See text for details.

Fig. 4C: Systemic hemodynamics obtained with 2 catheters one in the central aortic position (Ao) and a second through the arterial sheath (Ao'). These pressures match with no signs of peripheral pressure amplification. The left ventricular (LV) pressure was obtained via a transseptal catheter. See text for details.

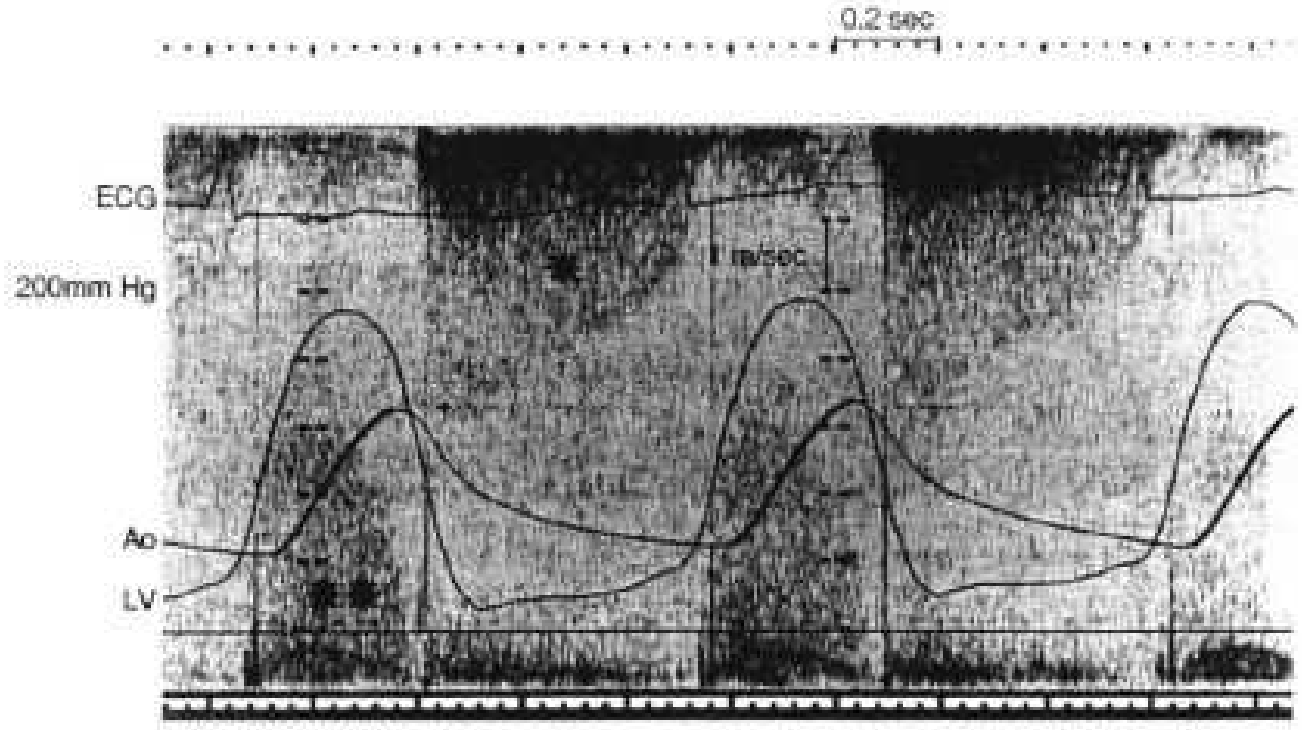


Fig. 5. Combined echo-Doppler and hemodynamic study in a patient with mixed aortic valve disease. Aortic (Ao) and left ventricular (LV) pressures measured as in figure 1. Flow velocities were obtained with continuous mode Doppler from the left ventricular outflow tract. Scale marks for Doppler are 1m/second. Scale marks for pressure are 40 mm Hg per division. (*) diastolic velocity integral; (**) systolic velocity intregral. See text for details.

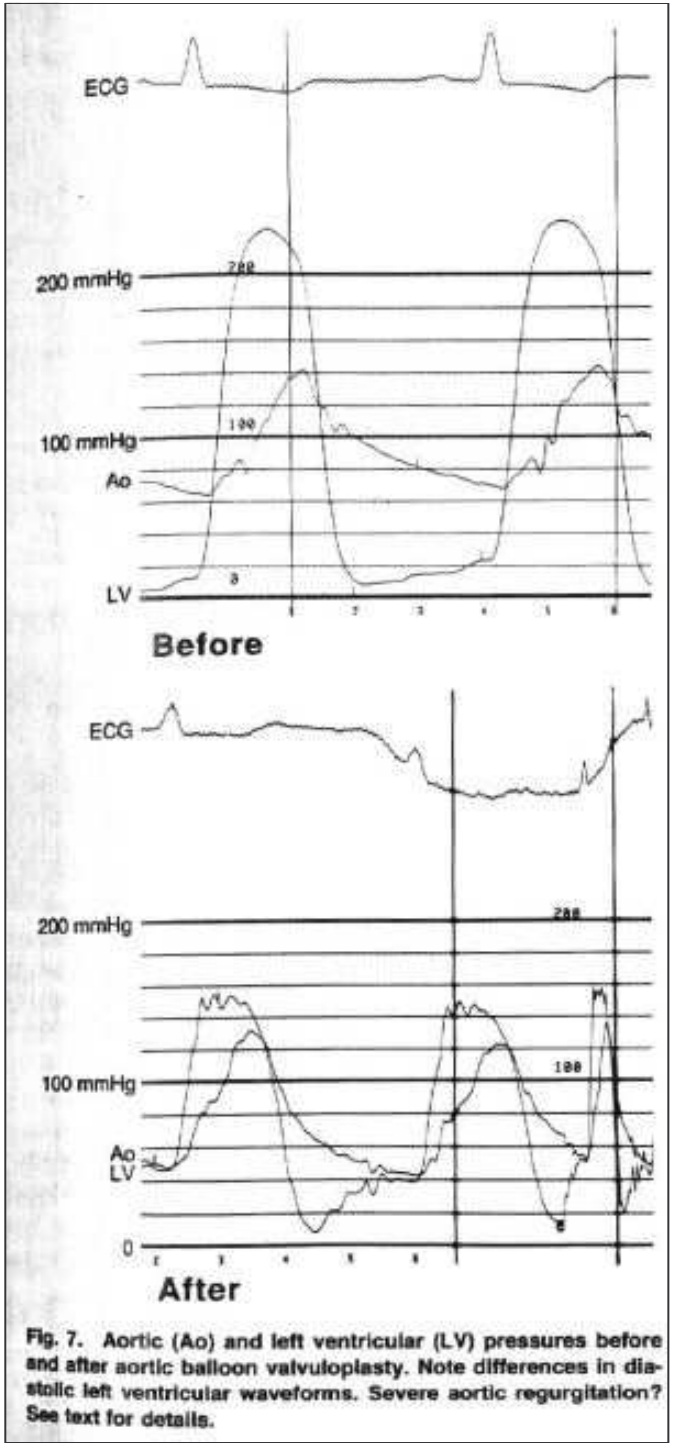


Fig. 7. Aortic (Ao) and left ventricular (LV) pressures before and after aortic balloon valvuloplasty. Note differences in diastolic left ventricular waveforms. Severe aortic regurgitation? See text for details.

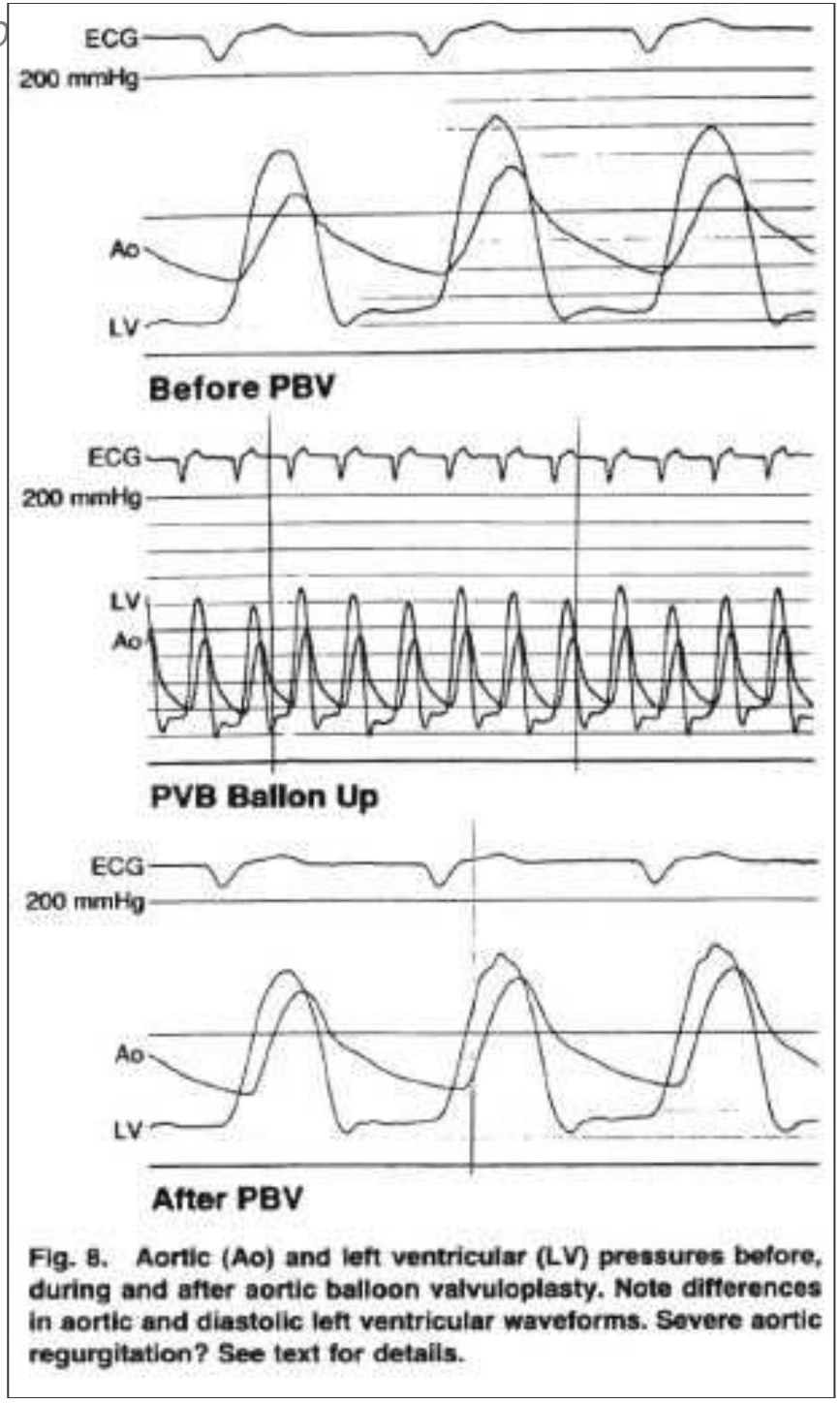


Fig. 8. Aortic (Ao) and left ventricular (LV) pressures before, during and after aortic balloon valvuloplasty. Note differences in aortic and diastolic left ventricular waveforms. Severe aortic regurgitation? See text for details.

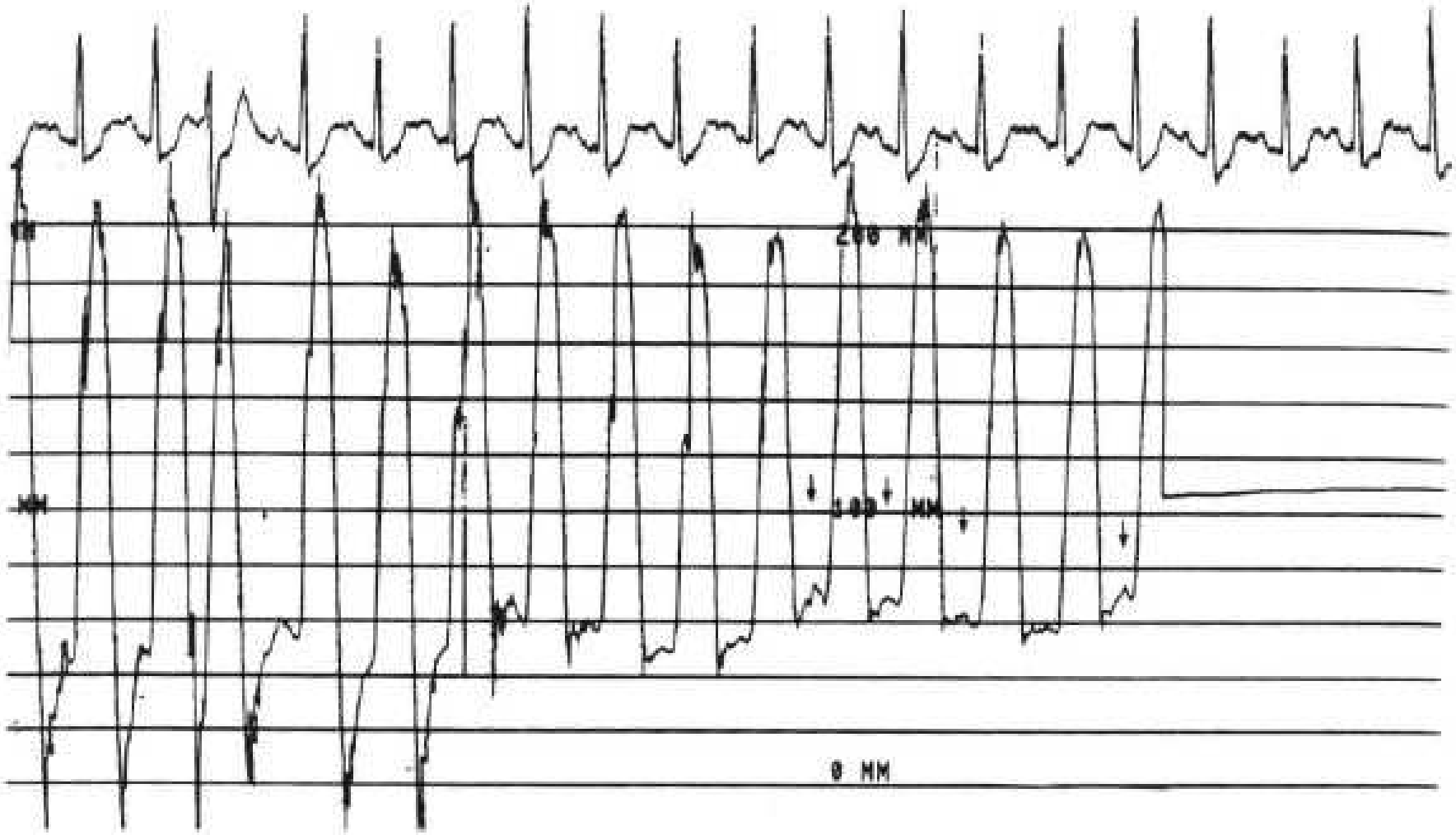


Fig. 3. Pressure recording from the LV pullback to ascending Ao. The pulse pressure is 140 mmHg and the Ao diastolic pressure is approximately equal to LV end diastolic pressure. Preceding the anacrotic limb of the Ao pressure tracing there are positive deflections correlating with the P waves on the EKG (arrow). These a-waves are due to premature opening of the Ao valve in acute AI. (Recording speed = 25 mm/sec.) Abbreviations: LV = left ventricle, Ao = aorta, AI = aortic insufficiency.

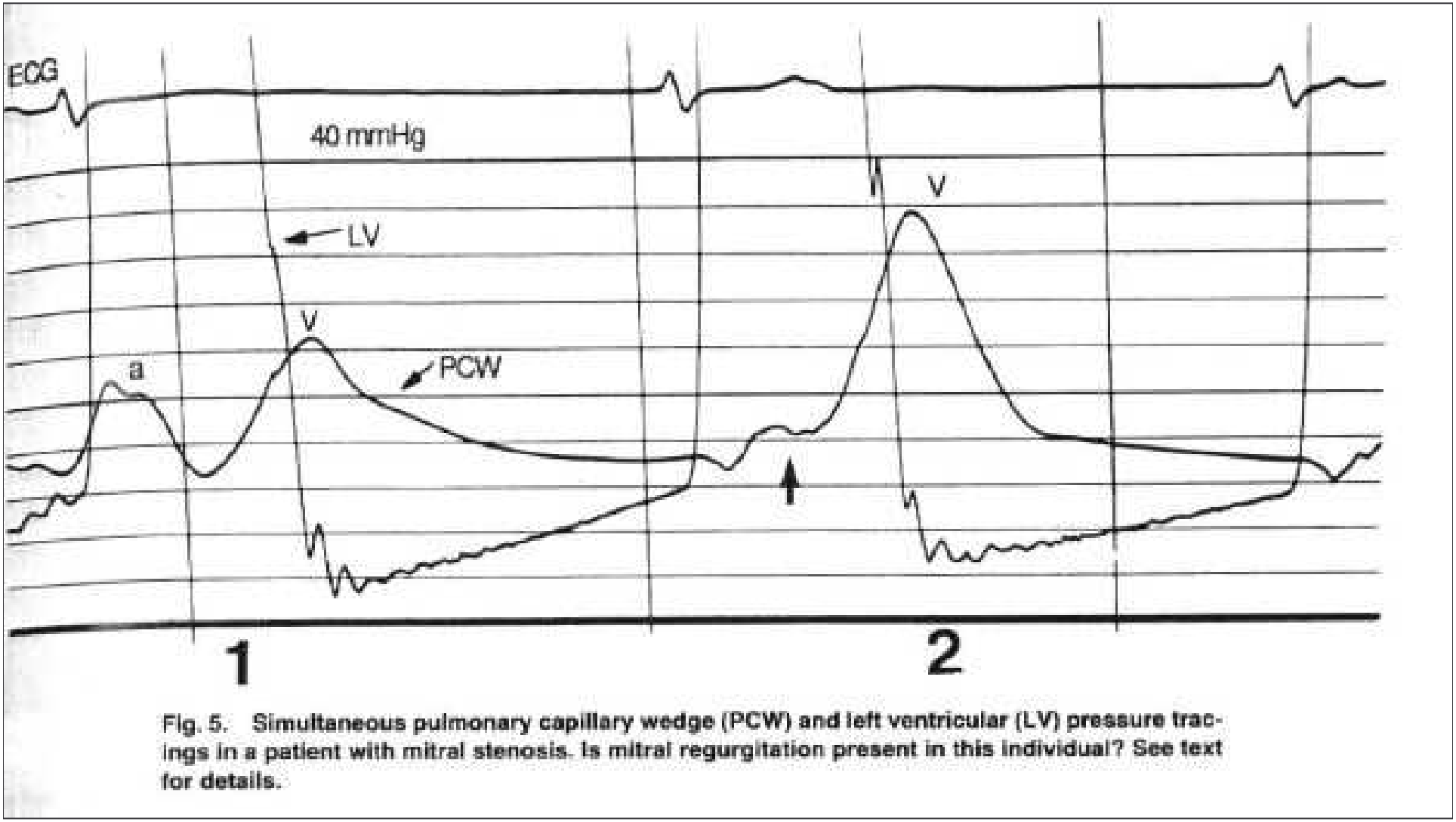


Fig. 5. Simultaneous pulmonary capillary wedge (PCW) and left ventricular (LV) pressure tracings in a patient with mitral stenosis. Is mitral regurgitation present in this individual? See text for details.

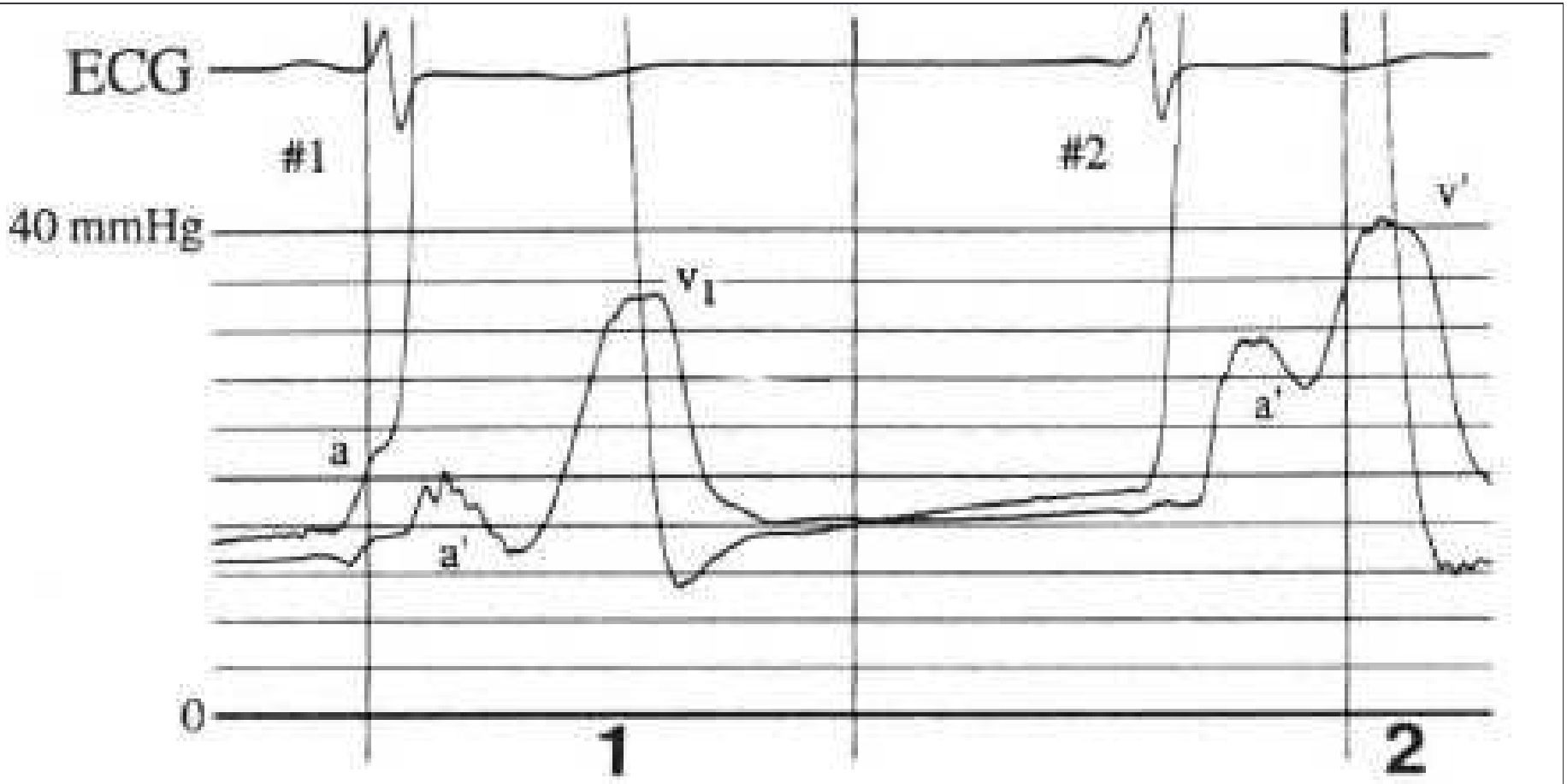


Fig. 6. Pulmonary capillary wedge and left ventricular pressure tracings in a patient with aortic stenosis. a′ and v′ are the pulmonary capillary wedge pressure waveforms. See text for details.

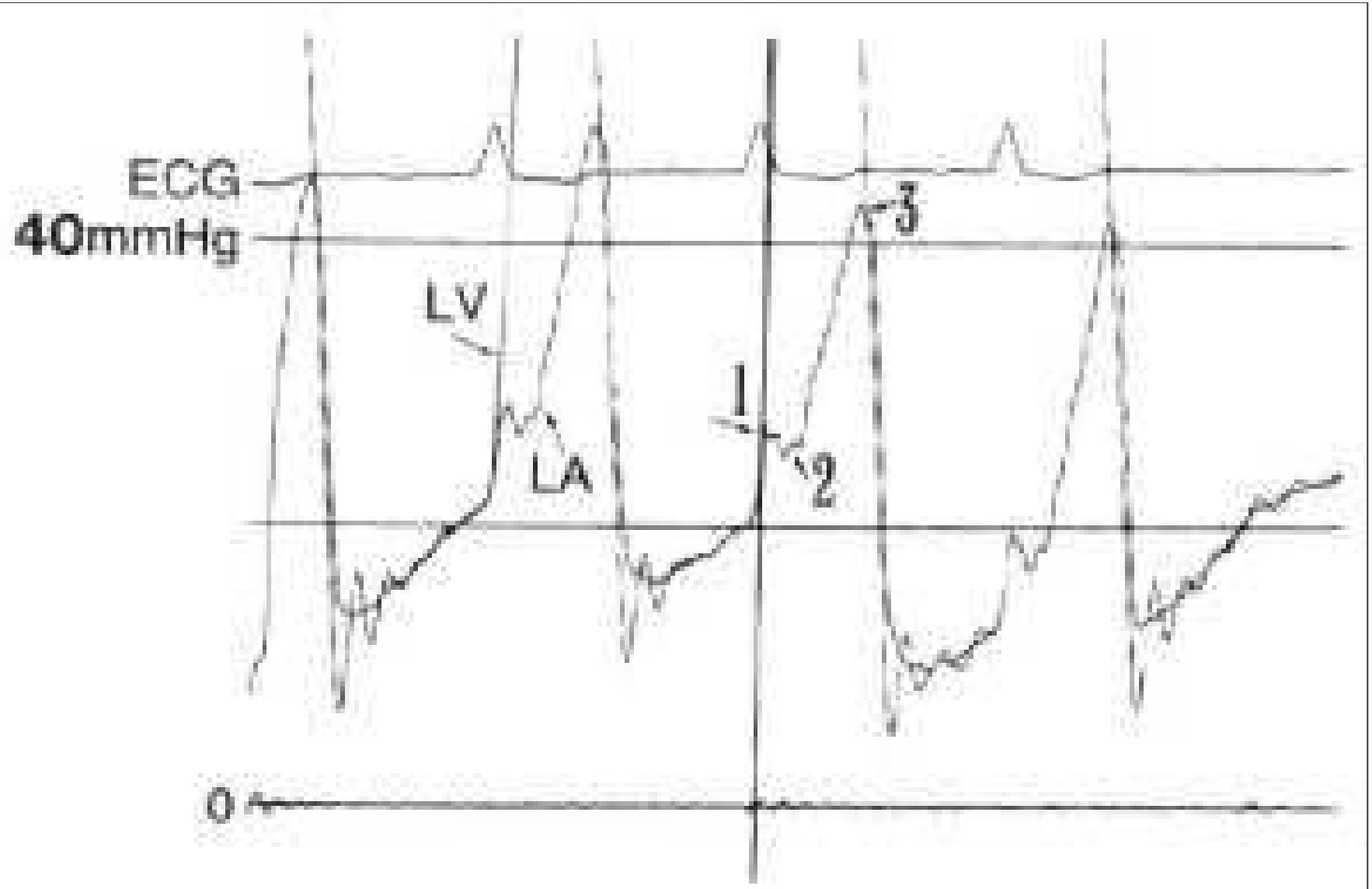


Fig. 7. Simultaneous left atrial (LA) and left ventricular (LV) pressure tracings (0–40 mm Hg scale) in a patient with new systolic murmur. See text for details.

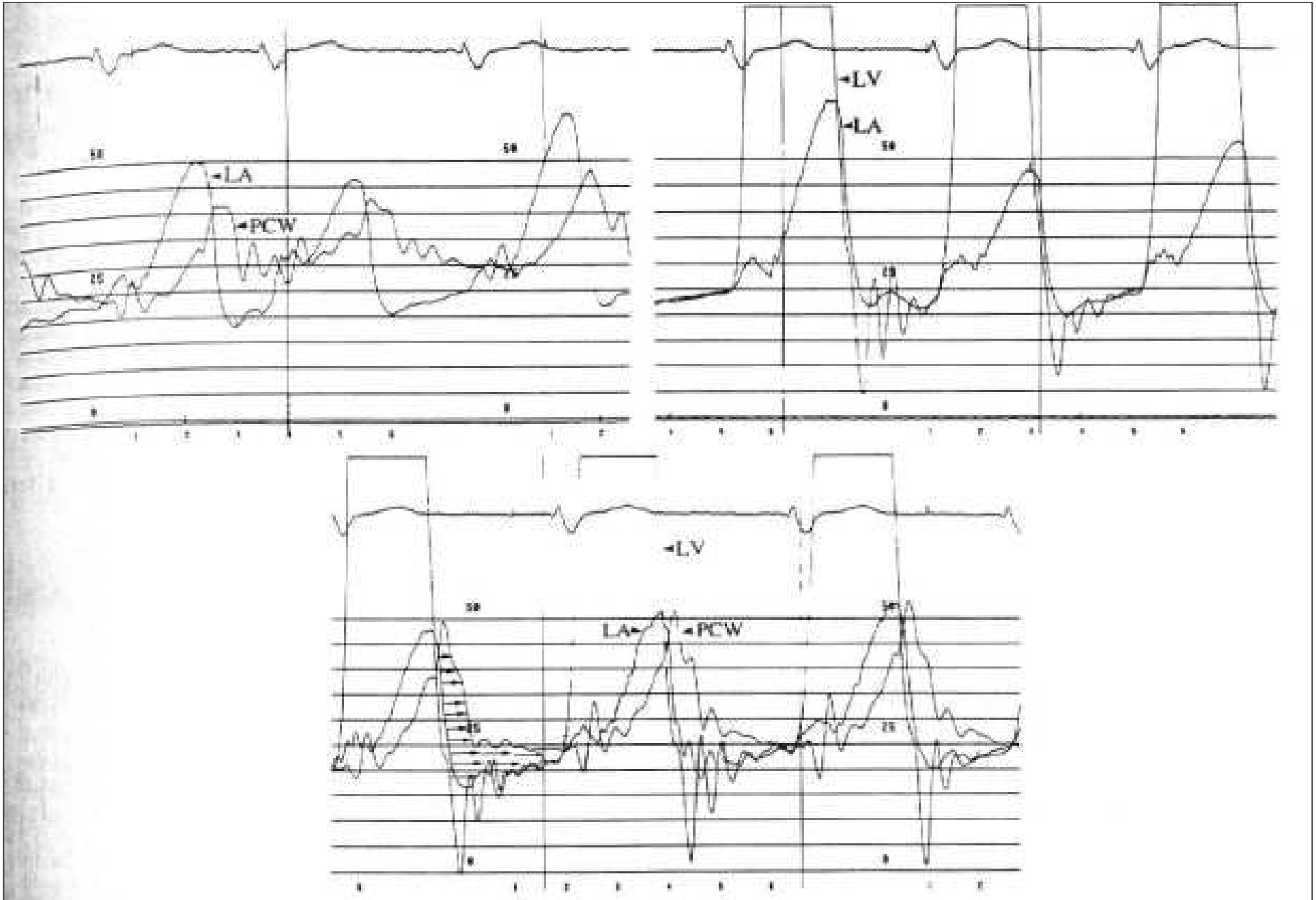


Fig. 8. Simultaneous left atrial (LA) and left ventricular (LV) pressure tracings (0–40 mm Hg scale) in a patient with new onset of fatigue. See text for details.

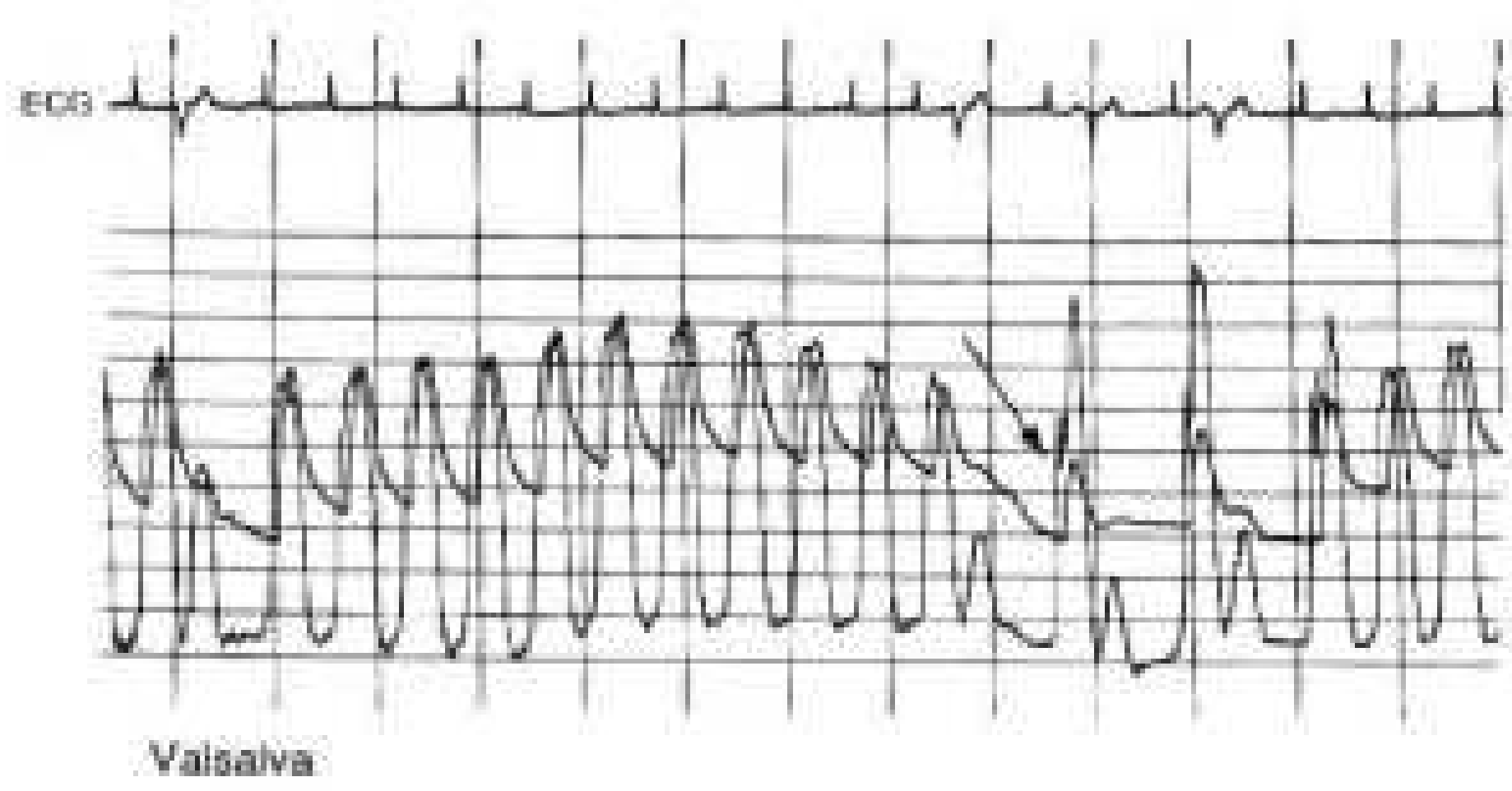


Fig. 2. Premature ventricular contraction (arrow) in a patient with hypertrophic cardiomyopathy. Note post-PVC reduction of pulse pressure.

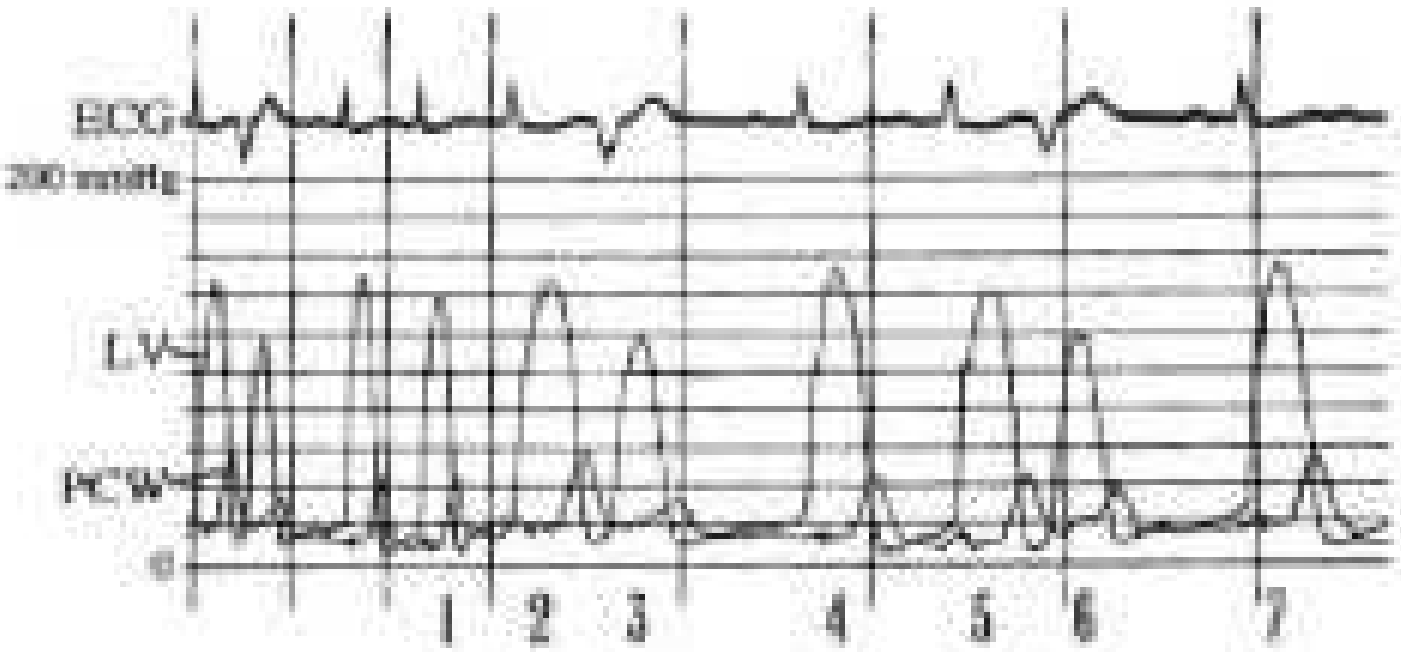


Fig. 3. Left ventricular (LV) and pulmonary capillary wedge pressure (PCW) in a patient with mitral regurgitation. Left ventricular pressures vary with the cardiac rhythm. Why is the V wave larger on beat #2? See text for details.

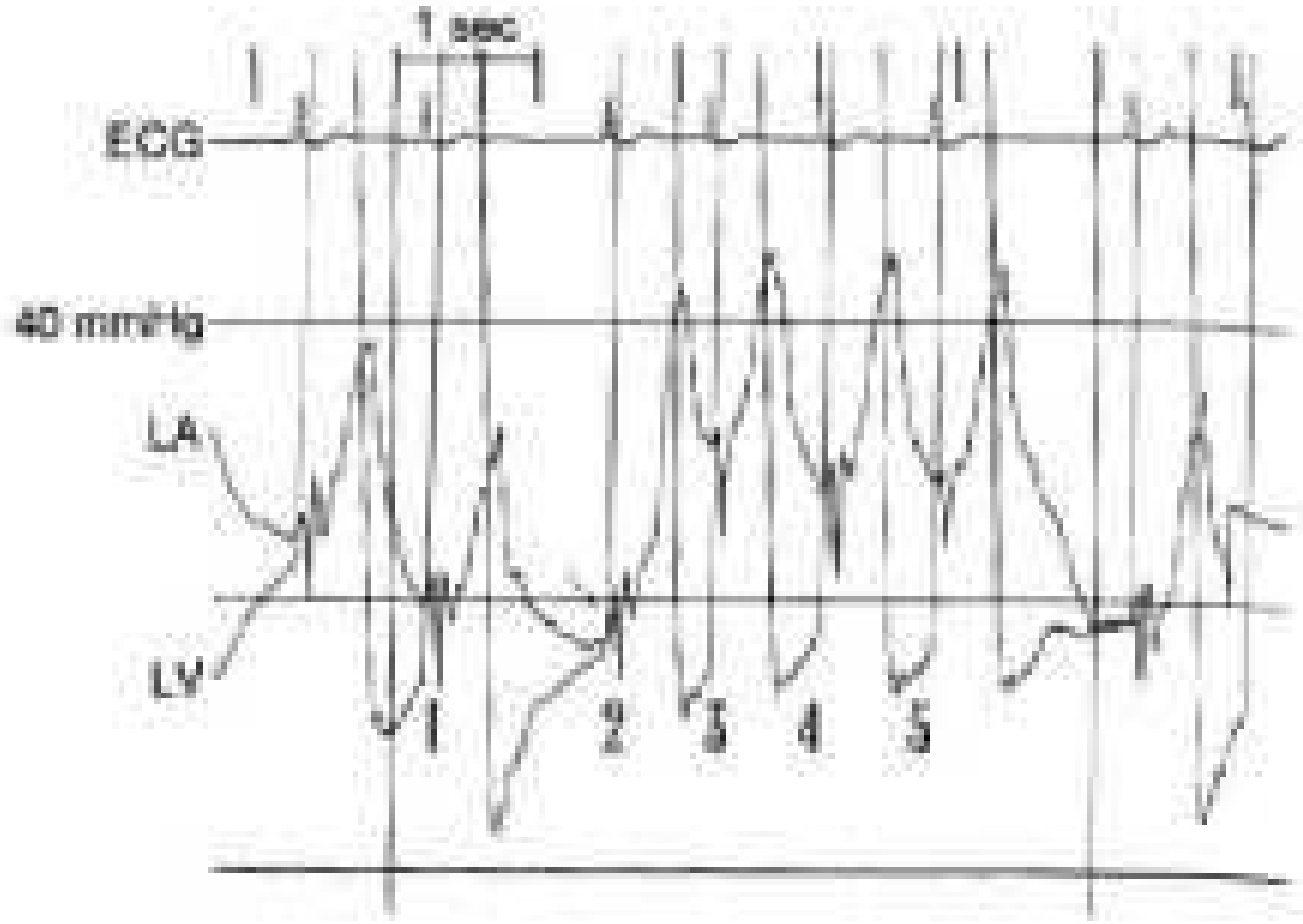


Fig. 4. Left atrial (LA) and left ventricular (LV) pressures in a patient with an irregular rhythm. Identify A, C, and V waves. See text for details.

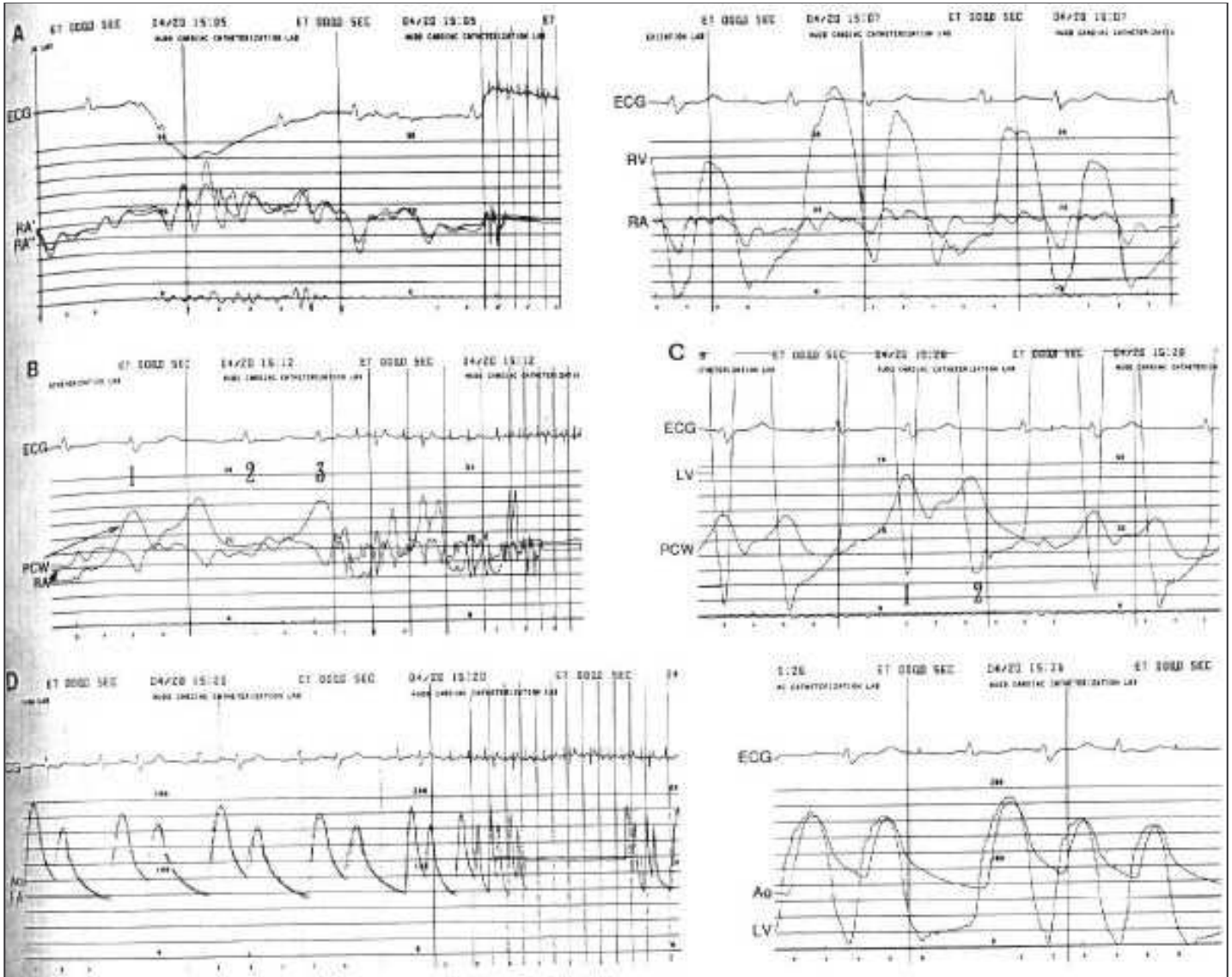


Fig. 5. A: Left panel: Simultaneous 2-catheter measurement of right (RA', RA'') pressure in preparation to assess tricuspid stenosis. Pressures match during the irregular rhythm. Right panel: Right ventricular (RV) and right atrial (RA) pressures in the same patient. Note grouped beating of right ventricular pressure. The magnitude of pressures varies due to respiratory activity. See text for details. B: Simultaneous pulmonary capillary wedge (PCW) and right atrial (RA) pressures. Note that the right atrial waveform has X and Y descents with smaller A and V waves. Does the pulmonary capillary wedge pressure waveform have large A and V waves? C: Left ventricular (LV) and pulmonary capillary wedge (PCW) pressures (0–50 mm Hg scale) demonstrating a coupled rhythm. Note the bigeminal pattern with V waves and no A waves. See text for details. D: Left: Femoral (FA) and central aortic (Ao) pressures (0–200 mm Hg scale) demonstrating coupled beats during the bigeminal rhythm. Right: The rhythm is atrial fibrillation with periods of coupled beats. LV = left ventricular pressure. See text for details.

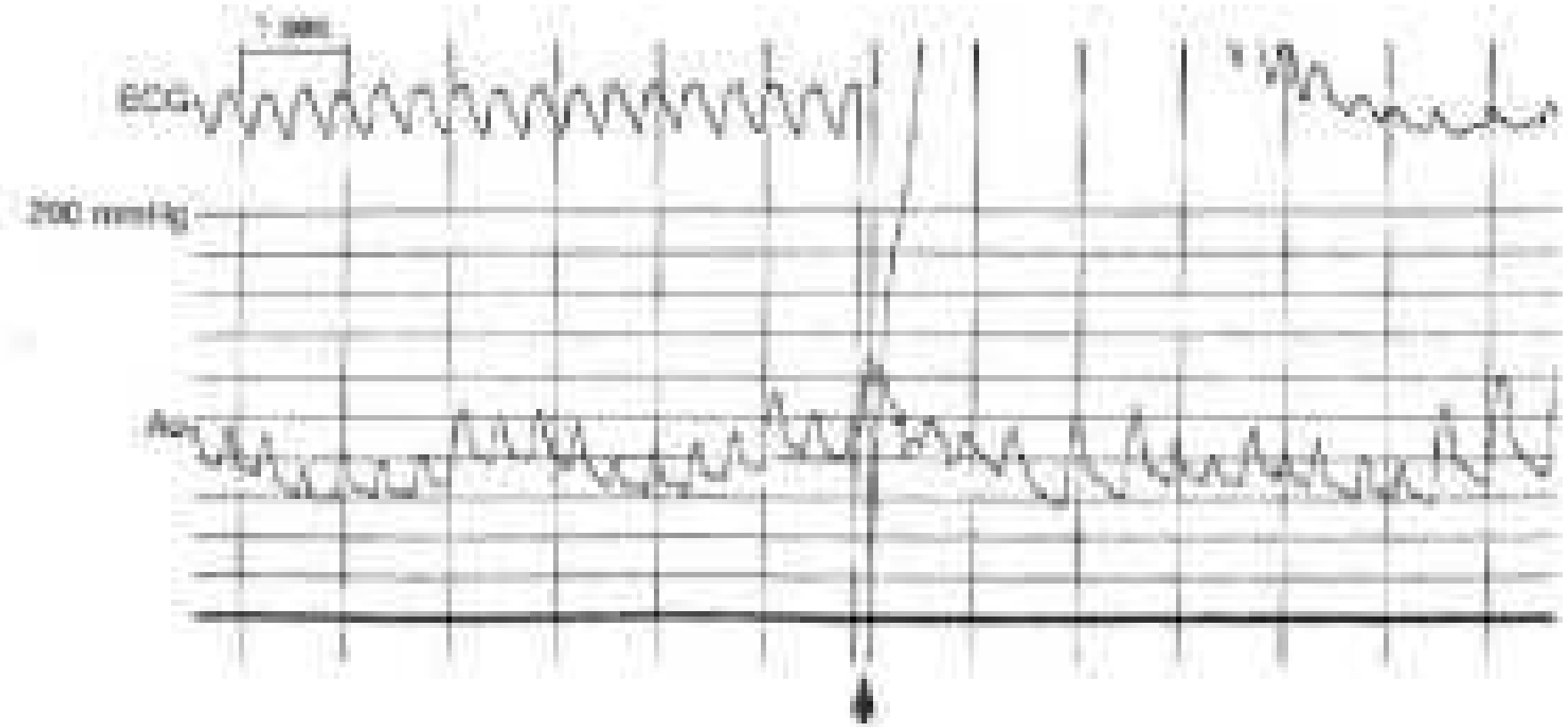


Fig. 6. Wide complex QRS tachycardia with mild arterial hypotension. Note the pattern changes at the arrow. See text for details.

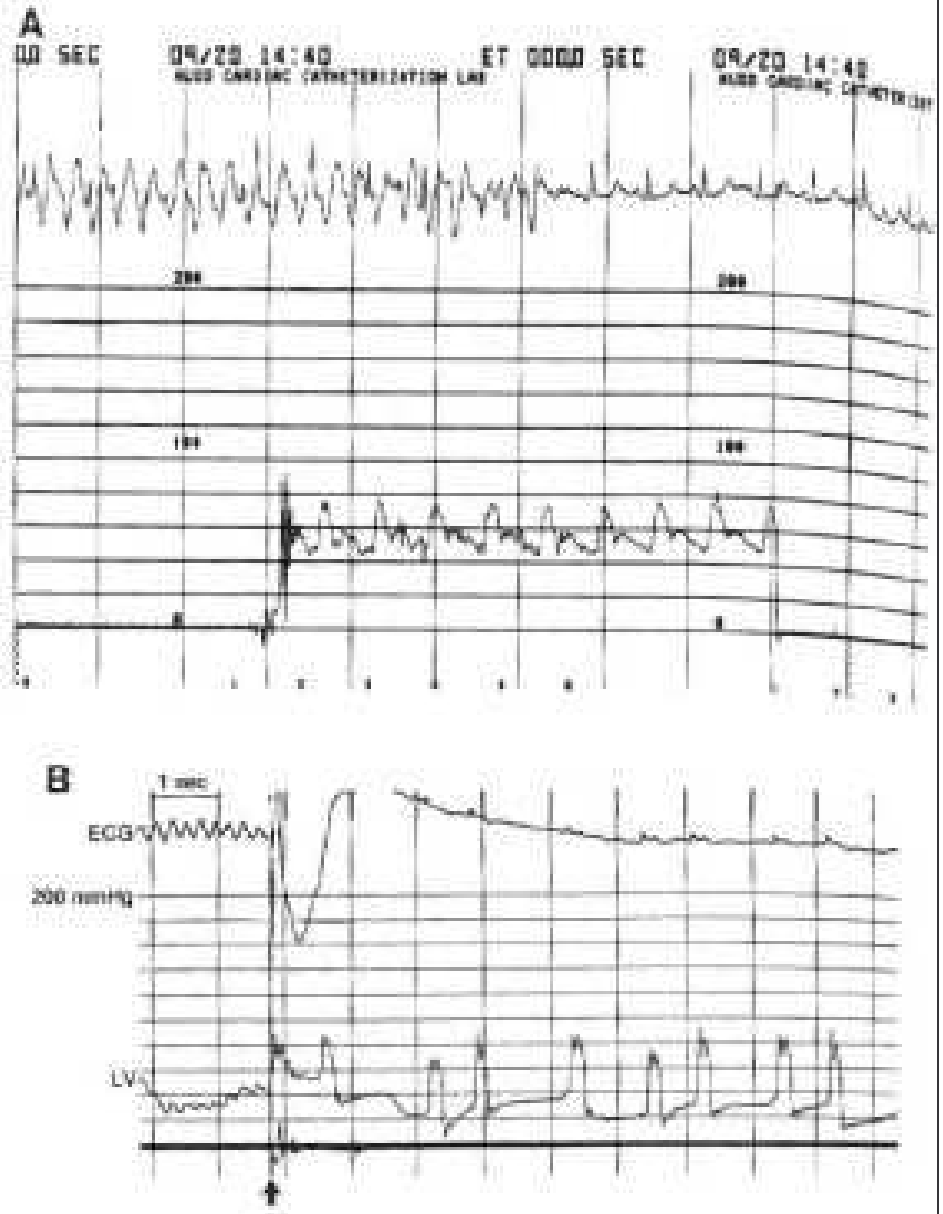


Fig. 7. A: Electrocardiogram showing ventricular fibrillation with preserved arterial pressure? B: Electrocardiogram showing ventricular fibrillation with gradual restoration of a sinus mechanism after electrocardioversion. See text for details.

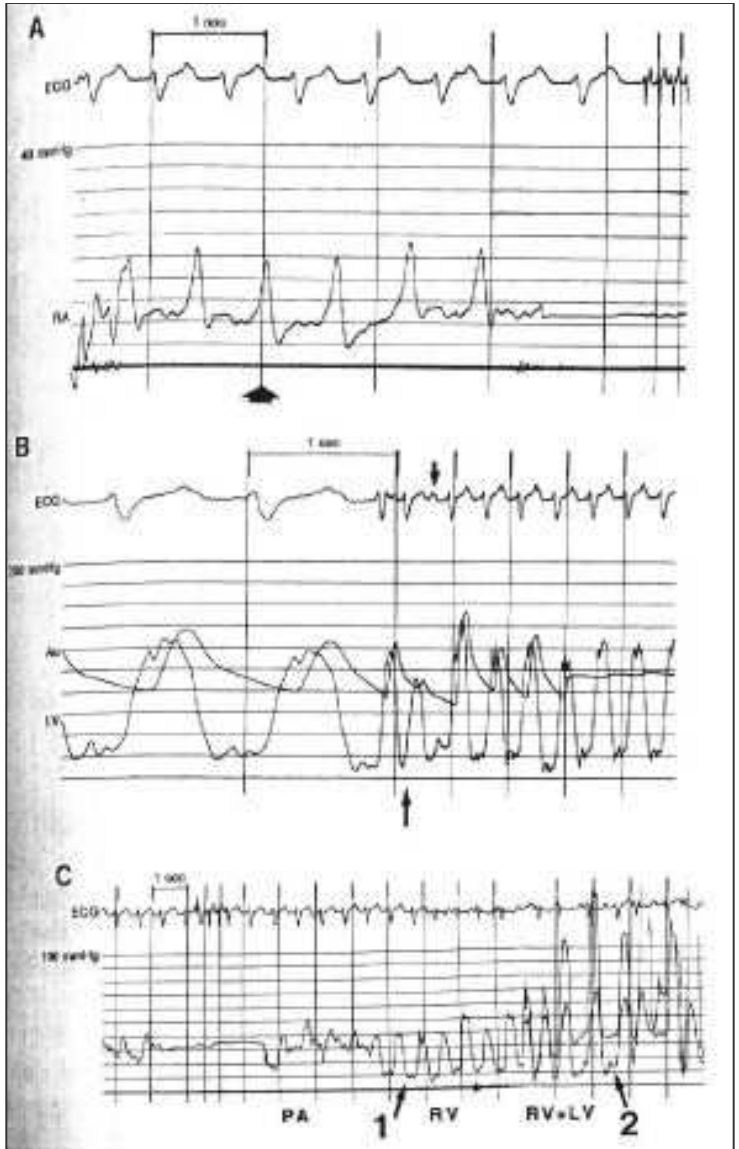


Fig. 8. A: Right atrial (RA) pressure with wide complex QRS rhythm. Are the sharp peaks (arrow) V waves? See text for details. B: Simultaneous left ventricular (LV) and aortic (Ao) pressures. A PVC (bottom arrow) produces a change in the electrocardiogram to reveal a P wave (top arrow). C: Right heart catheter pullback from pulmonary artery (PA) to right ventricle (RV) and then simultaneous left ventricle (LV) to right ventricle (0–100 mm Hg scale). Note the rhythm change after right ventricular beat #1.

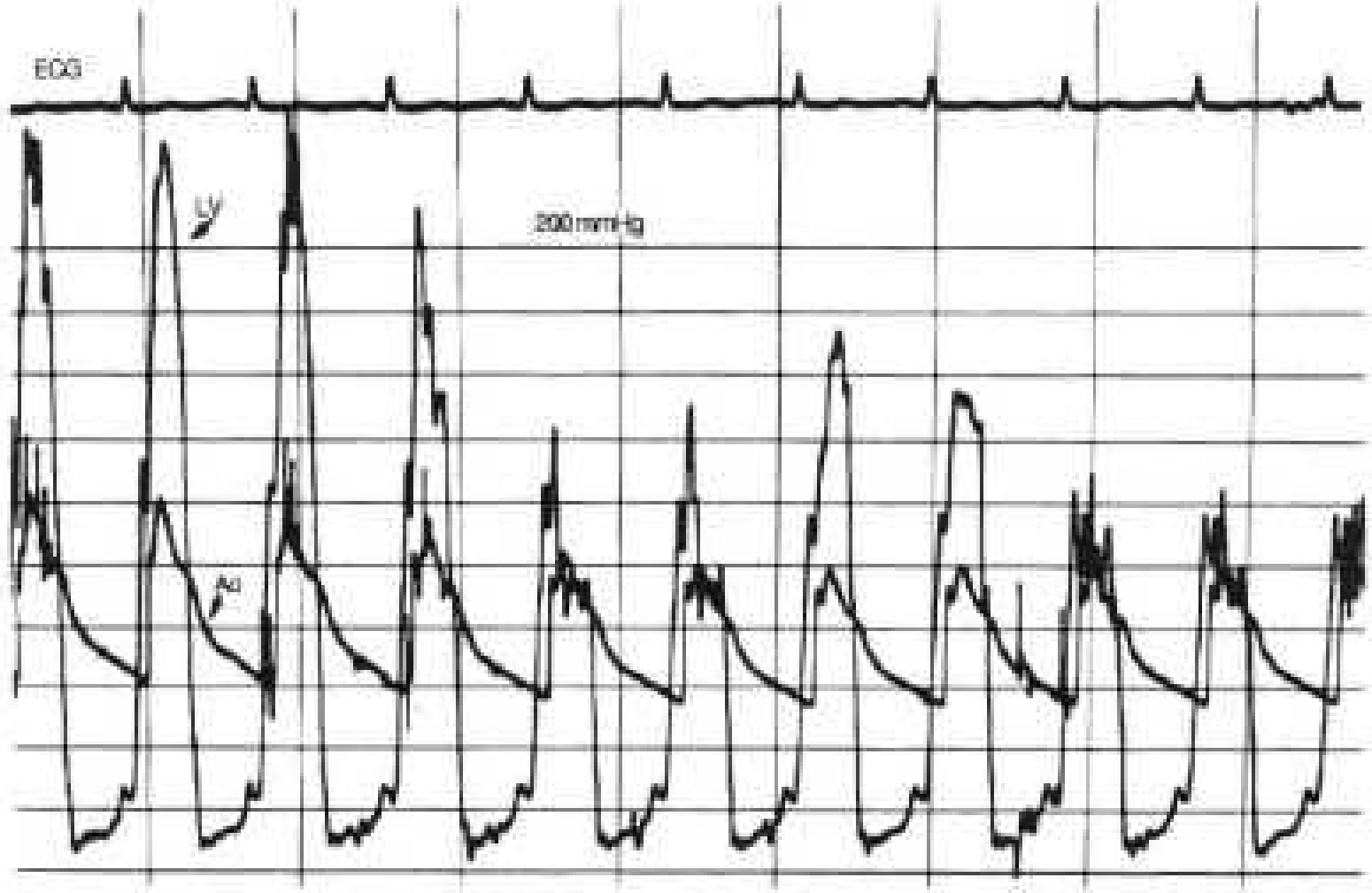


Fig. 1. Simultaneous left ventricular (LV) and aortic (Ao) pressures (200 mm Hg scale) in a patient with "disappearing aortic stenosis." See text for details.

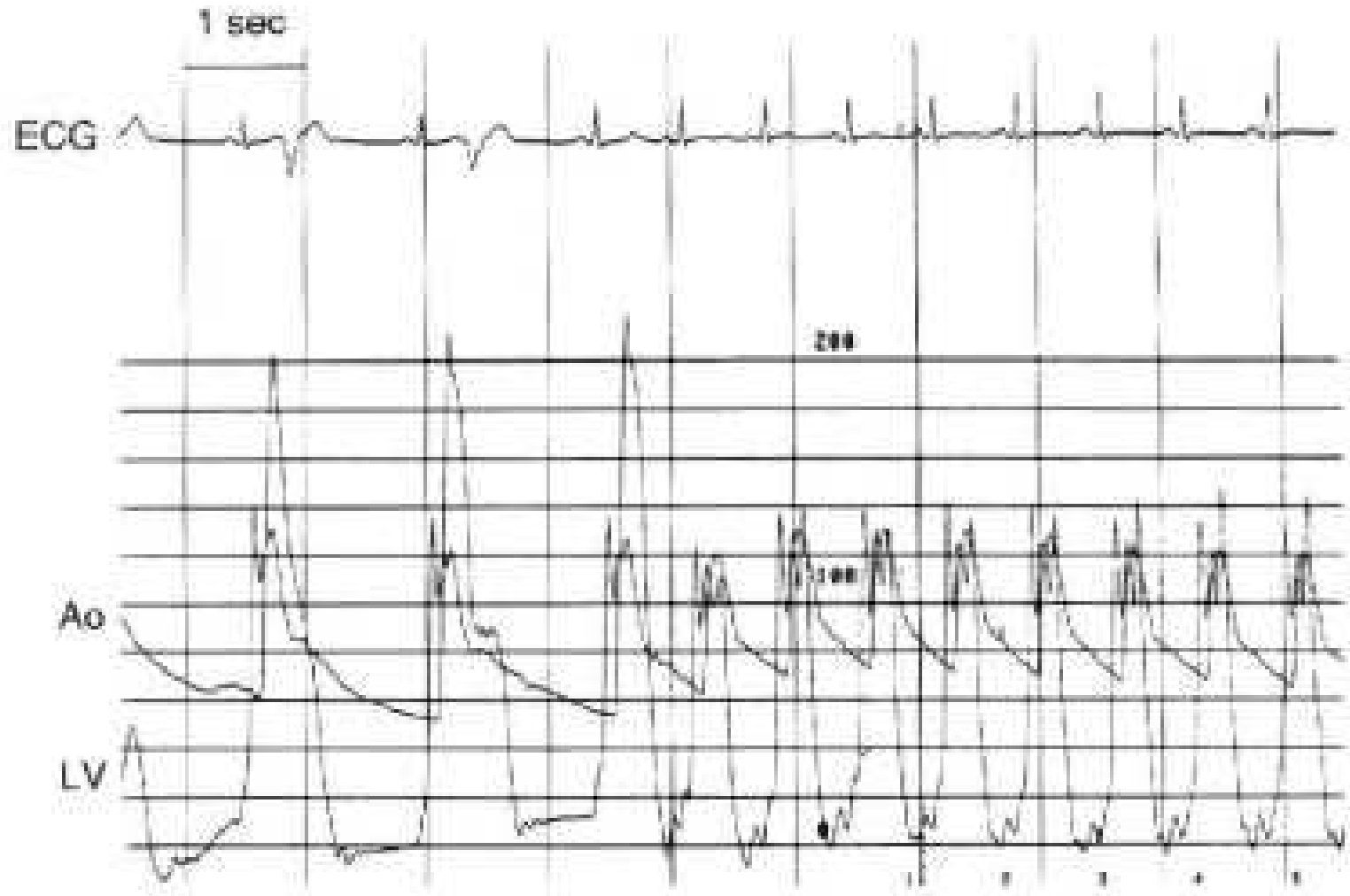


Fig. 2. Simultaneous aortic (Ao) and left ventricular (LV) pressures (200 mm Hg scale). Note the influence of premature ventricular contractions. See text for details.

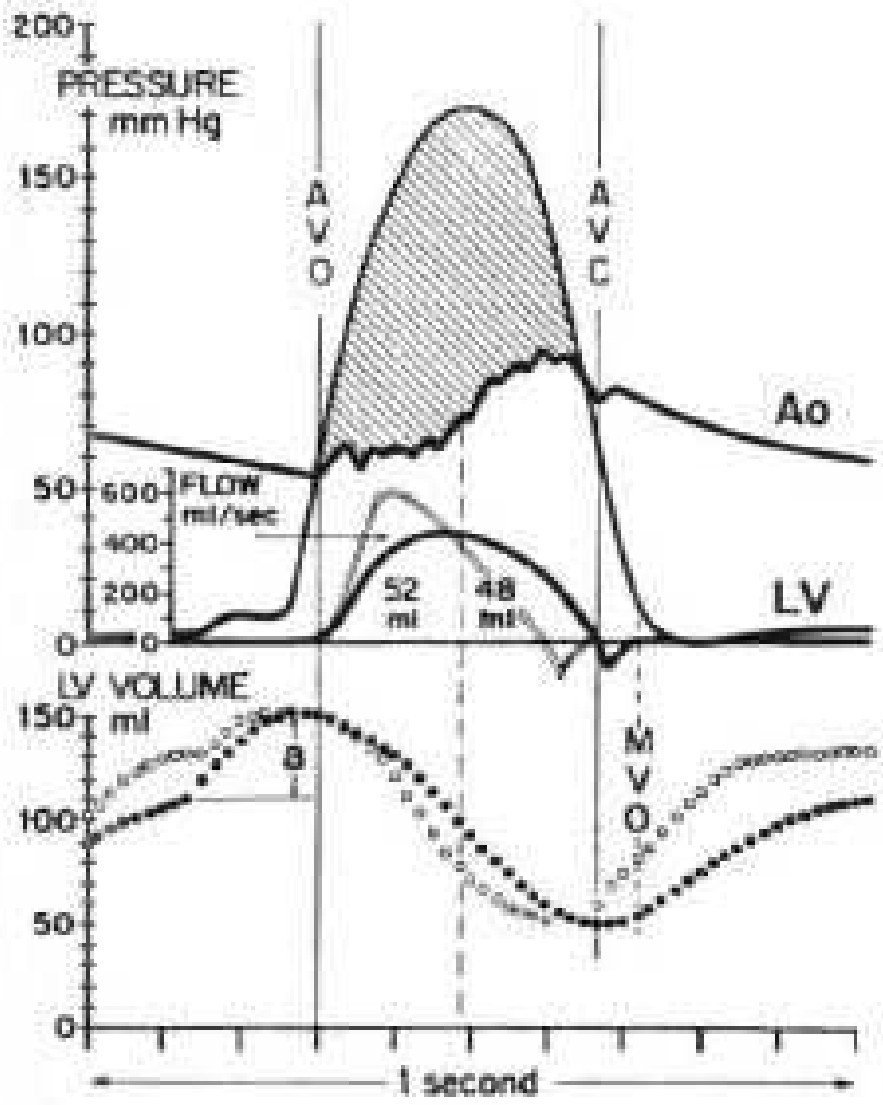


Fig. 3. Hemodynamic examples of a patient with discrete obstructive gradient with aortic stenosis. Ao = aorta; LV = left ventricle; MVO = mitral valve opening; AVC = aortic valve closure; AVO = aortic valve opening; a = atrial contribution to ventricular filling. See text for details. (Reproduced with permission from Criley [5].)

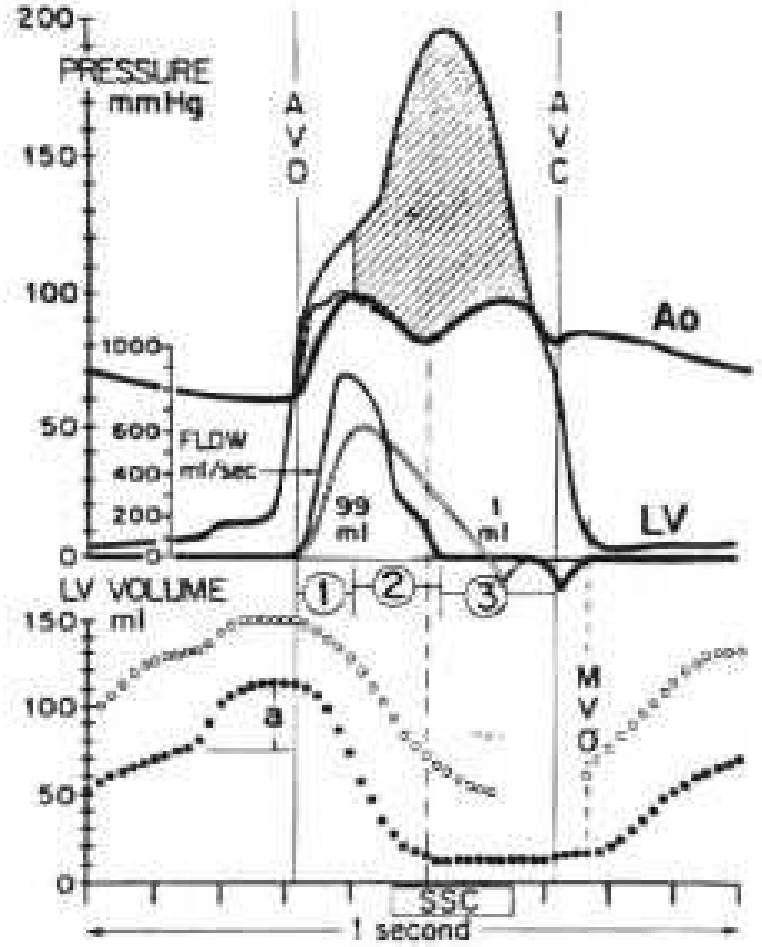


Fig. 4. Dynamic gradient in hypertrophic cardiomyopathy. Ao = aorta; LV = left ventricle; MVO = mitral valve opening; AVC = aortic valve closure; AVO = aortic valve opening; a = atrial contribution to ventricular filling; SSC = onset of septal anterior leaflet motion and septal contact. Numbers in circles 1, 2, and 3 identify the three phases of ejection from the left ventricle and discrete alterations in pressure and waveform due to motion and obstruction with intraventricular pressure gradient development. (See text for details. Reproduced with permission from Criley [5].)

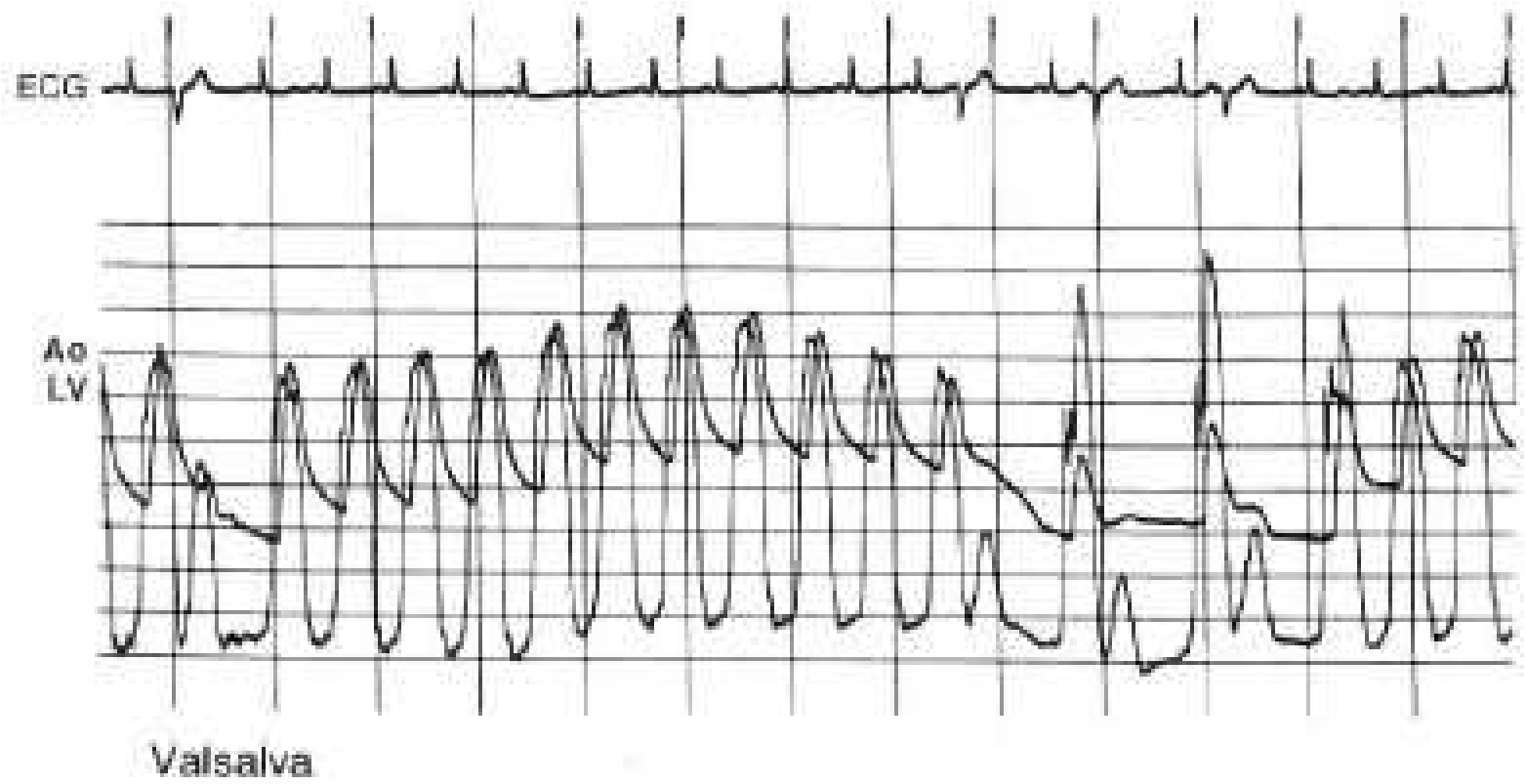


Fig. 5. Simultaneous aortic (Ao) and left ventricular (LV) pressures (200 mm Hg scale) during Valsalva maneuver. Note the influence of premature ventricular contractions before and during the maneuver. See text for details.

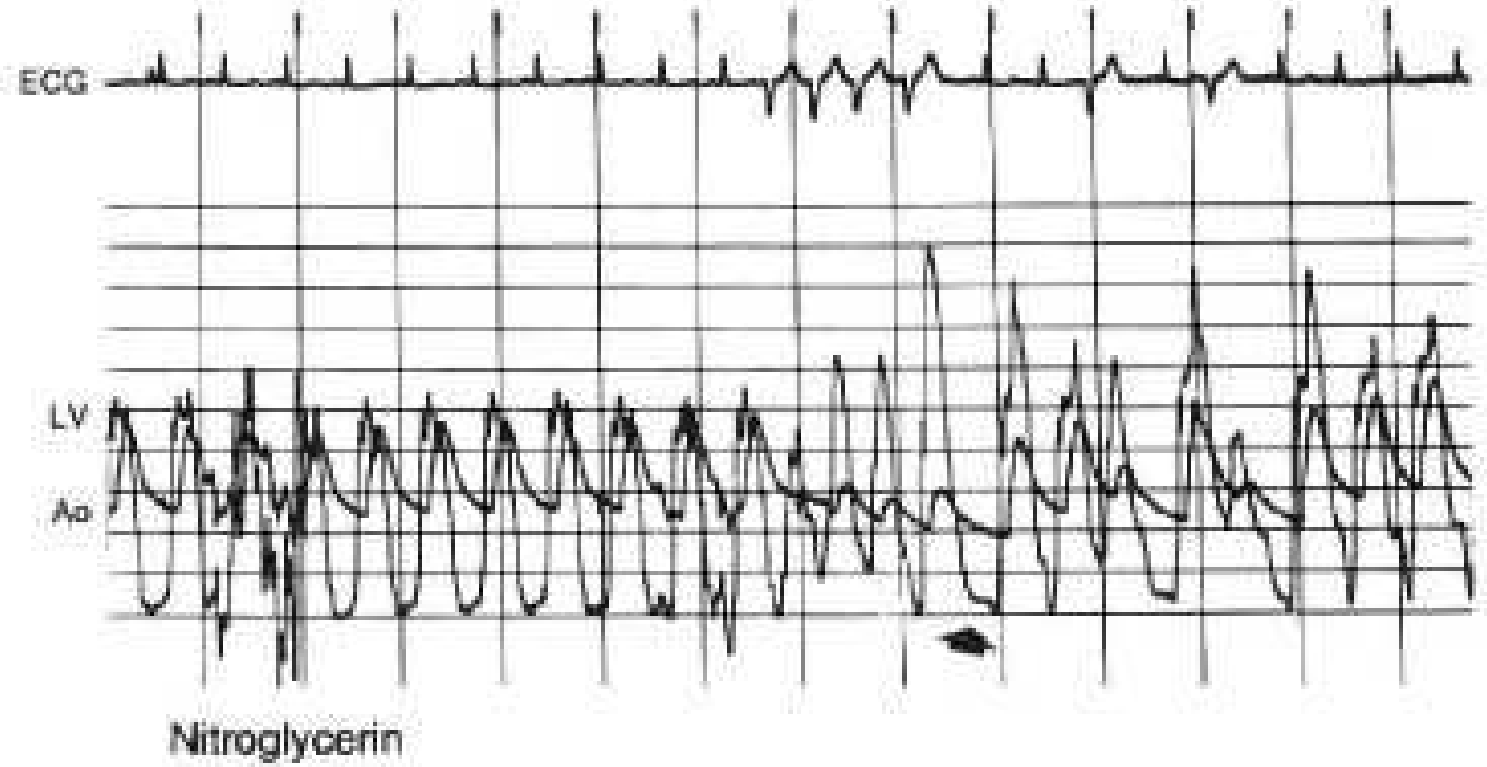


Fig. 6. Simultaneous aortic (Ao) and left ventricular (LV) pressures (200 mm Hg scale) during nitroglycerin and Valsalva maneuver (same patient as in Fig. 5). Note the influence of premature ventricular contractions. See text for details.

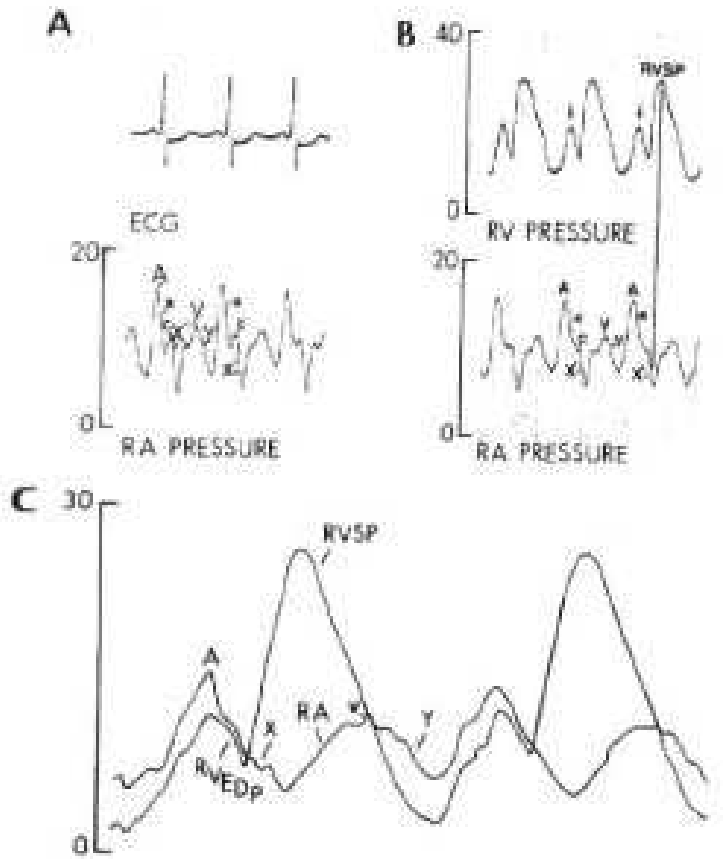


Fig. 2. Hemodynamic recordings from a patient with a W pattern. Peaks of W are formed by prominent a waves, and most prominent right atrial (RA) descent occurs just before T wave of electrocardiogram (ECG) (panel A). Simultaneous RA and right ventricular (RV) pressures (panel B) demonstrate that this prominent descent coincides with peak RV systolic pressure (RVSP) and is therefore an x' systolic descent, followed by a comparatively blunted y descent. Peak RVSP is depressed, RV relaxation is prolonged, and there is a dip and rapid rise in RV diastolic pressure. Prominent RA a waves are reflected in the right ventricle as an augmented end-diastolic pressure (EDP) rise (arrows). These wave form relations are confirmed by simultaneous superimposed RA/RV pressure recordings (panel C). Reprinted with permission from Circulation 82:359–368, 1990.

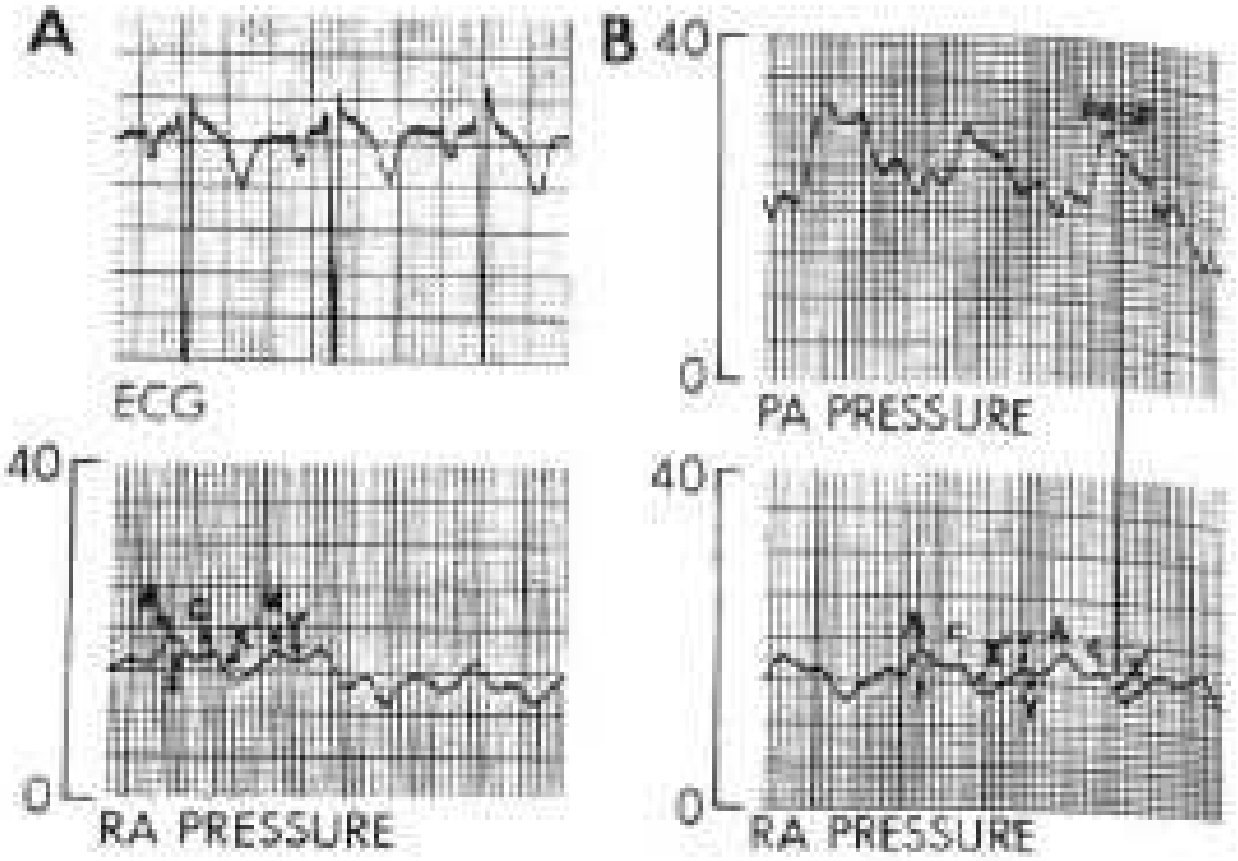


Fig. 3. Tracings of simultaneous right atrial (RA) pressure and electrocardiogram (ECG) (panel A) from a patient with an M pattern demonstrate most prominent negative deflection (x') coincident with T wave suggesting a y descent. However, timing of RA pressure with pulmonary artery systolic pressure (PASP) (panel B) demonstrates that this descent is coincident with peak PASP and therefore a systolic x' descent. Reprinted with permission from Circulation 82:359–368, 1990.

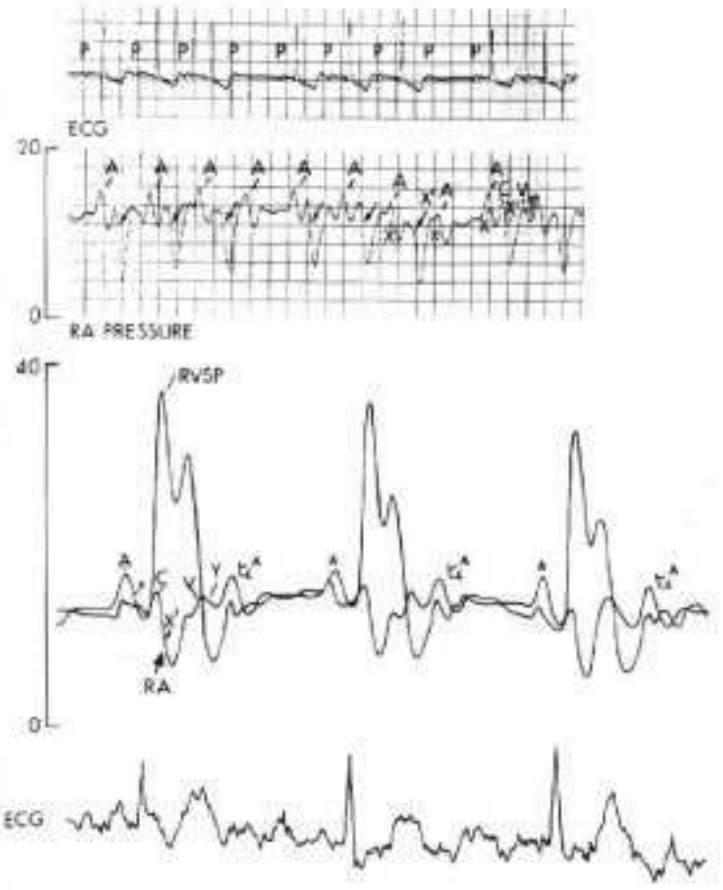


Fig. 4. **Hemodynamic recordings from a patient with a W pattern and severe low cardiac output precipitated by 2° atrioventricular block (upper panel). Recording of right atrial (RA) pressure and electrocardiogram (ECG) (upper panel) demonstrates augmented a waves and prominent x′ and blunted y descents, confirmed by simultaneous superimposed RA and right ventricular (RV) pressure recordings (lower panel). Presence of both x and x′ descents, delineated in conducted beats only (upper panel), demonstrates an additional problem with identification of components of RA pressure waveform. RV systolic pressure (RVSP) morphology is bifid, and a diastolic dip and plateau pattern is evident. Slight variation in timing of simultaneous superimposed RA/RV pressure recordings may be due to differences in maximal frequency response. RVEDP, RV end-diastolic pressure. Reprinted with permission from Circulation 82: 359–368, 1990.**

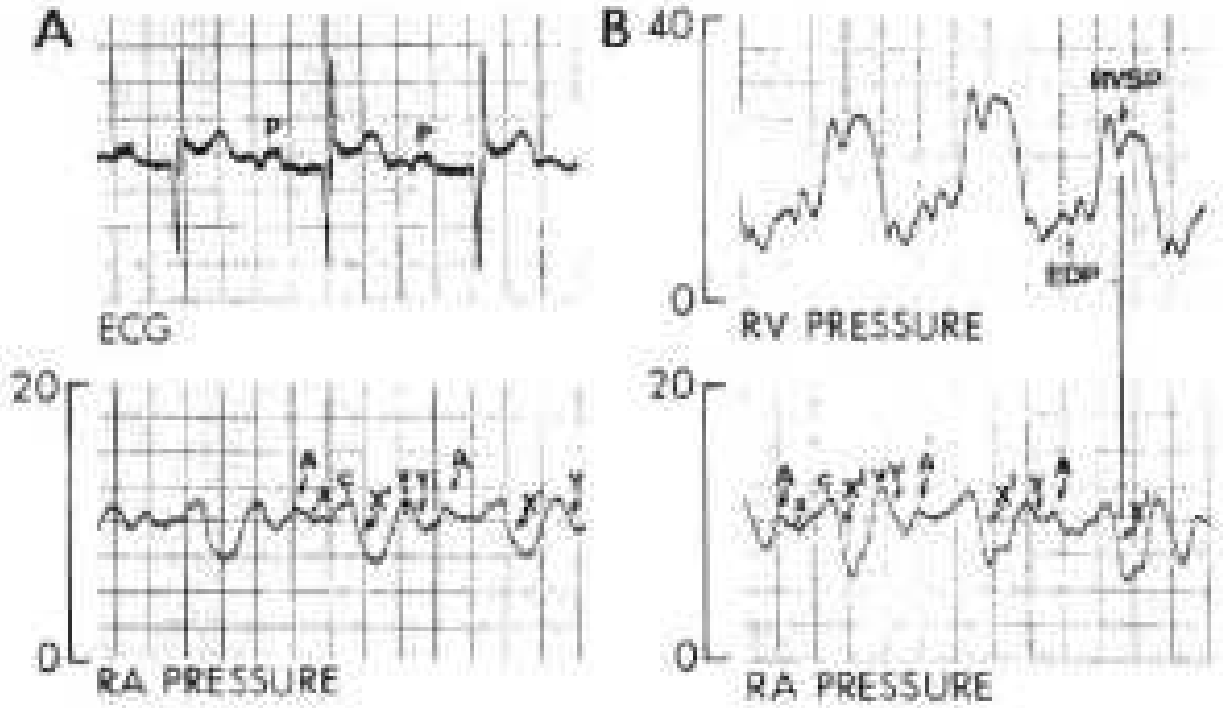


Fig. 5. Tracings of M pattern of right atrial (RA) pressure. When timed by electrocardiogram (panel A), most prominent negative deflection in right atrium is coincident with T wave, suggesting a diastolic y descent. In contrast, its relation to right ventricular (RV) pressure (panel B) demonstrates that this prominent descent coincides with peak RV systolic pressure (RVSP), indicating a systolic x′ descent, whereas diastolic y descent is blunted. M pattern comprises a depressed a wave, x descent before a small c wave, a prominent x′ descent, a small v wave, and a blunted y descent. Peak RV systolic pressure (RVSP) is depressed and bifid (arrow) with delayed relaxation and an elevated end-diastolic pressure (EDP). Reprinted with permission from Circulation 82:359–368, 1990.