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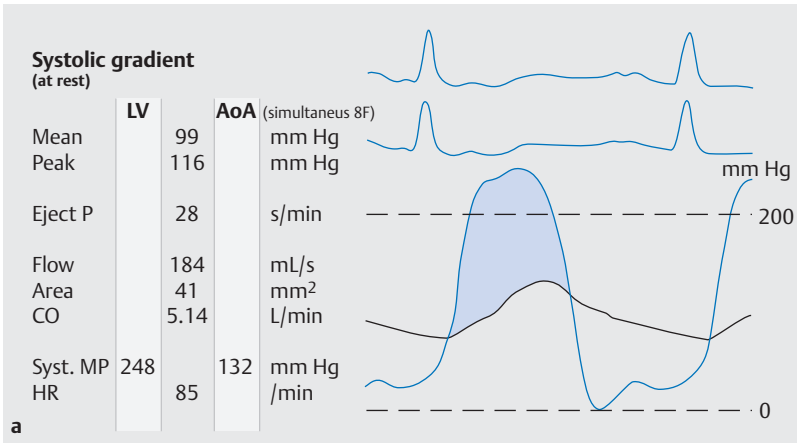

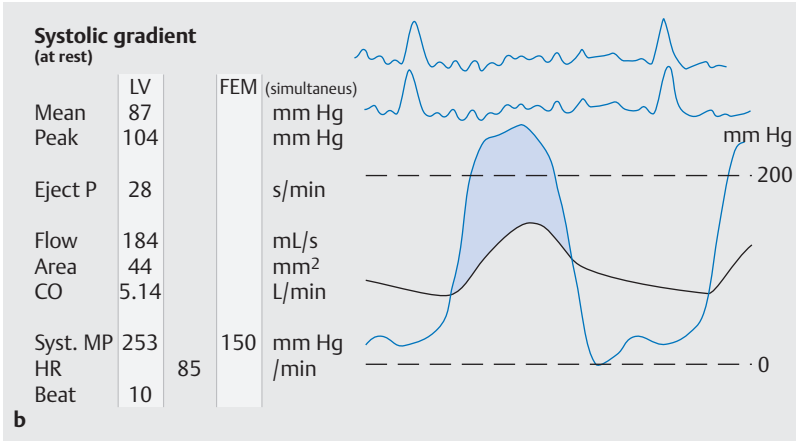

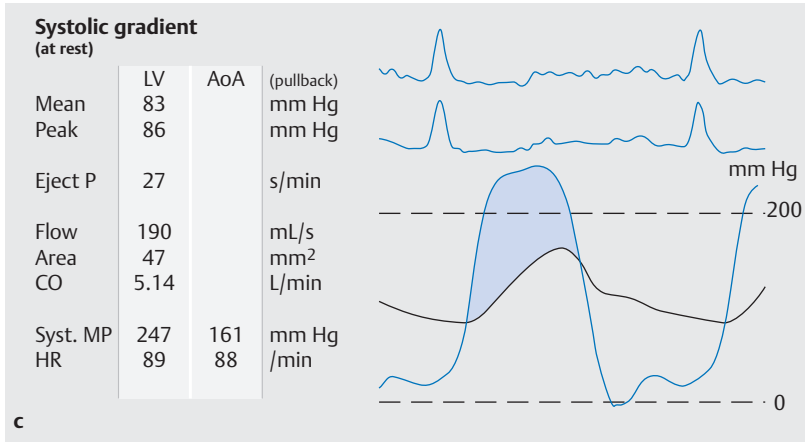

Fig. 14.9 a–c Severe aortic stenosis. Impact of different measuring sites and methods on pressure gradient and valve area.

a Simultaneous pressure measurement in the left ventricle and in the ascending aorta with a double-lumen 8F pigtail catheter; valve area 0.41 cm^2.

b Simultaneous pressure measurement in the left ventricle and in the femoral artery (via the side arm of the sheath); valve area 0.44 cm^2.

c Pressure recording during catheter pullback from LV–AoA (8F pigtail catheter); valve area 0.47 cm^2. Increase of the systolic aortic pressure by 29 mm Hg compared with the aortic pressure with simultaneous measurement in (**a**).

Eject P	systolic ejection period
Syst. MP	systolic mean pressure
HR	heart rate

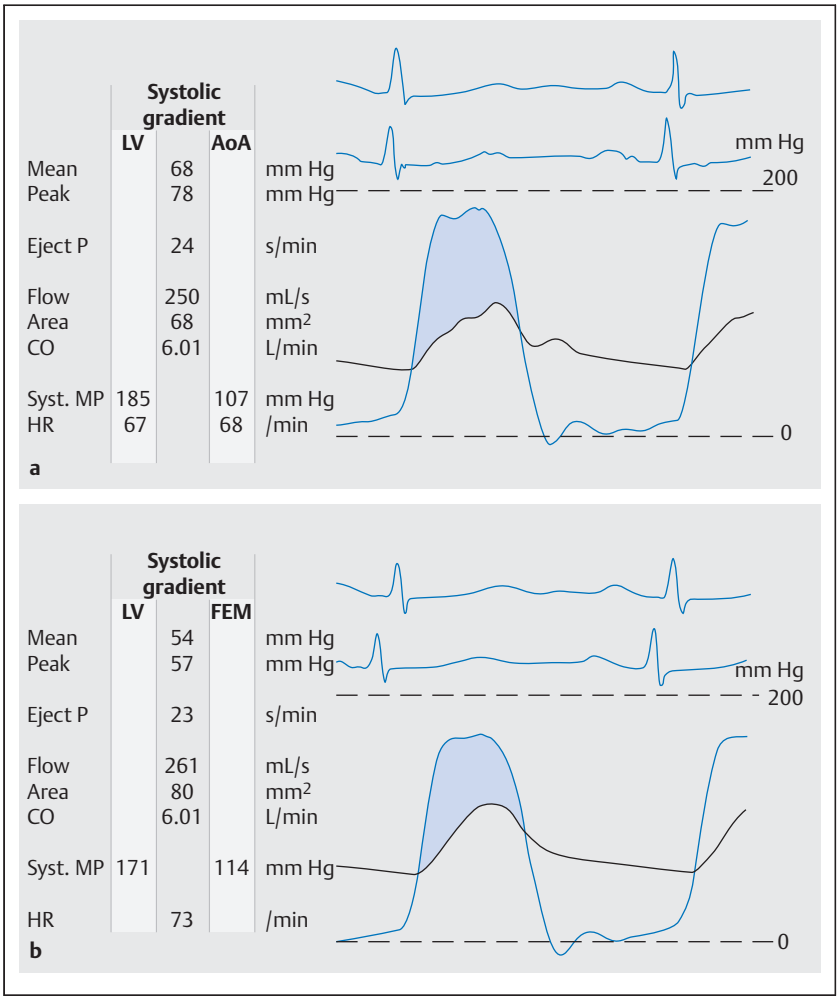

Fig. 14.10 a, b Moderate aortic stenosis. Impact of different sites and methods of measurement on pressure gradient and valve area.

a Pressure recording during catheter pullback from LV–AoA (5F pigtail catheter); valve area 0.68 cm² → **severe aortic stenosis.**

b Simultaneous pressure measurement in the left ventricle and in the femoral artery (via the side arm of the sheath); valve area 0.80 cm² → **moderate aortic stenosis.**

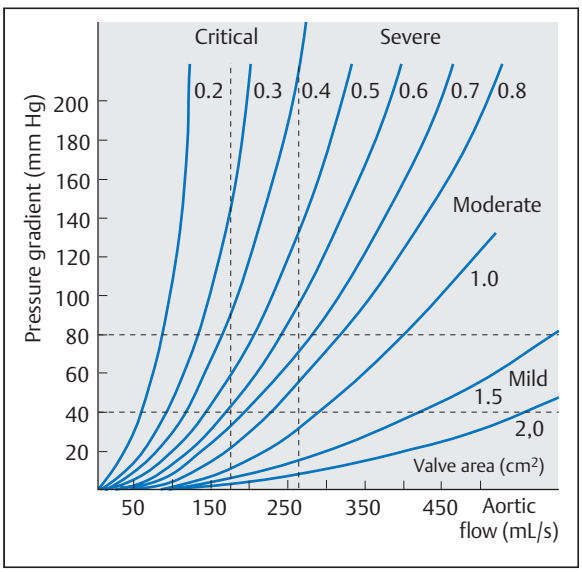

Fig. 14.11 Pressure gradient and valve area in aortic stenosis according to the Gorlin equation.

Besides clinical symptoms, calculation of the *valve area* is crucial for deciding whether an operation is indicated. Determination can be omitted only in unambiguously severe or critical aortic stenosis or in unambiguously mild aortic stenosis with normal ventricular function (**Table 14.6**).

Aortic Valve Resistance (R)

The Gorlin equation was originally developed to calculate mitral valve area and was modified using empirical data to also calculate aortic valve area. However, various studies have demonstrated that with low flow rates across the valve (< 150 mL/s) the calculated valve area is smaller than with higher flow rates and that the equation can result in incorrect values in patients with low cardiac output.

If the cardiac output is increased pharmacologically, the calculated valve area increases. This is partly attributed to the inaccuracy of the Gorlin equation at low flow rates, but also to better separation of the cusps with higher cardiac output. Due to these factors an apparently severe

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Table 14.5 Classification of severity of aortic valve stenosis

According to pressure gradient		According to valve area	
Grade I	< 40 mm Hg	**Mild**	> 1.5 cm^2
Grade II	40–80 mm Hg	**Moderate**	0.8–1.5 cm^2
Grade III	80–120 mm Hg	**Severe**	0.4–0.7 cm^2
Grade IV	> 120 mm Hg	**Critical**	< 0.4 cm^2

Table 14.6 Therapy of aortic valve stenosis depending on severity and hemodynamics

Severity/hemodynamics	Therapy
Pressure gradient < 40 mm Hg and good LV function	Medical therapy, regular follow-up, endocarditis prophylaxis
Pressure gradient > 75 mm Hg or valve area < 0.7 cm^2	Aortic valve replacement

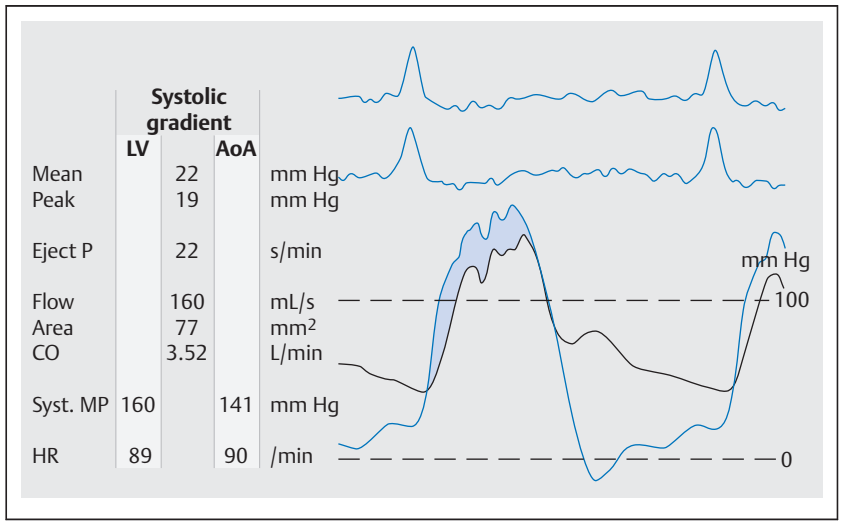

Fig. 14.12 Hemodynamics in aortic stenosis and severely impaired left ventricular function (EF = 29 %, cardiac index 2.0 [L/min]/m^2). Valve area 0.77 cm^2 → moderate to severe aortic stenosis? (Aortic valve resistance 382 dyn·s·cm^{-5}; thus, severe aortic stenosis is likely.)

aortic stenosis may turn out to be only moderate when cardiac output is increased.

These observations have practical relevance when a decision regarding surgical aortic valve replacement has to be made for patients with the following findings:

- Impaired ventricular function (CO < 4.5 L/min)
- Borderline aortic valve area (0.7–0.8 cm^2)
- Low pressure gradient (25–35 mm Hg)

These patients have a high surgical risk. Prior to surgery it should be unambiguous that there is indeed severe aortic stenosis rather than a mild to moderate stenosis with only apparently smaller valve area due to method-inherent miscalculation (**Fig. 14.12**).

To make differentiation more certain, **valve resistance** was introduced to complement the Gorlin equation. Valve resistance (R) is calculated as a simple ratio of pressure gradient across the valve and blood flow across the valve with the following equation:

$$R = \frac{HR \times \Delta P_m \times ET \times 1.33}{CO}$$

where:

R = valve resistance in dyn·s·cm^{-5}

HR = heart rate in beats/min

ΔP_m = mean pressure gradient in mm Hg

ET = ejection time in seconds

CO = cardiac output in L/min

1.33 = conversion factor

A valve resistance ≤ 250 dyn·s·cm^{-5} argues against a severe aortic valve stenosis.

In patients with pertinent findings some authors recommend the careful administration of dobutamine in the catheterization laboratory: patients with severe stenosis develop a marked increase in the pressure gradient with increased stroke volume, whereas in patients with only mild stenosis at most a small increase or even a decrease in the pressure gradient is observed.

However, other factors also need to be considered in patient management. For example, a valve area of 1 cm^2 in a large, active man can already represent severe aortic stenosis, whereas 0.75 cm^2 can still be sufficient for a small, inactive person.

Particularly difficult is the evaluation of valve area in patients with *borderline aortic valve stenosis* and *severe LV dysfunction with low stroke volume.* To differentiate the true anatomical valve area from a functionally decreased valve opening, hemodynamic examinations in the cardiac catheterization laboratory using low-dose dobutamine (corresponding to the doses used in stress echocardiography) are useful. Alternatively, this hemodynamic provocation can also be done echocardiographically by very experienced investigators, with continuous measurement of stroke volume and pressure gradient while observing LV function. If dobutamine infusion increases stroke volume and valve area and reduces valve resistance, then it is likely that the initial calculation overestimated the severity.

! If the patient is not a candidate for surgery due to a porcelain aorta or because of substantial comorbidities, the cardiologist and cardiac surgeon should together "heart team" evaluate whether alternative methodologies can be employed if there is a clear clinical indication. Percutaneous or transapical aortic valve replacement is a treatment option for high–risk patients. Compared with earlier reports, aortic valvuloplasty as a purely symptomatic treatment has gained in significance after recent reconsideration.

Aortic Regurgitation

Anatomical and Pathophysiological Basics

The possible causes of aortic regurgitation (AR) are manifold (**Table 14.7**). The valvular incompetence develops either as a consequence of direct pathological–anatomical changes at the cusps (shrinking, perforation, vegetations) or secondarily as a result of diseases of the aortic root and the ascending aorta.

Regarding etiology and especially regarding hemodynamics and patient management, acute and chronic aortic regurgitation are differentiated. Acute aortic regurgitation is in most cases due to bacterial endocarditis or aortic dissection. Chronic aortic regurgitation is predominantly a late complication of rheumatic fever. Aortic regurgitation causes volume overload of the left ventricle and the aorta. Due to the inability of the cusps to close, there is diastolic backflow of blood from the aorta into the left ventricle. This regurgitant volume depends on the following factors:

- Size of the incompetent area
- Diastolic pressure difference between aorta and left ventricle
- Duration of diastole (heart rate)

Table 14.7 Causes of aortic regurgitation

Acute aortic regurgitation
– Bacterial endocarditis – Aortic dissection – Trauma – Acute prosthetic dysfunction post aortic valve replacement
Chronic aortic regurgitation
– Rheumatic fever – Myxomatous degeneration of the aortic valve – Ankylosing spondylitis – Syphilitic aortitis – Reiter syndrome – Rheumatoid arthritis – Bicuspid aortic valve – Ventricular septal defect – Sinus of Valsalva aneurysm – Distension of the valvular annulus in connective tissue disease (Marfan syndrome, Ehlers–Danlos syndrome, Hurler syndrome, osteogenesis imperfecta) – Arterial hypertension – Appetite suppressants

The *total stroke volume* of the left ventricle is the sum of the regurgitant volume and the blood volume that flows into the periphery (the latter is the effective stroke volume). The volume overload leads to compensatory dilatation and eccentric hypertrophy of the left ventricle and to dilatation of the ascending aorta.

A normal effective stroke volume and thus a normal blood supply is maintained in the compensated stage of *mild aortic regurgitation* by increased systolic emptying of the left ventricle with correspondingly increased ejection fraction and total stroke volume.

In *compensated moderate to severe aortic regurgitation* a normal effective stroke volume is achieved by a sometimes considerably increased end-diastolic volume, without impairment of systolic function. The stage of *symptomatic left ventricular failure* as a consequence of aortic regurgitation is characterized by a reduced effective stroke volume with a reduced ejection fraction and enlarged left ventricle. Left ventricular end-diastolic pressure, left atrial pressure, pulmonary capillary wedge

pressure, and the other pressures in the pulmonary circulation are increased.

Specific Hemodynamics

Acute aortic regurgitation. Acute aortic regurgitation causes an acute and in many cases severe *volume overload* of the previously normal-sized and normally contracting left ventricle. The above-mentioned time-dependent compensatory mechanisms to increase cardiac output are not available or are available only to a limited extent. The acute volume load causes a moderate dilatation of the left ventricle with reduction of the effective stroke volume and cardiac output with usually unchanged ejection fraction. In contrast to chronic aortic regurgitation there is frequently tachycardia and the pulse amplitude is small.

With impaired compliance of the left ventricle there is a rapid rise in diastolic ventricular pressure with a highly increased end-diastolic pressure, which not infrequently increases to the same level as the diastolic aortic pressure. The rapid filling of the left ventricle results in premature closure of the mitral valve, so that both the left atrial pressure and the pulmonary capillary wedge pressure are markedly below the diastolic ventricular pressure. If with increasing regurgitation the mean diastolic pressure in the left ventricle also rises, this will necessarily also lead to a corresponding pressure increase in the pulmonary circulation.

Chronic aortic regurgitation. The characteristic hemodynamics of chronic aortic regurgitation are determined by increased systolic emptying of the left ventricle with a compensatory increase in end-diastolic volume and increased left ventricular compliance. Even with large regurgitant volumes, the LVEDP can remain in the normal range for years. There is no considerable difference between LVEDP and pulmonary capillary wedge pressure. Effective stroke volume and cardiac output are in the normal range. Left ventricular end-diastolic volume is markedly increased, whereas left ventricular end-systolic volume is usually moderately increased with normal ejection fraction. Corresponding to the increased total stroke volume, the systolic aortic pressure is increased. However, during diastole mild to moderately reduced end-diastolic pressure values are measured, depending on the severity of regurgitation. These hemodynamic characteristics of compensated aortic regurgitation change as expected when the chronic volume overload results in manifest left ventricular failure.

Indication

Acute aortic regurgitation. Acute aortic regurgitation usually develops as a result of bacterial endocarditis or aortic dissection with involvement of the aortic root. In most cases clinical findings and echocardiography are sufficient to diagnose aortic regurgitation and to evaluate its severity. Hemodynamically unstable patients with acute aortic regurgitation resulting from bacterial endocarditis need emergency surgery, but also hemodynamically stable patients require rapid surgery, as in many cases the valve destruction progresses despite antibiotic treatment. Furthermore, there is a high risk of septic emboli.

! Presurgical cardiac catheterization to confirm the diagnosis is not indicated. Retrograde catheterization of the left ventricle is actually contraindicated, as there is a high risk of embolization of bacterial vegetations when the catheter crosses the valve.

It should always be discussed with the cardiac surgeon whether a preoperative coronary angiogram is required to assess for coronary artery disease. Usually it is not done in younger patients (< 40 years) without risk factors for or a history suggestive of coronary artery disease. Emergency surgery is also indicated in acute aortic regurgitation as a result of aortic dissection. Therefore, preoperative cardiac catheterization with aortography to evaluate both the aortic regurgitation and the extent of the dissection is indicated only in patients with high risk of coronary artery disease. Aortic regurgitation and the extent of dissection can also be well evaluated with noninvasive imaging modalities (transthoracic and transesophageal echocardiography, cardiac MRI, cardiac CT).

Due to the high prevalence of hypertension among patients with aortic dissection, a coronary angiogram is required when a prosthetic replacement of the ascending aorta with reimplantation of the coronary arteries is planned.

Chronic aortic regurgitation. A prominent issue in chronic aortic regurgitation is to determine the right time for cardiac catheterization and surgical therapy. In most cases diagnosis and classification of severity can be achieved by (color) Doppler echocardiography with additional right heart catheterization under stress. If patients are asymptomatic and left ventricular function is not impaired, the long-term prognosis with moderate as well as severe aortic regurgitation is favorable and cannot be improved by valve replacement. Thus, it may be possible to treat these patients medically for a long time.

Left heart catheterization is indicated in

- All patients with symptomatic aortic regurgitation
- Asymptomatic patients with impaired ventricular function
- Asymptomatic patients with unfavorable prognostic indicators (diastolic blood pressure 40 mm Hg, systolic blood pressure 140 mm Hg, left ventricular dilatation, electrocardiographic signs of hypertrophy)
- Patients with concomitant aortic stenosis or concomitant mitral valve defect

- Patients in whom the severity of regurgitation or of left ventricular function cannot be adequately evaluated by noninvasive means

Goals

- Determination of the hemodynamic severity of the regurgitation (calculation of regurgitant fraction, angiographic classification of severity)
- Evaluation of ventricular function at rest, measurement of end-systolic and end-diastolic volumes
- Evaluate for concomitant valvular defects
- Evaluate for concomitant coronary artery disease

Procedure

- Arterial and venous puncture (4F to 6F)
- Catheterization of the left ventricle with a pigtail catheter
- Right heart catheterization with placement of the catheter in the wedge position (PCW)
- Simultaneous pressure recording PCW/LV
- Determination of cardiac output
- Ventriculography
- Calibration and measurement of left ventricular volume (sphere)
- Right heart catheter pullback with pressure recording
- Left heart catheter pullback with pressure recording
- Supravalvular aortography
- Coronary angiography

Special Characteristics

Left Heart Catheterization and Aortography

Catheterization of the left ventricle in the case of pure aortic regurgitation is in most cases accomplished without problems. Importantly, an *adjusted (larger) volume of contrast* has to be administered for left ventriculography and aortography to achieve sufficient opacification of the usually markedly dilated left ventricle and to ensure a reliable quantification of the aortic regurgitation (contrast requirement for aortography ≥ 40 mL; injection rate ≥ 14 mL/s).

Coronary Angiography

High flow velocities in the coronary arteries can be expected in the volume-overloaded left ventricle, and therefore *coronary catheters with larger lumens* (≥ 5F) are recommended. As in most cases the aortic root and the ascending aorta are markedly dilated, a Judkins catheter with a larger curve (5 or 6 cm) should be selected from the outset. At times engagement of the coronary arteries is easier with the Amplatz technique.

With *acute aortic regurgitation due to aortic dissection* both advancing the catheter and engaging the coronary ostia must be done very carefully and with little manipulation; the Sones technique is not suitable.

Particular diligence is also required in *bacterial endocarditis*. Catheter or guidewire manipulations in the area of the valve are absolutely to be avoided due to the high risk of bacterial emboli. For the same reason, retrograde catheterization of the ventricle is contraindicated.

Findings on Cardiac Catheterization

Typical findings in severe aortic regurgitation are summarized in **Table 14.8**.

Pressure Waves

Acute aortic regurgitation. In acute aortic regurgitation there is a steep increase in LVEDP during diastole with a simultaneous decrease in diastolic aortic pressure. In severe regurgitation this can lead to pressure equalization of the LVEDP and diastolic aortic pressure. A potential consequence is a critical reduction in the coronary perfusion pressure with typical angina as the guiding symptom and/or new ECG changes without demonstration of significant coronary stenosis during coronary angiography.

Most of the time there is no *a*-wave in the ventricular pressure wave. The systolic ventricular and aortic pressure waves usually have a single peak (**Fig. 14.13**). Due to the premature closure of the mitral valve the pulmonary capillary wedge pressure is clearly below the LVEDP.

Chronic aortic regurgitation. Depending on the severity of chronic regurgitation, there can also be an increase in LVEDP that is frequently associated with an elevation of the mean diastolic pressure and thus also increased pul-

Table 14.8 Typical findings in severe aortic regurgitation (AR)

Aortic pressure	Diastolic pressure ↓ In severe acute AR, there is pressure equalization between end-diastolic aortic pressure and LVEDP
LVEDP	Increased In chronic AR, rarely there is pressure equalization with aortic diastolic pressure
PCW pressure	In chronic AR: PCW = LVEDP In acute AR: PCW < LVEDP
Aortography	Diastolic flow of contrast into the LV
Left ventriculogram	End-diastolic volume ↑ End-systolic volume ↑ Total stroke volume ↑ Ejection fraction normal

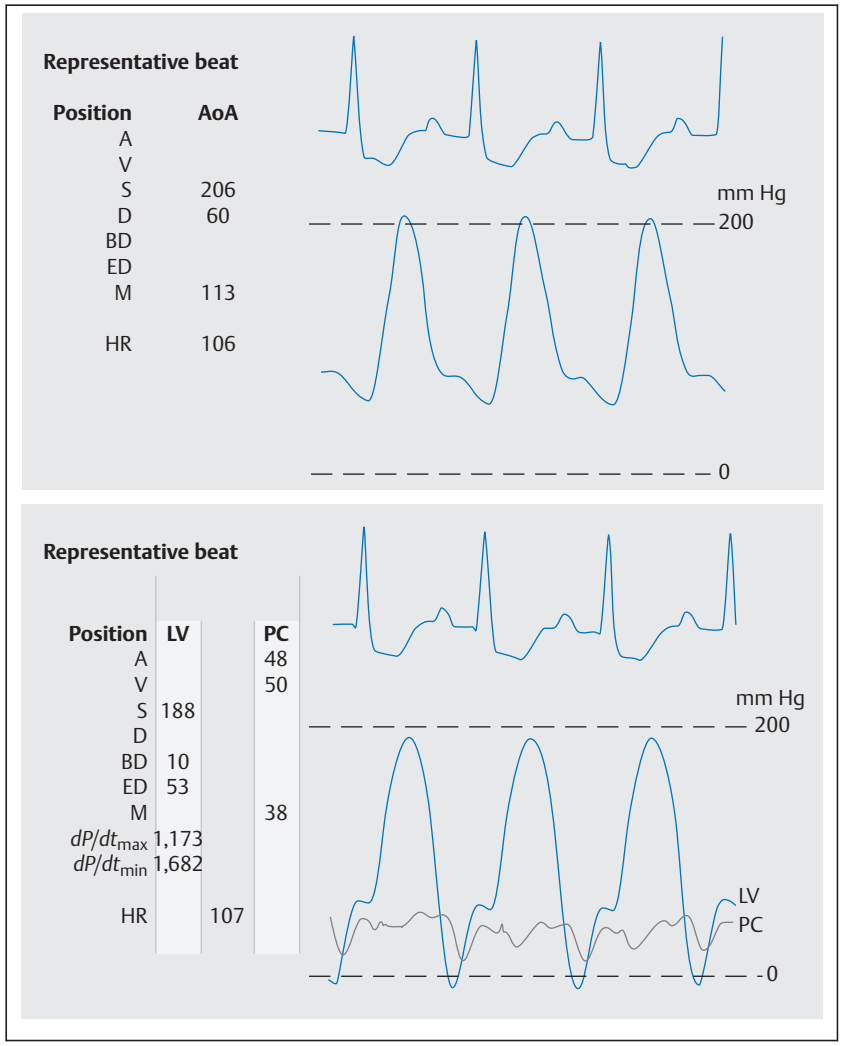

Fig. 14.13 Aortic pressure and left ventricular pressure in severe acute aortic regurgitation. Pressure equalization between diastolic aortic and LVEDP (guiding symptom: unstable angina), typical single peak in the systolic pressure wave.

monary capillary wedge pressure. However, there is rarely pressure equalization between LVEDP and end-diastolic aortic pressure.

! A special characteristic of severe aortic regurgitation is a markedly increased femoral artery systolic pressure compared with aortic pressure; the corresponding pressure difference is frequently from 20 to 50 mm Hg.

Left Ventriculography

In aortic regurgitation, end-diastolic, end-systolic, and total stroke volume can be markedly increased with disease severity. Left ventricular dilatation is frequently more pronounced in chronic versus acute aortic regurgitation.

! As left ventricular dilatation is one of the most important criteria for surgical therapy of aortic regurgitation, left ventricular volume indices should always be calculated in the setting of significant aortic regurgitation.

Ejection fraction in compensated aortic regurgitation is in the normal range or at most slightly reduced.

Angiographic Quantification of Aortic Regurgitation

Details for performing an aortography are described in Chapter 10. The severity of aortic regurgitation is classified by qualitative evaluation of contrast flow during diastole into the left ventricle (**Fig. 14.14**). Alternatively, it can be based on the regurgitant volume, which is calculated using the stroke volume derived from the ventriculogram and the cardiac output determination.