CHAPTER 7. CARDIAC IMAGING

Cardiovascular diseases and their complications are the leading reason for death rate in all industrially-developed countries. Modern technologies of cardiovascular pathology treatment are closely connected with radiodiagnostics. In patients with heart and vessels diseases the following radiological methods of research are used:

1. Primary methods:
   − fluoroscopy and radiography in standard projections;
   − echocardiography and doppler echocardiography.
2. Additional methods (noninvasive):
   − CT;
   − MRI;
   − scintigraphy, SPET or PET.
3. Additional methods (invasive):
   − ventriculography;
   − angiography.

To improve imaging echocardiography, CT and MRI with intravenous introduction of contrast agents can be used.

7.1. Technical considerations

Radiography is a standard screening examination in patients with suspected cardiac disease. Knowing the normal anatomy portrayed on the anterior-posterior (posterior-anterior) and lateral films and by analyzing the sizes of pulmonary arteries and veins, it is possible to make a correct diagnosis in the majority of cases.

Radiography of the thorax in standard projections: direct, left lateral, left and right anterior oblique projections remain a widespread research till nowadays thanks to following possibilities:

− estimation of pulmonary haemodynamics condition;
− detection of heart configuration sizes;
− revealing of calcification structures on the heart and vessels walls;
− exception of other organs pathology simulating clinical semiology of heart and vessels diseases.

Complex use of radiography and ultrasound research allows to do without oblique and lateral projections in most cases. Additional radiographs in oblique projections are required only in 15 % of cases.

The cardiac series is a four-view examination consisting of PA, lateral, and right anterior oblique (RAO) views with the patient drinking barium, and a left

anterior oblique (LAO) view without barium. Barium is used to determine whether or not specific chamber enlargement impinges on the esophagus. The LAO view does not use barium since that substance would obscure the aortopulmonary window. For appreciation of the anatomic relationships of the heart and its chambers, it is necessary to think in three-dimensional terms. Let us examine the position of the cardiac chambers, the great vessels, and the aortic and mitral valves as seen in the four-view cardiac series.

The chest (heart) radiograph in a direct projection. In a direct projection on the right contour there are two arches. Bottom is formed by the right atrium; top an ascending aorta (sometimes vena cava superior). Between arches there is an angle named right atriovascular angle. The left contour of the cardiovascular shadow is displayed as four arches. Top is formed by a shadow of the arch and the beginning of the descending aorta part. The second from above arch is a trunk and the left branch is the pulmonary artery, (nonconstantly) the arch the left atrium below settles down. Left ventricle arch closes the left contour of the heart. The angle between the arch the left atrium and an arch pulmonary artery refers to the left atriovascular angle. The distance from the left ventricle arch up to left medial clavicle lines should make not less than 1,5-2 cm, and from the median line up to the most protrudent point of the upper arch of the left contour − 3-4 cm. The upper contour of the cardiovascular shadow will be in a distance from the horizontal line connecting brest bone-clavicle joints, on 1,5-2 cm. Lengths of arches of the pulmonary artery trunk and the left auricle should be about 2 cm each one. The arch of the left auricle is visualised in norm only in 30 % of cases. Structure of the normal heart shadow and large vessels is usually homogeneous, without additional inclusions (fig.7.1).

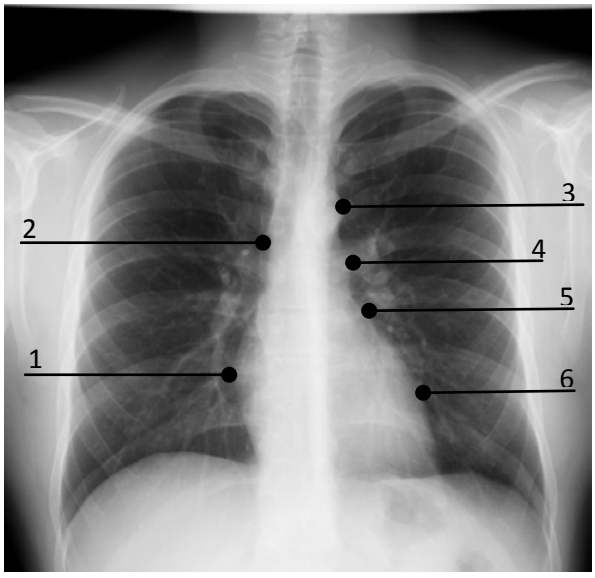

Fig. 7.1. Chest radiograph. Frontal view. 1– right atrium; 2 – ascending aorta; 3 – arch of aorta; 4 – trunk of pulmonary; artery; 5 – left atrium; 6 – left ventrickle. See also text. Norm

In the normal lateral view, the anterior border of the cardiac silhouette consists of the right ventricle. The posterior and inferior cardiac border is that of the left ventricle. Left ventricle adjoins to a diaphragm throughout 5-6 sm, as well as right ventricle to a sternum. The image of the inferior vena cava superimposes on the posteroinferior border of the left ventricle, occasionally extending just posterior to the left ventricular outline. The left atrium forms the superoposterior border of the heart.

The barium-filled esophagus courses almost immediately posterior to the cardiac silhouette. It should not be indented by the heart under normal circumstances. In the left lateral projection increase in the left auricle characterise change of radius of an arch of the contrasted esophagus rejected by it (to 5 sm – small, 5-6 sm – average, more than 6 sm – the big radius).

Occasionally, the image of the pulmonary artery may be observed arching up from the right ventricle and passing inferiorly to the arch of the aorta, which is also visible on the lateral film (fig. 7.2).

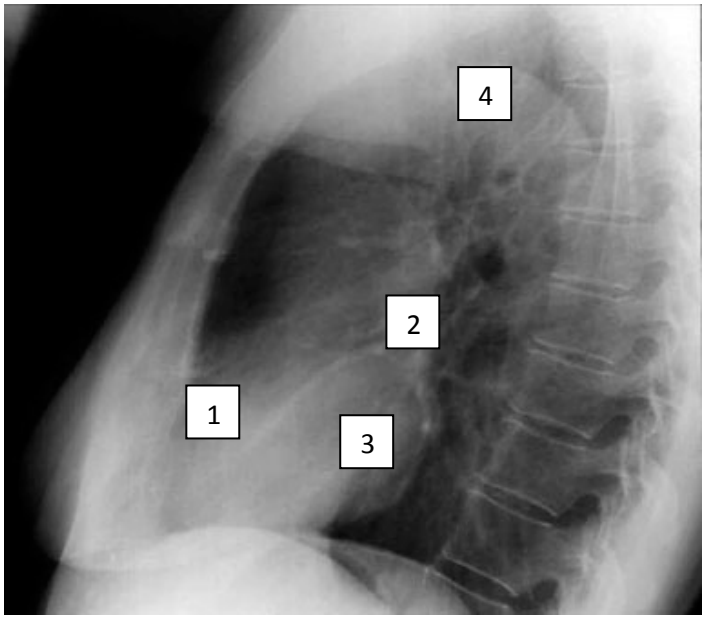

Fig. 7.2. The chest radiograph in the left lateral projection. 1 –the right ventriculus, 2 - the left atrium, 3 - left ventriculus, 4 - the aorta arch

In the right oblique projection (under a corner 45^{o} to the screen the right side) the increase in atrium sizes leads to the esophagus displacement. The dislocation can occur on the arch of the big and small radii. The arch of the big radius is more, and small is less than 6 cm (fig.7.3). In the left oblique projection (under a corner 45^{o} to the screen the left side) heart and vessels also have two contours – forward and back. The anterior contour is formed from below by the right ventricle; the arch of the right atrium is above. The uppermost arch is formed by the ascending aorta. A posterior contour consists of: from below – the arch of the left ventricle, from above – an arch of the left atrium (fig. 7.4).

Form of the heart and large vessels. Normal form of heart is characterized by well enough expressed arches, forming a contour of the heart and large vessels. The shadow of the heart is oblique and also has the normal sizes.

Mitral form of the heart is allocated with the following features. The length and chamber of the arches is formed by a trunk and the left branch of the pulmonary artery and the left atrium increase. Left atriovascular angle decreases and right atriovascular angle is displaced upwards (fig. 7.5).

Aortical form (left ventricular configuration). The following signs are characteristic:

1. Emphaticalness of the waist (expressiveness, dredging on the left contour between the aorta arch and the left ventricle arch, therefore the distance between atriovascular corners seems small).

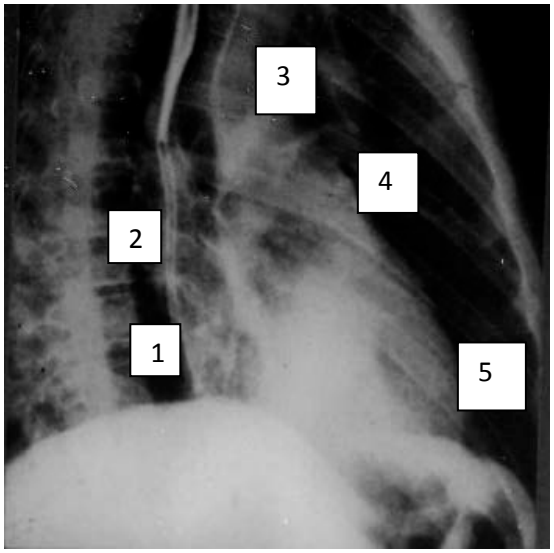

Fig. 7.3. The heart radiograph in the right (first) oblique projection. The contrasted oesophagus settles down rectilinearly. 1 − arches of the right atrium, 2 − arch of the left atrium, 3 − arch of an ascending aorta, 4 − arch of the pulmonary cone, 5 − left ventriculus arch. Norm [31]

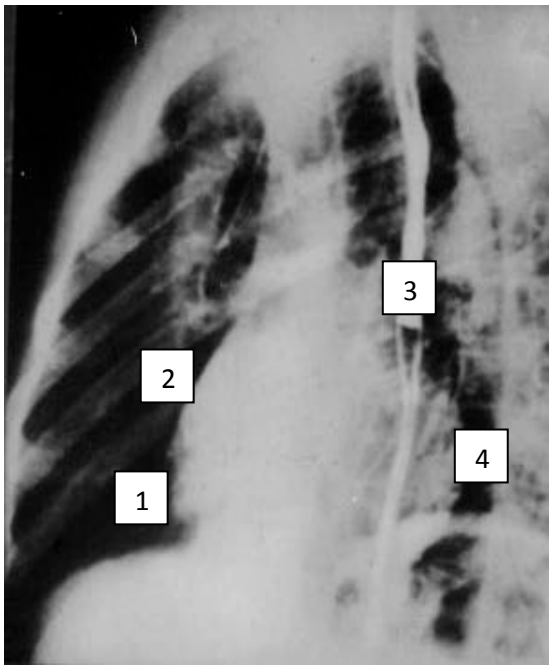

Fig. 7.4. The heart radiogrraph in the left (second) oblique projection. 1 − right ventriculus arch, 2 − arch of the right atrium, a 3 − arch of the left atrium, 4 − arch of the left ventriculus.
Norm [31]

2. Increase of the arch forming the left ventricle.
3. Lengthening of the arch and expansion of the shadow in the projection of ascending aorta (the right contour, the top arch).
4. Lengthening the top arch of the left contour is conditioned by the arch and the descending part of the aorta.
5. Displacement downwards of the right atriovascular angle.

At the triangular (spherical) form of the heart its sizes are in regular intervals increased and division of contours into arches is not observed. In a direct projection the form of the heart shadow and large vessels has similarity with triangle or a trapeze.

A popular method used to determine cardiac size is the cardiothoracic ratio: the maximum width of the cardiac shadow on the posterior-anterior (PA) or anterior-posterior (AP) chest film divided by the maximum width of the thorax (norm is < 0,5).

Fluoroscopic examination of the heart and pulmonary vessels is used for (a) assessment of cardiac motion, contour, and dynamics (useful for evaluation of cardiac aneurysms), (b) investigation of intracardiac calcifications (valvular, coronary artery, or pericardial), and (c) assessment of patients with suspected pericardial effusion (dampened pulsations).

It has largely been replaced by echocardiography.

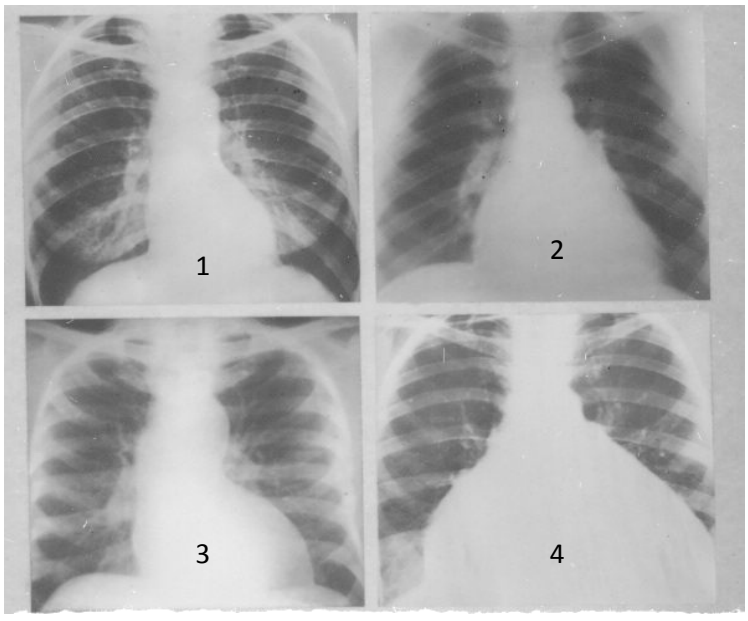

Fig. 7.5. Heart configurations.
1 − normal form of the heart, 2 − mitral configuration of the heart, 3 − aortic configuration of the heart, 4 − spherical configuration of the heart [31]

Cardiac catheterization and coronary arteriography are invasive procedures performed almost exclusively by cardiologists or cardiovascular radiologists. These procedures allow accurate evaluation of the size and configuration of the cardiac chambers, the great vessels, and the coronary arteries. They are also performed to

evaluate patients with suspected shunt lesions.

Computerized tomography, performed with electrocardiographic CT gating, is used with contrast enhancement for a variety of cardiac conditions. In this technique, dynamic scanning – multiple images of one section – is performed to evaluate flow through a particular chamber or vessel. In addition, CT is used to evaluate the patency of coronary artery bypass grafts, to assess the extent of myocardial infarcts, to depict the size and location of left ventricular aneurysms, to detect aneurysms of the thoracic aorta, to diagnose aortic dissections, to define certain congenital abnormalities such as coarctation of the aorta and anomalous venous connections, and to assess the pericardium for effusion and calcification (fig. 7.6).

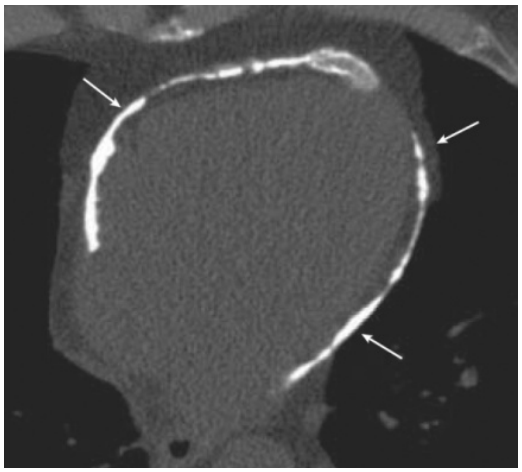

Fig. 7.6. The computer tomogram of the chest. Calcification of the pericardium is detected. (arrows)

Dynamic CT is also used for determining myocardial wall thickness and dynamics, although echocardiography is used much more commonly.

Modern technology CT provides three-dimensional reconstruction of the vascular tree. CT-angiography becomes in some cases alternative to angiography as a definitive method of stenosis and aneurysm diagnostics. Unlike angiography this method allows to visualise not only the vessel lumen, but also a clot of blood with surrounding tissue. Spatial resolution of CT-angiography is lower, than that of angiography. One of indications for CT – angiography is visualisation of the trunk veins in thrombosis, occlusion, anomalies of development, tumours (fig. 7.7, 7.8, 7.9).

The left side of the heart can also be shown by following the contrast agent through the lungs to the left auricle and ventricle on serial films. However contrast values are not as good as in direct left heart ventriculography.

For left heart angiocardiography the catheter tip is sited in the left ventricle by the method of transfemoral catheterisation just described. Injections are made through the catheter and rapid serial films taken. The left ventricle is best studied by video-filming, and this method is essential in the study of the ischaemic heart in coronary

disease. Left ventricular function is assessed radiologically by noting the adequacy of left ventricular contraction and the presence of dyskinesia areas. Mitral incompetence can be demonstrated at left ventriculography by opacification of the left auricle and the degree of incompetence quantified.

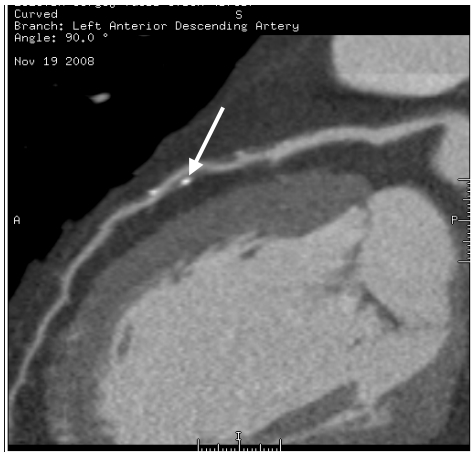

Fig. 7.7. CT with contrast enchanced. The forward descending coronary artery with calcification (arrow) is visualised

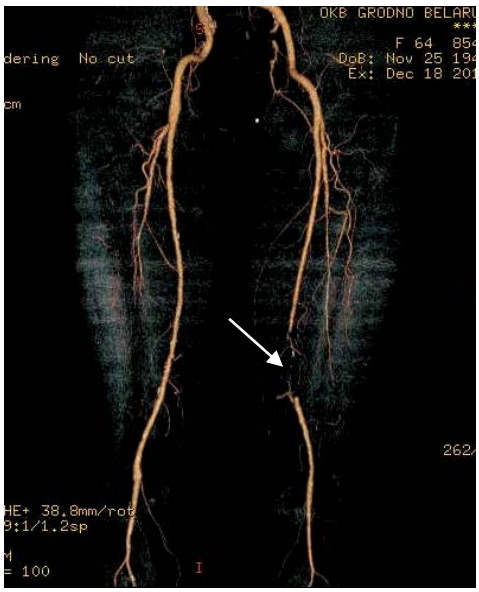

Fig. 7.8. Multi-slice spiral computed tomography – volume rendering. Frontal view. Three-dimensional reconstruction arteries hips. On the left is absent the image distal part of superficial femoral artery because of it occlusion (arrow). Occlusion of the lef tsuperficial femoral artery

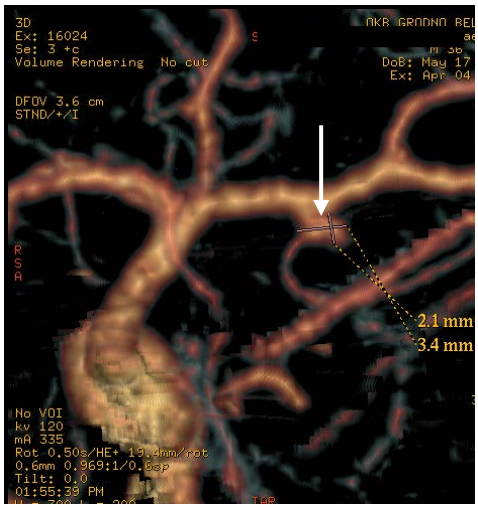

Fig. 7.9. 3 D spiral CT. Aneurism at posterior brain artery (arrow)

Angiocardiography may be performed from either the right or the left side of the heart. In venous angiocardiography the catheter tip was sited either in the superior or inferior vena cava, and a bolus of contrast medium injected at high pressure. Rapid films were taken demonstrating its passage through the various chambers of the heart.

Right heart angiocardiography is now more usually performed by siting the catheter tip in the right atrium. In many congenital heart conditions selective angiocardiography is performed with the catheter tip sited in the right ventricle or pulmonary outflow tract (fig. 7.10).

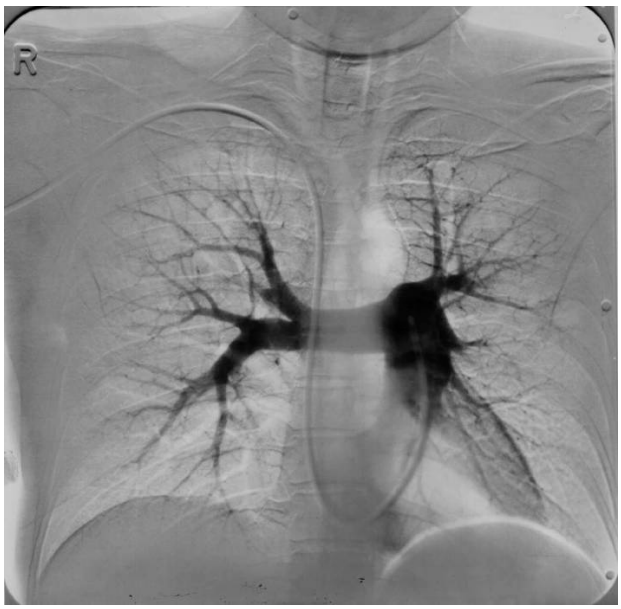

Fig. 7.10. Digital right angiocardiography in a direct projection. Phase of contrasting of pulmonary arteries

The aortic valves may be studied by injections made into the root of the aorta. With aortic incompetence there will be regurgitation into the left ventricle; with aortic stenosis the narrowed jet of blood from the ventricle will be shown as a defect in the opacified aorta.

Echocardiography. The cardiac series, too, has largely been replaced by echocardiography. Real-time echocardiography has decreased the number of catheterizations used to determine cardiac chamber size and configuration.

Ehocardiography, cardiac imaging technique based upon the velocity of sound travelling through and reflected from acoustic interfaces in cardiovascular structures. It has progressively evolved from M-mode echocardiography to the current multifaceted capabilities including transthoracic and transoesophageal echocardiography, three-dimensional echocardiography, Doppler velocity measurement, colour flow mapping and intravascular imaging. Echocardiography has become the most frequently performed diagnostic study for cardiac diseases. Echocardiography is the most widespread beam method of research of heart and

vessels, thanks to the availability and information. Combination echocardiographyand doppler echocardiography allows to estimate:

− a status of departments of heart and large vessels;

− a status of intracardiac structures;

− intracardiac and central haemodynamics;

− total and segmentary myocardium contraction function;

− presence of pathological intracardiac shunts;

Transoesophageal echocardiography requires passage of an oesophagoscope with an ultrasound transducer at its tip which can be angled and placed at different levels behind the heart. This enables high quality images to be obtained in cases where the conventional techniques are difficult or unsuccessful.

M-mode echocardiography provides a one-dimensional (distance from the transducer versus time) view of cardiac structures. Cardiac motion is displayed as a change in position of cardiac structures; i.e. mitral leaflet motion, over the cardiac cycle. The distance between and changes in distance between various cardiac structures is displayed on one-dimensional echocardiograms. The M-mode method provides interrogation of moving cardiac structures with a sampling rate of nearly 1000 cycles/sec. The M-mode echocardiogram also depicts abnormal patterns or velocity of motion in cardiac structures such as the mitral leaflet in flail mitral valve and mitral stenosis, respectively.

Two-dimensional echocardiography (2DE) uses rapid movement of the one-dimensional ultrasonic beam across the heart to provide real-time cross-sectional images. It is the standard ultrasound imaging method for the heart. There are two major types of two-dimensional imaging devices, mechanically driven large crystals and electronically driven phased crystal arrays. The electronically driven systems are now dominant.

Doppler echocardiography allows the measurement of intracardiac and intravascular flow velocities by detecting changes in the frequency of reflected ultrasound emitted by and then returned to the transducer. After emitted ultrasound strikes moving red blood cells, the frequency of the ultrasound is shifted in proportion to the velocity of the cells. This velocity difference of the ultrasound is displayed as a function of time and direction of the flow in relation to the transducer. There are two types of Doppler modalities: pulse wave Doppler and continuous wave Doppler. Pulse wave Doppler is capable only of measuring velocities accurately in the lower range due to aliasing. Flow mapping or colour Doppler is a special form of pulsed wave Doppler. Colour Doppler is used to screen the heart for flow disturbances such as valvular regurgitation and stenosis. Continuous wave Doppler obviates aliasing and can be used to accurately measure high velocity flows such as

those associated with stenoses.

Ultrasonic anatomy of heart. At heart research standard positions of the transduser (fig. 7.11, 7.12, 7.13) are used:

1. Parasternal position – area between the III-V ribs to the left from the breast.
2. Apex position – area of the cardiac top shove.
3. Subcostal position – area under processus xiphoideus.
4. Suprasternal position – fossa jugularis.

Overall, the most common indications for echocardiography are suspected chamber enlargement, congenital heart disease, abnormalities of heart valves, abnormalities of contractility, and suspected pericardial effusions. This examination is performed primarily by cardiologists.

Cardiac magnetic resonance imaging, noninvasive cardiac imaging technique in which intrinsic contrast exists between the blood pool and cardiac structures. There are several features which are useful for cardiac imaging. High contrast between the blood and cardiac structures exists because of the low or absent signal of flowing blood on T1WI and T2WI (fig. 7.14) or the high signal of blood on gradient echo images.

A wide range of contrast among soft tissues provides the potential for myocardial tissue characterization. The capability to acquire tomograms in any plane allows images to be acquired along the long axis (parallel) or short axis (perpendicular) of cardiac chambers and other cardiovascular structures. It is essentially a three-dimensional imaging technique which provides the most accurate and reproducible measurements of cardiac volumes and myocardial mass. Flow-sensitive MR sequences provide spatially precise measurements of velocity and volume of blood flow in cardiac chambers and blood vessels. Magnetic resonance imagingis employed to diagnose many of the same abnormalities that can be seen with CT.

Electrocardiographic gating is used for "stop-action" images of the heart and great vessels. Magnetic resonance imaging has the advantage of portraying flowing blood as a signal void (black) so that it is easy to distinguish blood from solid structures. Magnetic resonance imaging is most useful for evaluating patients with aortic dissections and aortic coarctation as well as chamber abnormalities.

Myocardial metabolic imaging, techniques used clinically to assess myocardial viability with nuclear scanning techniques and in research to evaluate various metabolic pathways in the heart in vivo with nuclear scanning techniques and MR spectroscopy. The classical example is PET imaging with fluorodeoxyglucose (FDG). In this technique, a PET perfusion image is acquired first followed by images of myocardial FDG uptake.

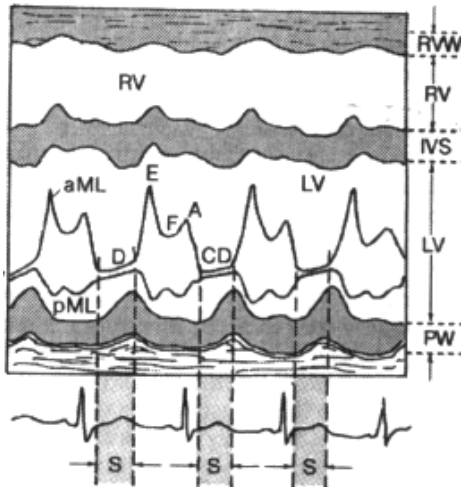

Fig. 7.11. Ultrasound examination (M-mode) from left parasternal position on the heart long axis. Position of a transducer ultrasonic beam is at the level of leaflets mitral valve. RVW – forward wall right ventriculus. Behind it the cavity of right ventriculus (RV) and septum interventriculare (IVS) is visible. LV – cavity of the left anterior (aML) and posterior (pML) leaflets of mitral valve move. PW – the back wall left ventriculus. Normally in diastole diphasic M-shaped movement of aML and W-shaped movement pML is defined. On a curve of movement of aML some sites are allocated: 1) interval C-D corresponds to systole LV and full closed valve leaflets; 2) interval D-E reflects a divergence of leaflets of the valve during a phase of fast filling LV; 3. interval E-F − incomplete cover of leaflets of the valve during a phase of slow filling; 4) the wave A is caused by a repeated divergence of leaflets during systole LA [72]

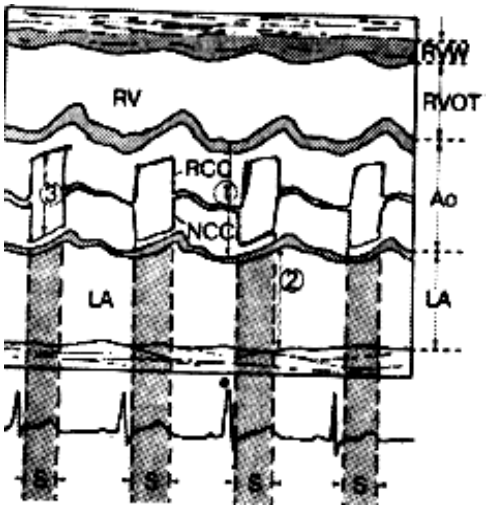

Fig. 7.12. The scheme of ultrasound examination (M-mode) from left parasternal position at the level of the aorta and aortic valve. The note: S − systole ventriculus, RVOT − right ventricular outflow tract, RCC − right coronary leaflet aortic valve, NCC − not coronary − leaflet aortic valve, «1» − diameter of the aorta, «2» − diameter LA, «3» − amplitude of disclosing of the aortic valve [72]

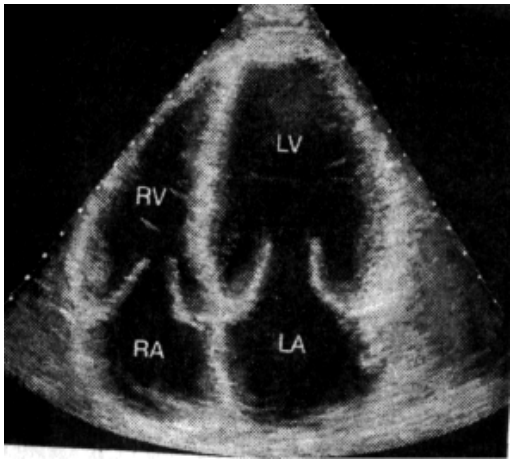

Fig. 7.13. The scheme of ultrasonic scanning in apex positions. Ultrasound examination (B-mode). Research from apex position. The note: septum interventricular and septum interatrial partitions settle down in the centre. To the left of them right ventricle (RV) and the right atrium (RA) are visualised and on the right − left ventricle (LV) and left atrium (LA). In this position leaflets mitral and tricuspid valves are well visualised also [72]

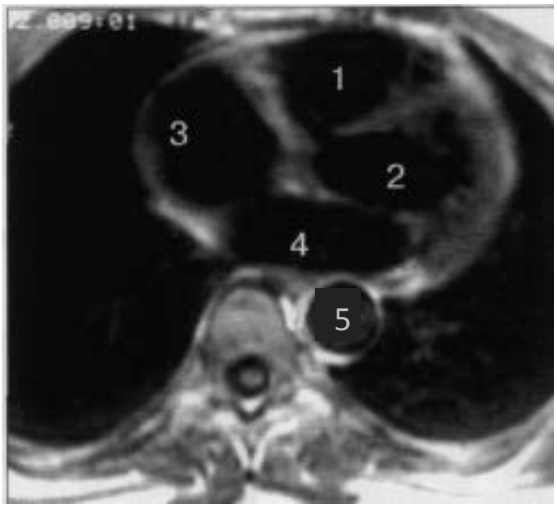

Fig. 7.14. MRI (T1 –WI) of the chest at the level of Th VII vertebra. 1 – right ventricle, 2 – left ventricle, 3 − right atrium, 4 – left atrium, 5 − descending aorta

Areas which show persistent metabolism (FDG uptake) but poor perfusion are identified as hibernating myocardium. Thallium-201 (^{201}Tl) can also be used as a marker of myocardial vitality, as rest injection and imaging after at least 1 hour will show regions of delayed uptake which are also identified as hibernating myocardium. PET imaging is considered the gold standard for this diagnosis while thallium is considerably less sensitive. An alternative method used to identify hibernating myocardium is stress echocardiography.

Nuclear myocardial perfusion imaging is the method for displaying the regional myocardial distribution of radiolabelled perfusion agents as an indicator of myocardial blood flow. Usually, the localization of the agent involves blood flow in capillaries, extraction from capillaries and retention in viable myocytes. Regional distribution of nuclear perfusion agents is influenced by two major factors: myocardial blood flow and myocardial cellular viability. Myocardial perfusion imaging is the most frequently employed method for the diagnosis and determination of the severity of ischaemic heart disease. The technique has evolved from planar imaging to single photon emission tomography (SPECT). The perfusion agents most frequently used are thallium-201 (^{201}Tl) and technetium-99m (^{99m}Tc) labelled agents. The initial distribution of ^{201}Tl is related to regional blood flow and the relative myocardial extraction of the tracer from the blood. The early myocardial perfusion deficits are produced predominantly by regional reduction in blood flow. Initial images are done after injection of ^{201}Tl during stress, and rest images are done about four hours later. Disappearance of initial perfusion defects on delayed images indicates redistribution of the agent caused by slower clearance of ^{201}Tl from under perfused compared to normally perfused regions. Tc-99m-labelled perfusion agents such as ^{99m}Tc sestamibi and ^{99m}Tc-teboroxine have been used in recent years because of better imaging properties compared to ^{201}Tl. Because ^{99m}Tc-sestamibi does not redistribute, separate injections must be done for rest-stress studies.

Nuclear perfusion imaging may be done after injection of the agent at rest or during peak of exercise or pharmacological stress. Pharmacological stress or near maximal vasodilatation is induced by dipyridamole, adenosine or dobutamine. Based on the changes in regional perfusion defects from stress to rest states, defects can be diagnosed as fixed, reversible or partially reversible. Fixed defects are those which are nearly identical in both states and indicate infarction. Reversible defects are present on stress images but not on rest images and indicate ischaemia without infarction. Partially reversible defects show a defect during stress in which the concentration of the agent in the defect increases on the delayed rest image but does not equalize with the normal regions. This pattern is considered to represent a mixture of nonviable and viable but ischaemic myocardium.

The clinical applications of myocardial perfusion imaging are: detection of coronary artery disease (CAD) in asymptomatic and symptomatic patients; estimation of the severity of CAD; distinction between single and multivessel CAD; stratification of risks for coronary events; risk stratification after acute myocardial infarction; and risk stratification in patients undergoing noncardiac surgery (fig. 7.15).

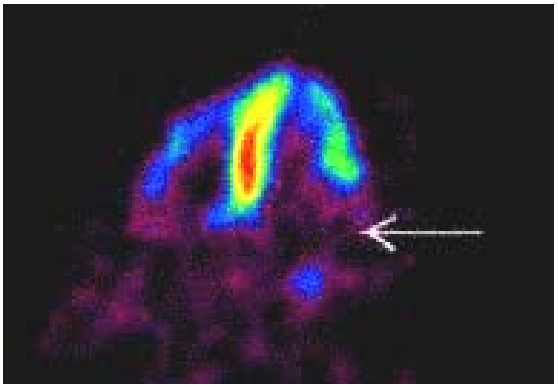

Fig. 7.15. Myocardial perfusion imaging. PET data with the labeled fatty acid – ^{11}C-sodium butyrate (horizontal section on a long axis). Pronounced decrease in perfusion hypoperfusion area indicated by the arrow) [28]

Nuclear angiography can also be done in two different ways.

1. First-pass technique involves rapid i.v. injection of a bolus of a simple radionuclide (^{99m}Tc - pertechnetate). Its passage through the cardiac chambers is then recorded. The method is most useful for the study of intracardiac shunts.

2. Multigated equilibrium studies (MUGA) follow injection of an isotope which remains fixed within the vascular space (^{99m}Tc -labelled human serum albumen or red blood cells) thus labelling the total blood pool. Cardiac movement is then recorded. Abnormalities of ventricular function, particularly those due to ischaemic heart disease and cardiomyopathy are readily assessed by this method. Computer manipulation of the data also enables ventricular ejection fractions to be obtained.

7.2. Pathologic considerations

There are many ways to classify cardiac diseases. A popular classification uses two large categories, congenital and acquired cardiac disease.

From a physiologic standpoint, all types of cardiac disease may be categorized into the following:

I. Obstruction.

II. Volume overload.

A. Shunt (right-to-left, left-to-right).

B. Valvular insufficiency.

III. Disorders of contraction or relaxation.

A. Myocardial disease.

B. Conduction disorders (arrhythmias).

IV. Combination of the preceding.

No matter what the etiology is, all cardiac diseases will show evidence of one or more of these patterns.

Evaluation of the pulmonary vascularity is an important step that enables exclusion of many diseases. The physiologic type of disease may be inferred from the pattern of pulmonary blood flow.

Normal pulmonary vessels should be about the same size as that of an accompanying airway. Any significant disparity in size is abnormal.

Surprising as it may seem, patients with normal pulmonary vascularity may have significant cardiac disease. In these patients, the heart has compensated for the abnormality by enlarging. The pulmonary vascularity remains normal until the heart decompensates. Diseases that produce cardiac chamber enlargement without appreciable change in the pulmonary vascularity until decompensation occurs include cardiomyopathy, coronary artery disease, hypertensive cardiovascular disease, aortic stenosis, and coarctation of the aorta. All these conditions except coarctation and a form of aortic stenosis are acquired.

Decreased vascularity indicates a severe obstruction to the outflow of blood from the right ventricle, usually at the pulmonic valve or subvalvular level. Patients exhibiting this pattern are often visibly cyanotic. If the decreased vascularity is of a diffuse nature, a congenital anomaly is most likely. This pattern is seldom seen in the adult, since the abnormalities that produce this pattern will result in the patient's death unless corrective surgery is performed during childhood.

Decreased vascularity may be apparent locally or unilaterally. A local decrease in vascularity may be the result of pulmonary embolism (Westermark sign), emphysema, or scarring with rearrangement of vessels in a lung. Increased vascularity is of four types: (1) shunts, (2) pulmonary venous obstruction, (3)

precapillary hypertension, and (4) high-output state.

Shunts represent an increased flow through the pulmonary bed. They are characterized by large vessels in the upper and lower lobes. A similar pattern may occur in high-output states. In patients with a shunt who are not in congestive heart failure, the redistribution of blood will be in the same proportion as that occurring normally: greater to the lung bases than to the upper lobes. This vascular pattern occurs most commonly in a left-to-right shunt at the cardiac or great vessel level (septal defect or patent ductus arteriosus). This pattern is uncommon in adults since the condition is usually diagnosed and treated in childhood.

Patients with pulmonary venous obstruction (PVO) demonstrate large veins in the upper lobe as a reflection of reversal of the normal flow pattern (fig. 7.16).

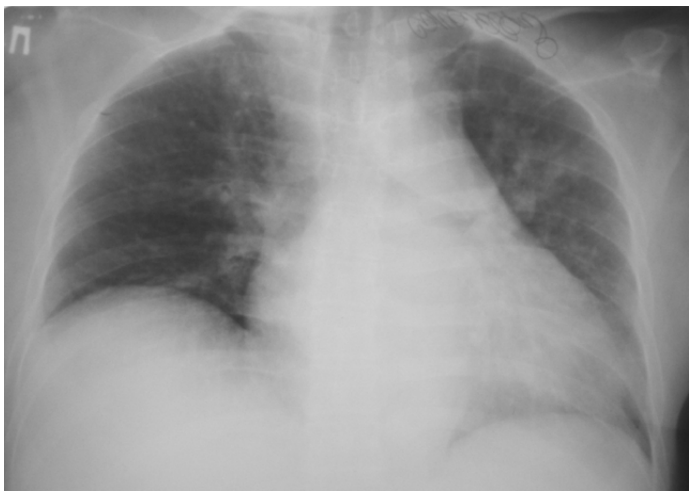

Fig. 7.16. The frontal chest radiograph. Cardiomegaly, heart size exceeding normal dimensions. Cardiomegaly is usually initially identified by plain radiography. In adults, cardiomegaly is considered to be present if the ratio of the maximum cardiac diameter to the maximum thoracic dimension on a standard posteroanterior radiograph exceeds 0,50. This constitutes a cardiothoracic ratio greater than 0,50. Alveolar pulmonary edema. Engorgement of the pulmonary veins

This indicates increased left atrial pressure. Severe PVO is manifested by pulmonary edema and prominent interlobular septal (Kerley) lines.

Patients with precapillary hypertension (pulmonary arterial hypertension) have large central vessels that taper rapidly into small vessels peripherally. This is referred to as centralized flow and occurs in patients with severe pulmonary disease, recurrent pulmonary embolism, and Eisenmenger phenomenon.

Once the pulmonary vascular pattern is decided on, look at the heart to determine if specific chamber enlargements are present. If there is evidence of left atrial enlargement (with or without PVO), rheumatic heart disease (mitral stenosis) or an obstruction at or proximal to the mitral valve is present. If there is evidence of left ventricular enlargement with a "concavity" in the area of the main pulmonary artery, the disease is one of left ventricular stress such as hypertensive cardiovascular disease, coronary artery disease, aortic stenosis, or coarctation of the aorta.

Pulmonary venous obstruction plus left ventricular configuration (LVC) equals left ventricular stress with failure. All the preceding conditions occur with this pattern. It is possible to further narrow the list of causes in this situation by scanning the film for evidence of rib notching and/or decreased size of the aortic knob, as in aortic coarctation, or for calcification in or about the aortic valve, as in calcific aortic stenosis.

A high-output state, such as severe anemia or thyrotoxicosis, may result in increased vascularity with a normal distribution as a result of the increased volume being pumped through the heart. The heart itself may be normal or slightly enlarged as a result of this increased activity.

Pericardial effusion must always be considered when evaluating a patient with the enlarged heart. The diagnosis may be made by one or a combination of imaging studies. In general, a large heart of nonspecific configuration, particularly in the absence of pulmonary venous enlargement (fig. 7.17), should suggest a pericardial effusion. Cardiac fluoroscopy is a useful procedure for the diagnosis of pericardial effusion. A dampened cardiac pulse in the presence of an enlarged heart and no congestive heart failure suggests the condition. However, this is by no means pathognomonic, since a poorly contracting heart in a patient with cardiac arrhythmia, scarred myocardium, or infiltrated myocardium will produce poor pulsations. A pulsating subepicardial fat line within the immobile fluid band is, however, diagnostic of pericardial effusion. Echocardiography is probably the most useful examination for detecting this condition and with the least risk to the patient. Ultrasound reflected off the pericardial and myocardial surfaces will demonstrate an abnormal collection of fluid in the pericardial sac (norm size of fluid up to 4 mm). Computerized tomography scanning may be also used to diagnose pericardial effusion. A CT number near the density of water surrounding the heart ensures the diagnosis. This diagnosis is usually made as an incidental finding in patients studied for other reasons.

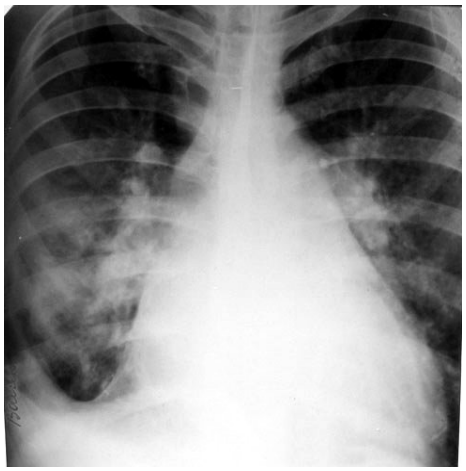

Fig. 7.17. The frontal chest radiograph. Pericardial effusion: a smoothness of contours of heart, its triangular form. Pleural effusion: in the bottom department of the right pulmonary field shadow is defined. Shadows of hila are increased, because of expansion of vascular trunks forming them

Trauma. Patients who have suffered severe thoracic trauma may have injury to the heart or great vessels. The most common mechanism for this is an accident in which the unrestrained driver of a motor vehicle strikes the steering wheel. Radiographically, the most common finding is a widened superior mediastinal shadow that is fuzzy. In the appropriate clinical setting, an aortogram should be obtained to rule out aortic injury.

Mitral regurgitation. Mitral regurgitation, systolic flow of blood from the left ventricle into the left atrium due to insufficient closure of the mitral valve. It may be caused by pathology of the mitral leaflets, subvalvular mechanism (chordae or papillary muscles) or mitral annulus. There are a large number of aetiologies for mitral regurgitation including rheumatic heart disease, infectious endocarditis.

The haemodynamic consequence of mitral regurgitation is systolic increase in left atrial pressure and pulmonary venous pressure during systole. The increase in pulmonary venous pressure is usually less severe than with mitral stenosis. However, severe elevation in pulmonary venous pressure occurs with acute onset of regurgitation. Mitral regurgitation imposes a volume load on the left atrium and ventricle. Left ventricular end-diastolic volume is increased. The total stroke volume of the left ventricle is increased since it includes the effective stroke volume (blood ejected to the aorta) and the regurgitant volume. Plain radiography shows various degrees of pulmonary venous hypertension and cardiomegaly. The severity of pulmonary venous hypertension is generally less than in predominant mitral stenosis. Cardiomegaly is a consequence of left atrial and left ventricular enlargement (fig. 7.18).

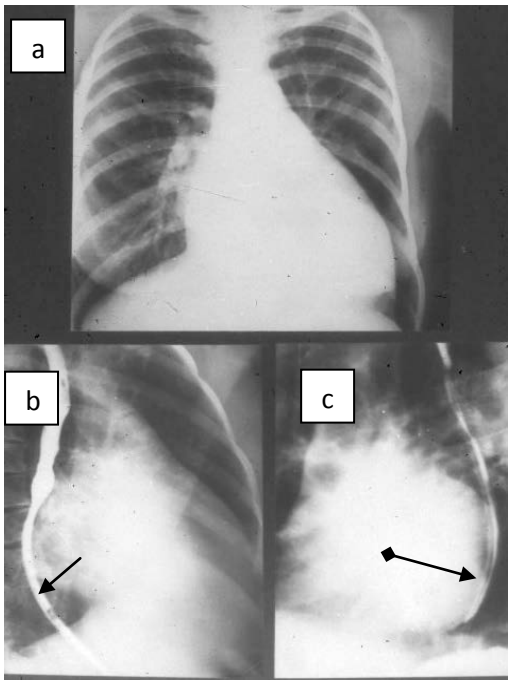

Fig. 7.18. Heart radiographs in a direct (a), right (first) oblique projection (b), left (second) oblique projection (c); a) the mitral configuration of the heart. On the left contour lengthening of arches left ventriculus, an ear of the left atrium, a pulmonary artery. On the right contour the arch of the right atrium is increased; b) in the right (first) slanting projection displacement on an arch of the big radius (> 6 sm) the contrasted oesophagus the increased left atrium (arrow), increases an arch left ventriculus; c) in the left (second) oblique projection on a forward contour the arch the bottom arch right ventriculus is increased. On a back contour arches of the left atrium and left ventriculus are extended (arrow with rombus). Mitral regurgitation [31]

Two-dimensional echocardiography shows the above signs described for M-mode echocardiography. This study may reveal the aetiology of the regurgitation by showing mitral valve prolapse; papillary muscle rupture or cordal rupture; thickened leaflet with fused commissure and decreased motion in rheumatic disease; vegetations or perforated leaflet in infectious endocarditis; parachute or cleft mitral valve in congenital disease; or mitral annular calcification. In chronic mitral regurgitations left ventricular volumes are increased and can be effectively monitored with two-dimensional echocardiography. The extent of the increase in left ventricular volumes is a prognostic indicator for surgical outcome. A left ventricular systolic volume over 60 ml/m^2 is associated with a worse prognosis. Left ventricular dimensions at end diastole greater than 7 cm and at end systole greater than 5 cm are indicative of severe diseases.

Pulse wave Doppler echocardiography is extremely sensitive for detecting mitral regurgitation; it appears as a turbulent systolic signal within the left atrium directed away from the transducer. The extent of the penetration and area of the regurgitant jet can be used to estimate the severity (fig. 7.19).

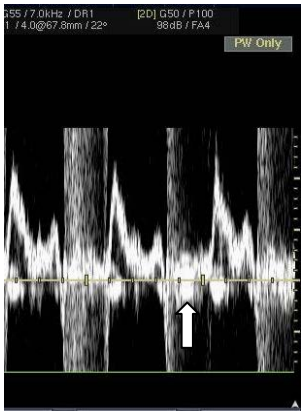

Fig. 7.19. Pulse wave Doppler echocardiography from position heart apex. Transmitral the blood stream is partially directed below a base line of the spectrogram that is a sign of mitral regurgitation (arrow). Mitral regurgitation

Colour flow Doppler provides a nearly real-time flow map of the origin and direction of mitral regurgitation. Large colour jets that occupy more than a half of the left atrium, extend to the posterior portion of the atrium or into the appendage or pulmonary veins indicate significant regurgitation.

Left ventriculography shows escape of contrast media from the left ventricle into the left atrium during systole. Comparison of the intensity of opacification of the left atrium with the left ventricle provides a semiquantitative estimate of severity. Quantitative left ventriculography reveals increased left ventricular end diastolic, end systolic and stroke volumes. The regurgitant volume in isolated mitral disease can be calculated as the difference in stroke volume calculated from left ventriculography (end-diastolic – end-systolic volume). Structural abnormalities of the valve such as flail valve or vegetations are revealed by left ventriculography. For the most part,

echocardiography has supplanted angiography for the diagnosis and assessment of severity of mitral regurgitation.

Preoperative catheterization is performed mainly for the purpose of coronary arteriography in patients over 40 years old.

Cine MRI displays the regurgitant jet as a signal void emanating from the mitral valve projecting into the left atrium during systole. The size of the signal void bears a rough relationship to the severity of regurgitation. Cine MR images encompassing the entire heart can be used to measure left atrial and ventricular volumes with high precision and reproducibility. Velocity-encoded cine MRI can be used to measure the volume of regurgitation. It can be measured as the difference in the inflow volume across the mitral annulus in diastole and the outflow volume through the ascending aorta in systole.

Mitral stenosis. Mitral stenosis, abnormal resistance to blood flow from the left atrium to the left ventricle due to narrow mitral orifice. There are a number of causes of mitral stenosis but most cases are due to rheumatic heart disease. Rarely, it is caused by congenital defects such as parachute mitral valve or mitral annular and valvular hypoplasia. The mitral valve can also be obstructed secondarily by tumours such as left atrial myxoma or left atrial thrombus or rarely by mitral annular calcification. Deterioration of artificial mitral valves can cause mitral stenosis. Mitral stenosis is complicated frequently by chronic atrial fibrillation and left atrial thrombus. The haemodynamic consequences of mitral stenosis are increases in left atrial, pulmonary venous and pulmonary arterial pressures. In some patients severe pulmonary arterial hypertension develops, causing secondary pulmonary regurgitation, tricuspid regurgitation and substantial right-sided chamber enlargement.

The chest radiography demonstrates signs of pulmonary venous hypertension in nearly all patients with haemodynamically significant mitral stenosis. In milder disease, there is merely equalization of the calibre of blood vessels in the upper and lower lobe regions, while with other cases there is interstitial and/or alveolar pulmonary oedema. The cardiac size is usually not substantially enlarged but there is invariably left atrial enlargement. The left atrial appendage is enlarged, especially in patients in whom stenosis is caused by rheumatic heart disease. In isolated mitral stenosis the left ventricle is not enlarged. Often the combination a mitral stenosis and mitral regurgitation is observed (fig. 7.20).

However, mitral regurgitation is sometimes also present which may cause left ventricular enlargement. The right ventricle may be either slightly or substantially enlarged depending on the severity of pulmonary arterial hypertension. Pulmonary arterial hypertension is evident by enlargement of the main pulmonary artery.

Calcification of the mitral valve, left atrial appendage or left atrial wall may be evident on the radiograph or revealed by fluoroscopy.

The M-mode echocardiography demonstrates slow initial closure of mitral valve (decreased EF slope), anterior motion of the posture leaflet as well as the anterior leaflet, decreased diastolic separation of leaflets, and thickened leaflets by them fibrosis (fig. 7.21). Two-dimensional echocardiography shows thickened and relatively immobile leaflets, doming of the valve, chordal foreshortening and thickening.

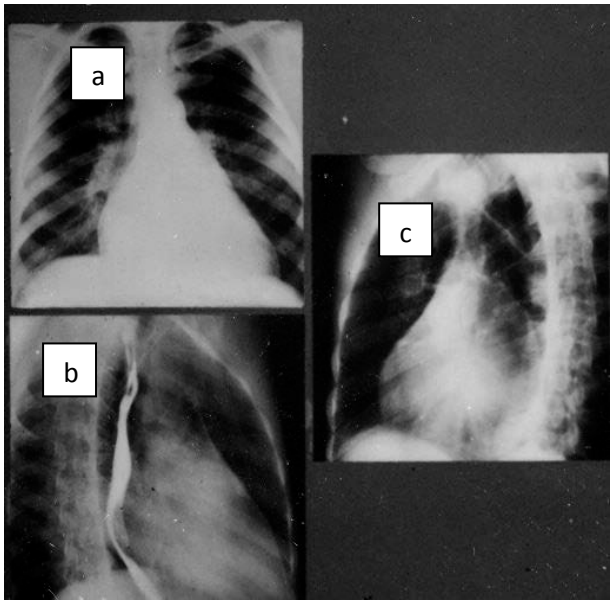

Fig. 7.20. Heart radiographs in a direct (a), right (first) oblique projection (b), left (second) oblique projection (c); a) the mitral configuration of the heart. On the left contour lengthening of the left ventriculus arches, ear of the left atrium, pulmonary artery is detected. On the right contour the arch of the right auricle is increased); b) in the right (first) oblique projection on the arch of small radius (<6 sm) the contrasted oesophagus is displaced due to the increased left auricle, increases the left ventriculus arch); c) in the left (second) oblique projection on a forward contour the arch the bottom arch right ventriculus is increased. On a back contour arches of the left auricle and left ventriculus are extended.

Mitral regurgitation and mitral stenosis [31]

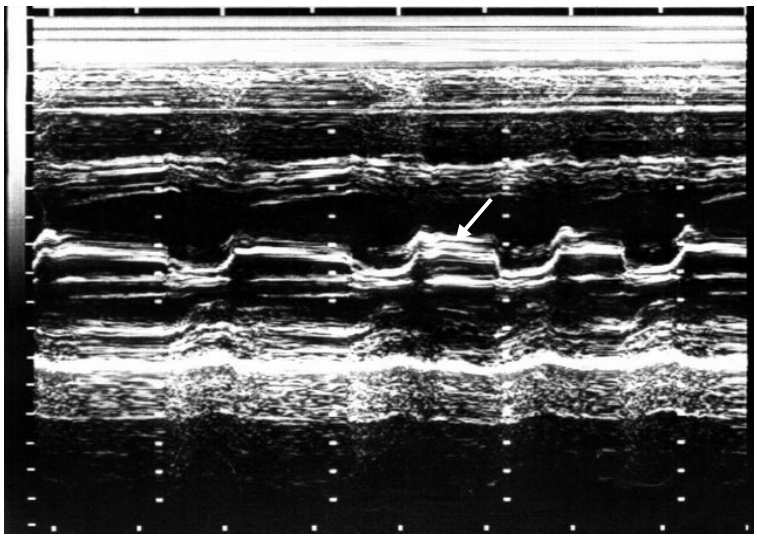

Fig. 7.21. M-mode echocardiogram (parasternal a position). Mitral stenosis: fibrosis of leaflets of mitral valve and restriction of mobility of a back leaflet of mitral valve (arrrow)

It may also disclose calcification of the valve or subvalvular apparatus. Doppler

echocardiography shows characteristic features. The normal Doppler mitral inflow pattern shows two peaks for early diastolic filling (E peak) and late filling atrial contraction (A peak). The normal peak mitral inflow velocity is less than 1,3 m/sec. In mitral stenosis, the peak is usually increased to 1,5 to 3,0 m/sec. The rate of left ventricular filling decreases as reflected by reduced downslope of the E wave. Quantification of the Doppler flow pattern in mitral stenosis is also made by the "pressure half time" which is the time needed for the initial diastolic gradient to decrease by one half. The pressure half time correlates with the valve orifice area. Colour flow Doppler imaging has been used to depict the width of the flow jet across the stenotic valve; width of the inflow jet has been correlated with the orifice area. Transoesophageal echocardiography can provide exquisite detail of the mitral valve morphology and demonstrate thrombus in the left atrium. Left ventriculography demonstrates doming and restricted leaflet motion and a narrowed stream of nonopacified blood flow ("wash in" jet) into the opacified left ventricle during diastole. The thickened and fused chords of the valve may be shown as lucent extensions of the papillary muscle towards the valve leaflets. This finding indicates the likelihood of significant subvalvular obstruction as well as valvular stenosis. Abnormal motion of the anterior leaflet of the mitral valve may be assessed also in the left anterior oblique view. The normal valve displays biphasic motion in diastole with opening towards the left ventricle in early diastole followed by a drift back toward the annulus and then a second presystolic opening toward the left ventricle with atrial systole. In mitral stenosis, the motion is continuously toward the left ventricle in diastole because a pressure gradient exists between the left atrium and ventricle throughout diastole.

Many patients with predominant mitral stenosis also have some degree of mitral regurgitation revealed by left ventriculography.

Cine MRI shows a signal void caused by the flow jet across the stenotic mitral valve. It may also reveal the signal void caused by associated mitral regurgitation. This imaging sequence usually demonstrates normal left ventricular size and contraction. Highly accurate and reproducible measurement of left atrial and ventricular volumes are provided from cine gradient MR images encompassing the entire heart. Velocity-encoded cine gradient echo image acquired perpendicular to the direction of flow across the valve orifice can be used to measure the peak flow velocity and enable estimation of the pressure gradient. MRI are effective for demonstrating left atrial thrombus.

Aortic stenosis, narrowing of the valve between the left ventricle and the ascending aorta causing a pressure gradient during systole. It is usually caused by limitation of motion of the aortic valve cusps (valvular aortic stenosis) but can also

occur in the aorta within a few cm of the valve, supravalvular aortic stenosis or beneath the valve, subvalvular aortic stenosis. Commonly, aortic stenosis and aortic regurgitation coexist but one of the lesions is usually dominant. Left ventricular systolic pressure is elevated. Left ventricular wall stress is frequently increased; left ventricular hypertrophy tends to equalize wall stress even in the presence of considerable increase in left ventricular systolic pressure during the compensated state. Inadequate hypertrophy and myocardial failure in advanced disease is associated with marked increase in wall stress, left ventricular dilatation and eventually subendocardial myocardial ischaemia. The causes of valvular stenosis include congenital abnormalities such as bicuspid and unicuspid valves and deformed tricuspid valves. Acquired abnormalities include rheumatic fever and degenerative scarring and calcification.

Plain radiography varies from entirely normal to severe cardiomegaly and pulmonary oedema. There is usually mild or no cardiomegaly and no evidence of pulmonary venous hypertension. The most frequent feature of the plain radiograph is dilatation of the ascending aorta (poststenotic dilatation); aortic enlargement does not usually involve the arch or descending aorta. Aortic valvular calcification bears a rough relationship to the severity of valvular stenosis in patients under 60 years of age. Calcification is readily identified on fluoroscopy but only dense calcification is recognized on plain radiography. Ascending aortography demonstrates restriction of systolic opening (doming) of the thickened aortic valve and a jet of unopacified blood entering the opacified ascending aorta. The aortogram also reveals the extent of dilatation of the ascending aorta. It also displays any diastolic reflux of contrast media into the left ventricle due to associated aortic regurgitation. The severity of valvular aortic stenosis cannot be accurately judged from angiography but rather is reflected by the pressure gradient measured across the valve. Left ventriculography displays the limitation of excursion and thickening of the valve in valvular stenosis. Left ventriculography typically shows normal to slightly reduced left ventricular volumes and increased ejection fraction. Left ventricular wall thickness and myocardial mass are increased (fig. 7.22).

Echocardiography, two-dimensional and Doppler, is the most frequently employed modality for the diagnosis and assessment of severity of aortic stenosis. Colour flow mapping displays the high velocity jet across the valve. It also demonstrates the presence of associated regurgitation. Doppler sampling of the velocity of flow across the aortic valve is used to estimate the severity of aortic stenosis employing the modified Bernoulli equation (fig. 7. 23).

Magnetic resonance imaging (MRI) and computed tomography (CT) can demonstrate the precise dimensions of the dilated ascending aorta. This is useful in

monitoring aortic size in patients who develop aneurysmal dilatation as a complication of aortic stenosis. Although not generally used in the evaluation of aortic stenosis, cine MRI can define the high velocity jet across the aortic valve. Velocity-encoded cine MRI has been effective for measuring the peak velocity and pressure gradient using the modified Bernoulli equation. Cine MRI is a precise method for quantifying left ventricular volumes and myocardial mass in aortic stenosis.

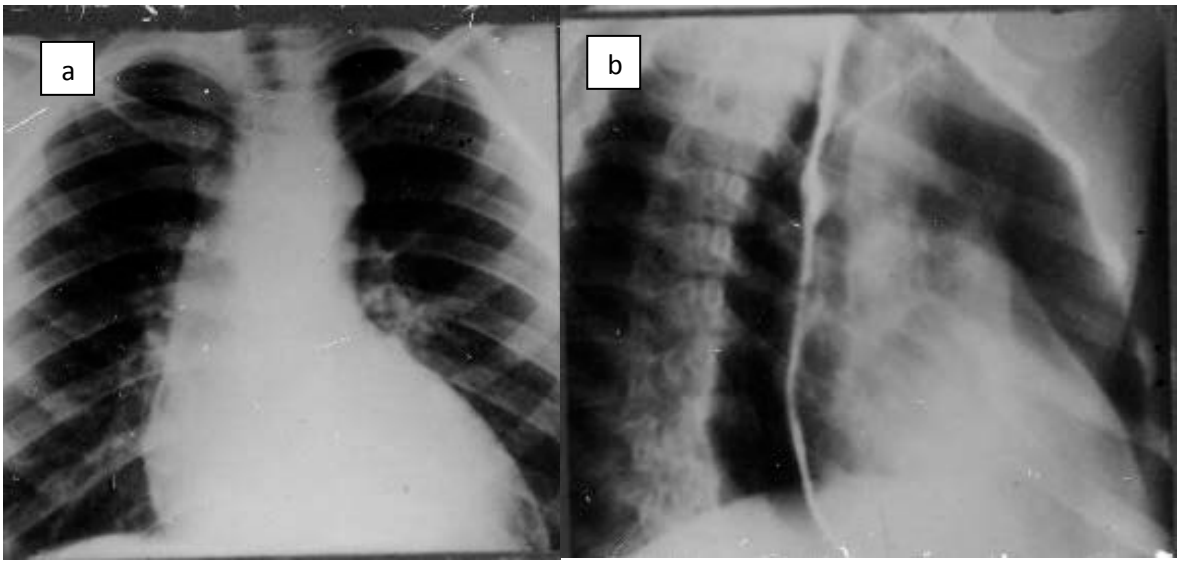

Fig. 7.22. Heart radiographs in a direct (a), right (first) oblique projection (b); a) aortic configuration of the heart. Arches of the ascending aorta and left ventriculus in a direct projection are increased); b) in the right (first) oblique projection the bottom arch of a forward contour is formed left ventriculus is increased. Aortic stenosis [31]

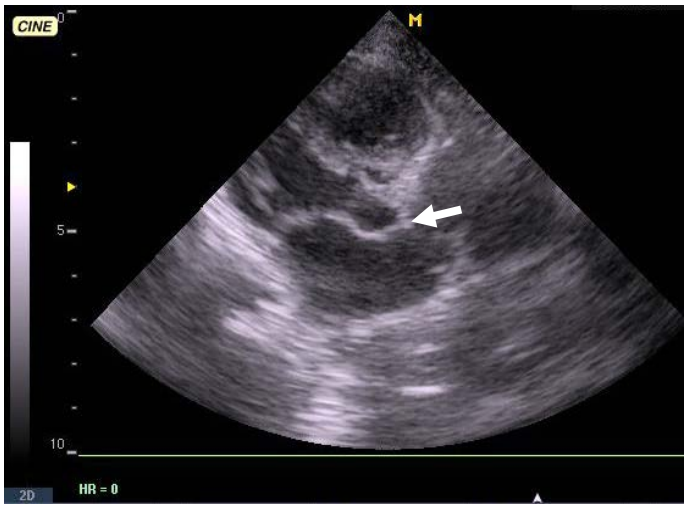

Fig. 7.23. Two-dimensional echocardiography (research from apex position). Aortic stenosis: fibrosis and thickening of leaflets of aortic valve (arrow)

Aortic regurgitation, retrograde flow across the closed aortic valve during diastole. It is caused by abnormalities of one or more of the following structures:

aortic cusp, aortic annulus, aortic sinuses. It is invariably associated with dilatation or aneurysm of the ascending aorta. There are many causes of aortic regurgitation including rheumatic heart disease, infective endocarditis, bicuspid aortic valve, aortoannular ectasia, aortic dissection and aneurysm, and several systemic diseases. The systemic diseases in which aortic regurgitation may be a manifestation are: ankylosing spondylitis, rheumatoid arthritis, Reiter's syndrome, giant cell aortitis, psoriatic arthritis, relapsing polychondritis, syphilis. Mild or moderate aortic regurgitation is frequent in patients with long-standing systemic hypertension. It may also be caused by trauma and radiation therapy. Aortic regurgitation is a frequent complication of valvuloplasty of aortic stenosis. Degeneration or infection of prosthetic valves causes aortic regurgitation. Most aetiologies produce chronic aortic regurgitation. Acute severe regurgitation may be caused by infective endocarditis, trauma and aortic dissection.

Radiography shows cardiomegaly due predominantly to left ventricular enlargement (fig. 7.24).

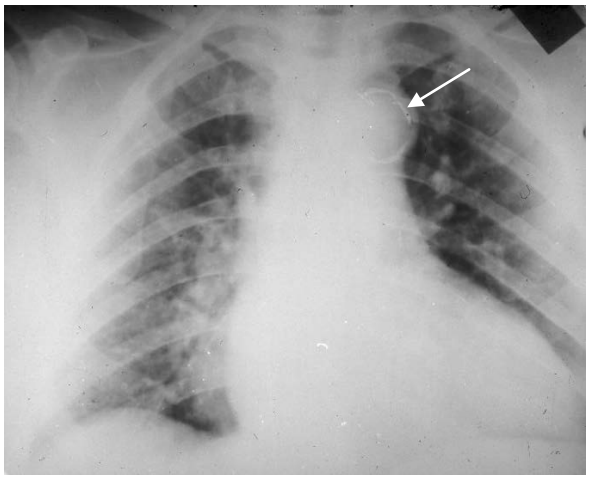

Fig. 7.24. The frontal chest radiograph.
The first and fourth arches on the left contour are increased. Calcification of aorta arch (arrow). Aortic configuration of the heart

Typically, enlargement of the ascending arch and descending thoracic aorta is evident.

For most of the course of chronic aortic regurgitation there is no pulmonary venous hypertension or pulmonary oedema. On the other hand, acute aortic regurgitation not uncommonly causes severe pulmonary oedema and little or no cardiomegaly.

Angiography demonstrates the presence and severity of aortic regurgitation consisting of retrograde diastolic flow of opacified blood into the left ventricle. The density of opacification of the left ventricle relative to opacification of the aorta is used as a semiquantitative method for grading the severity of aortic regurgitation.

Echocardiography, either transthoracic or transoesophageal, is the most frequently employed technique for the diagnosis and assessment of the severity of

aortic regurgitation. Doppler colour flow mapping is highly sensitive for identifying aortic regurgitation but provides only a semiquantitative assessment of severity. Echocardiography is very effective for demonstrating vegetations on the aortic valve indicative of infective endocarditis. Transoesophageal echocardiography is the preferred noninvasive technique for the evaluation of regurgitation of prosthetic aortic valves including suspected infective endocarditis. Echocardiography is used to monitor increases in left ventricular dimensions, volumes and ejection fraction in order to define the severity of regurgitation and as a guide to timing of valve replacement (fig. 7.25).

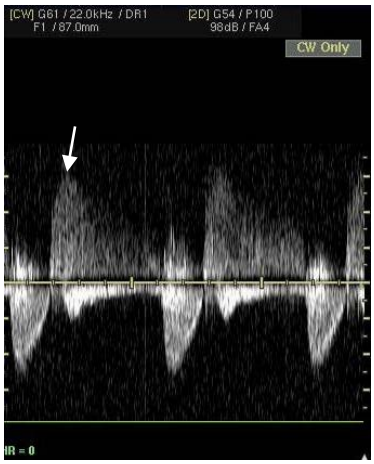

Fig. 7.25. Research from apex position**:** doppler echocardiography (continuous wave Doppler). Aortic regurgitation (arrow). Aortic insufficiency

Magnetic resonance imaging is highly accurate for demonstrating the presence of aortic regurgitation. On cine gradient echo images the regurgitant jet is displayed as a signal void emanating from the closed aortic valves into the left ventricle during diastole. The size of this signal void serves as a semiquantitative estimate of the volume of aortic regurgitation. Magnetic resonance imaging is the optimal technique for detecting abnormalities of the aortic sinuses, annulus, and ascending aorta associated with aortic regurgitation. It is also the preferred method for monitoring the dimension of the sinus and ascending aorta in patients with aortoannular ectasia as the cause of aortic regurgitation.

Coronary artery disease. Echocardiograms show contraction function infringement of separate sites of the left ventriculus wall in the form of reduction of amplitude of movement and myocardium thickening in systole; decrease in fraction of left ventriculus emission. Stress echocardiogram may be performed to further evaluate abnormal findings from an exercise treadmill test or a routine echocardiogram. Examples include identifying exactly which part of the heart may be involved and quantifying how much muscle has been damaged. It may be the first test done when the exercise treadmill test cannot be performed due to certain abnormal rhythms.

Radionuclide imaging is useful for diagnosing and determining:

- severity of unstable angina when less expensive diagnostic approaches are unavailable or unreliable;
- severity of chronic coronary artery disease;
- success of surgeries for coronary artery disease;
- whether a heart attack has occurred.

Myocardial perfusion (blood flow) test show presence of sites of a myocardium with accumulation reduction of radiofarmaceutical. This radionuclide test is typically used with an exercise stress test to determine blood flow to the heart muscles. It is a reliable measure of severe heart events. It may be useful in determining the need for angiography if CT scans have detected calcification in the arteries. About a minute before the patient is ready to stop exercising, the doctor administers a radioactive tracer into the intravenous line. Immediately afterwards, the patient lies down for a heart scan. If the scan detects damage, more images are taken 3 or 4 hours later. Damage due to a prior heart attack will persist when the heart scan is repeated. Injury caused by angina, however, will have resolved by that time.

Angiography is an invasive test. It is used for patients who show strong evidence for severe obstruction on stress and other tests, and for patients with acute coronary syndrome. It is required when there is a need to know the exact anatomy and disease present within the coronary arteries. A limitation of angiography is that it is not always the most occluded (blocked) blood vessel that causes the next heart attack. In an angiography procedure:

- a narrow tube is inserted into an artery, usually in the leg or arm, and then threaded up through the body to the coronary arteries;
- a dye is injected into the tube, and an x-ray records the flow of dye through the arteries;
- this process provides a map of the coronary circulation, revealing any blocked area.

Magnetic resonance angiography (MRA). MRA can provide three-dimensional images of the major arteries to the heart.

Computed tomography (CT) scans may be used to evaluate coronary artery disease.

Calcium scoring CT scans of the heart. May be used to detect calcium deposits on the arterial walls. The presence of calcium correlates well with the presence of atherosclerosis of the heart. If the calcium score is very low, a patient is unlikely to have coronary artery disease. A higher calcium score may indicate an increased risk of current and future coronary artery disease. However, the presence of calcium does

not necessarily signify narrowing of the arteries that would need further immediate evaluation or treatment.

CT angiography. CT scans are also used to visualize the coronary arteries. When compared to invasive angiography, CT angiography is not as accurate in identifying who truly has coronary artery disease and who does not. Other types of newer CT techniques include electron beam computed tomography and multidetector computed tomography.

Myocardial infarction, death of myocardial cells due to inadequate blood supply. The two main types are transmural and subendocardial infarction. Most myocardial infarctions result from atherosclerosis of the coronary arteries usually with superimposed thrombosis. Rarely, infarction can occur as a consequence of coronary arterial spasm, mural dissection, trauma or embolization. Infrequently, infarction occurs as a consequence of drastically increased myocardial oxygen demands causing imbalance in oxygen demand – supply ratios in diseases with severe left ventricular hypertrophy such as aortic stenosis and hypertrophic cardiomyopathy. Myocardial infarction most frequently occurs in the left ventricle; however, a substantial number of patients with inferior infarction have some infarction of the right ventricle also. Isolated infarction of the right ventricle is infrequent. The major pathophysiological consequences of acute myocardial infarction are diminished systolic function due to loss of functioning myocardium. With extensive infarction stroke volume and cardiac output are reduced; this may result in cardiogenic shock. Another consequence of acute infarction is diastolic dysfunction resulting in a decrease in ventricular compliance with elevation in left ventricular diastolic and pulmonary venous pressure. This may cause pulmonary oedema. The major complications of myocardial infarction are: heart failure, cardiac rupture, true left ventricular aneurysm, false (pseudo) aneurysm, acute mitral regurgitation from papillary muscle rupture, ventricular septal rupture (defect) and mural thrombus with or without peripheral embolization. Acute pericarditis may develop in some patients with transmural infarction (Dressler's syndrome).

Plain radiography is normal in about half of patients presenting with acute myocardial infarction. The most frequent abnormal finding is pulmonary venous hypertension or oedema without discernible cardiomegaly. The chest X-ray discloses pulmonary overcirculation and oedema in patients with ventricular septal rupture. A dramatic increase in cardiac size several days after infarction suggests pericardial effusion. Ventricular aneurysm is depicted as an abnormal bulge along the left ventricular margin. It is usually located in the anterior, lateral or apical region with true aneurysms. False (pseudo) aneurysms are usually larger and located on the posterior or diaphragmatic margin. Sudden onset or worsening of pulmonary oedema

occurs with papillary muscle rupture; pulmonary oedema confined to or worse in the right upper lobe is particularly characteristic.

Echocardiography demonstrates abnormal regional wall motion of the left ventricle in nearly all patients with acute infarction. A wall motion abnormality may not be evident in some patients with nontransmural infarction. Echocardiography can also be used to monitor remodelling of the ventricle after infarction and to follow end-diastolic and end-systolic size. Doppler and colour flow mapping echocardiography demonstrate mitral regurgitation and flail motion of a mitral leaflet due to papillary muscle dysfunction or rupture.

Radionuclide imaging using blood pool imaging demonstrates regional wall motion abnormality and in some instances reduced ejection fraction in acute infarction. Perfusion imaging at rest, employing thallium-201 or technetium-99m sestamibi demonstrates a perfusion deficit. Infarct avid tracers such as technetium-99m pyrophosphate show accumulation at the site of infarction (hot spot imaging). Perfusion imaging within the first 6 hours after onset of symptoms invariably demonstrates a perfusion defect but at a later time interval reperfusion may occur spontaneously so that a perfusion deficit is not evident. In patients who have successful therapeutic reperfusion of acute infarction ^{99m}Tc sestamibi perfusion imaging shows a decrease in size of the perfusion defect and can confirm the effectiveness of thrombolytic agents or acute catheter interventions.

CT and MRI have been employed to demonstrate complications of acute myocardial infarction. They show the presence, size and type of ventricular aneurysm. False aneurysms are characterized as large in size with a narrow ostium. CT and MRI are more accurate than echocardiography or contrast X-ray ventriculography for demonstrating mural thrombus. CT and cine MRI can be used to depict the regional wall motion abnormality and to quantify ventricular volumes. Magnetic resonance imaging shows increased signal intensity on T2-weighted images and greater contrast enhancement on T1-weighted images of the acutely infarcted myocardium compared with normal myocardium.

Left ventriculography is infrequently done in patients with acute infarction while coronary arteriography in recent years has been performed with increasing frequency in order to guide percutaneous transluminal interventional procedures. Left ventriculography documents a regional wall motion abnormality in acute infarction and is sometimes later used to evaluate complications of infarction.

Coronary arteriography usually shows total or near total occlusion of a coronary artery. The occlusion is usually due to acute thrombosis at the site of a nonobstructive or obstructive plaque in the coronary artery. The acute thrombosis is frequently not at the site of the most severe stenosis. Arteriography demonstrates

reperfusion of the vessel after thrombolysis and/or angioplasty.

Aneurysm, thoracic aorta, focal or diffuse dilatation of the thoracic aorta usually caused by degenerative diseases such as atherosclerosis. The normal diameter of the thoracic aorta is less than 4,0 cm for the ascending, and less than 3,0 cm for the descending portions. A diameter exceeding 5 cm is usually considered as aneurysm. A diameter of the aorta greater than 1,5 times of the normal diameter also constitutes aneurysm. Aneurysms may be fusiform (concentric radial dilatation) or saccular (eccentric radial dilatation). Atherosclerosis and cystic medial necrosis usually produce fusiform aneurysms while infections cause saccular aneurysms (mycotic aneurysm). True aneurysms have all three layers of the wall while false aneurysms consist only of media. Infection and trauma cause false aneurysms. Aneurysms may be caused by eccentric jet flow across a stenotic or nonstenotic bicuspid aortic valve or coarctation of the aorta.

Plain radiography demonstrates generalized or focal bulging of the aortic contour. Aneurysm of the ascending aorta causes enlargement of the right superior mediastinum and obliteration of the retrosternal air space (fig. 7.26).

The aneurysmal contour not uncommonly is calcified. Definition of the diameter and extent of aneurysm can be provided by aortography, CT, MRI and magnetic resonance angiography (MRA). Currently, the preferred imaging modalities for initial diagnosis and monitoring of the diameter are MRI, MR angiography and spiral or electron beam CT. These modes allow to estimate the form, diameter, extent, a status of surrounding tissues, presence clot of blood, wall stratification of aorta.

Aneurysm, abdominal aorta. A diameter exceeding 2,0 cm is considered aneurysmal while a diameter over 4,5–5,0 cm indicates the need for early surgery. Most are fusiform (concentric radial dilatation) but infrequently may be saccular (eccentric radial dilatation). Plain radiography may suggest the presence and size of the aneurysm by showing aortic calcification. Initial diagnosis and monitoring of maximum diameter is now usually done by ultrasonography (fig. 7.27).

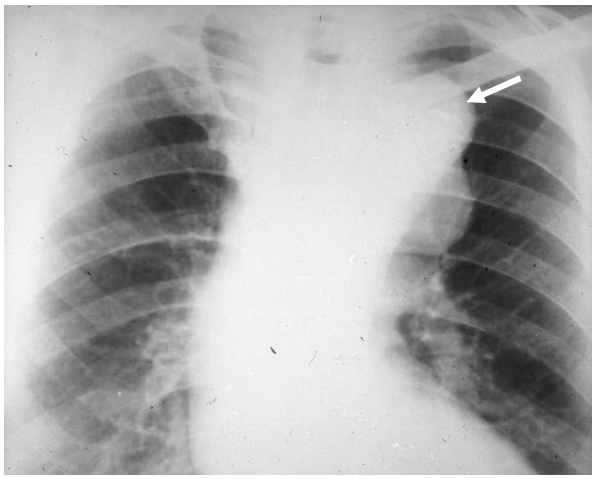

Fig. 7.26. The frontal chest radiograph. The size of the aorta arch is sharply increased (arrow). Aneurysm of the aorta arch

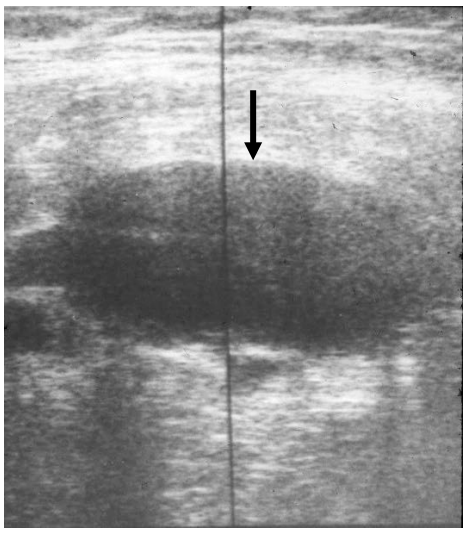

Fig. 7.27. Ultrasound examination of abdomen cavity. Sharply increased abdomen partof the aorta (arrow). Abdominal aortic aneurysm [32]

Preoperative evaluation is performed by either x-ray angiography (fig. 7.28), CT angiography or MR angiography.

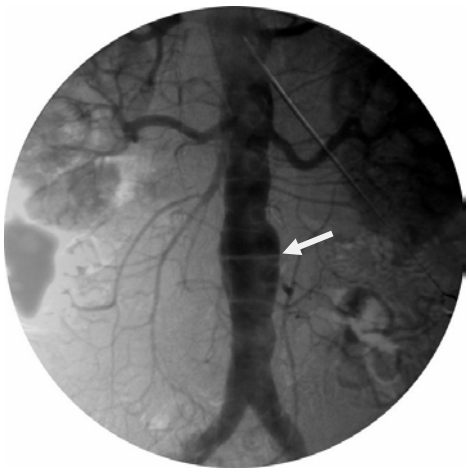

Fig. 7.28. Digital aortography. Abdominal aortic aneurysm: local expansion of an aorta (arrow)

Each of these studies can define the relationship of the aneurysm to the renal, mesenteric, and iliac arteries and exclude significant stenosis of these arteries (fig. 7.29).

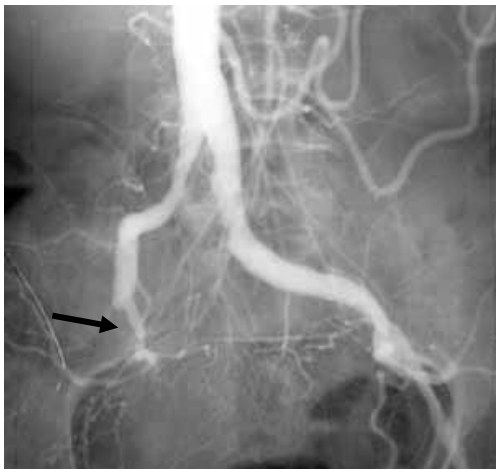

Fig. 7.29. Aortography. Embolic occlusion of the right iliac artery (arrow)

MRI and CT are more accurate for displaying the maximum diameter and mural hrombus. The major complication is rupture with retroperitoneal haemorrhage.