CARDIAC IMAGING

Cardiovascular radiology is a subspecialty shared by radiologists and cardiologists. Too often the clinician is content to merely have made a diagnosis of "large heart," "congestive heart failure," or "congenital heart disease." It is possible, however, for you as the clinician to recognize certain patterns of disease based on the alterations those diseases produce in the pulmonary vascularity and in specific chambers of the heart. The evaluation of the patient with suspected cardiac disease should be directed along two distinct lines: imaging of anatomic changes and demonstration of physiologic function, often simultaneously. While the former goal can be attained through plain radiographic means, the latter may be achieved only through cardiac catheterization, which permits hemodynamic measurements and injection of contrast material. As will be discussed below, recent developments in imaging technology have now made it possible to make accurate diagnoses using noninvasive techniques.

Technical considerations

The advances in diagnostic imaging techniques in the past 2 decades have revolutionized cardiac imaging. Plain film radiography, cardiac series, cardiac fluoroscopy, and cardiac catheterization were the mainstays of cardiac imaging prior to 1975. However, real-time ultrasound imaging, radionuclide cardiac perfusion studies, computerized tomography (CT), and magnetic resonance imaging (MR) have all become standard evaluation tools in the hands of the cardiovascular radiologist and cardiologist in the 1990s. As with any other organ system, your choice of diagnostic studies will depend on getting the most information in the safest way at the lowest cost. Therefore, consulting with your radiologist or referring cardiologist is mandatory.

The same plain film technical considerations that were discussed for pulmonary disease apply to evaluation of the cardiovascular system. The degree of penetration on the film, the presence of motion, and the degree of inspiration are all important factors to consider. A film that is too light will accentuate the pulmonary vessels: a film with the patient not in maximum inspiration may result in further accentuation of pulmonary vessels and cause an appearance of cardiac enlargement and/or an erroneous diagnosis of congestive heart failure.

It is also necessary to pay close attention to the patient's body habitus. In the bony thorax, particular attention should be given to the under-surfaces of the ribs for any evidence of rib notching. Although the most common cause of rib notching is coarctation of the aorta, you should keep in mind that many other conditions such as tetralogy of Fallot, truncus arteriosus, or neurofibromatosis produce this abnormality.

There are eight basic techniques used for evaluation of the heart: (1) plain film radiography, (2) cardiac fluoroscopy, (3) cardiac series, (4) cardiac catheterization and coronary arteriography (angiocardiography), (5) echocardiography, (6) radioisotope studies, (7) computerized tomography, and (8) magnetic resonance imaging.

Plain film radiography is a standard screening examination in patients with suspected cardiac disease. By knowing the normal anatomy portrayed on the anterior-posterior (posterior-anterior) and lateral films and by analyzing the sizes of pulmonary arteries and veins, you may be able to make a correct diagnosis in the majority of cases.

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 an 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. 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. The anatomic relationships will be discussed in the next section. The cardiac series, too, has largely been replaced by echocardiography.

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. Real-time echocardiography has decreased the number of catheterizations used to determine cardiac chamber size and configuration.

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

Magnetic resonance imaging is 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. Areas which show persistent metabolism (FDG uptake) but poor perfusion are identified as hibernating myocardium. Thallium-201 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.

Echocardiography

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.

Types of echocardiographic studies

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 a 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.

Echocardiography is an ultrasound examination of the heart and great vessels using one of three techniques: M-mode, cross-sectional (two-dimensional) imaging, or Doppler technique. M-mode (motion-mode) echocardiography records echoes from cardiac structures within the ultrasonic beam and provides one-dimensional information. It is displayed on a strip chart that allows measurements to be made of the depth of the structure as well as its motion.

Cross-sectional echocardiography provides images of the moving heart chambers. By shifting the transducer and altering the depth of penetration of the ultrasound beam, one can obtain a tomographic image of

the heart and its chambers.

Doppler echocardiography is used primarily to assess the direction and velocity of blood flow within the heart and great vessels. It is particularly useful in evaluating the carotid vessels in patients with transient ischemic attacks.

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.

Nuclear myocardial perfusion imaging

Nuclear myocardial perfusion imaging, 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 (Tl-201) and technetium-99m (Tc-99m) labelled agents. The initial distribution of Tl-201 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 Tl-201 during stress, and rest images are done about four hours later. Disappearance of intial perfusion defects on delayed images indicates redistribution of the agent caused by slower clearance of Tl-201 from underperfused compared to normally perfused regions. Tc-99m-labelled perfusion agents such as Tc-99m sestamibi and Tc-99m teboroxine have been used in recent years because of better imaging properties compared to Tl-201. Because Tc-99m 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 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.

Myocardial perfusion imaging

Myocardial perfusion imaging, techniques with which myocardial perfusion is assessed at rest or under stress conditions. The most widely used myocardial perfusion imaging methods are based on nuclear imaging techniques. Then perfusion imaging is based on the wash-in principle of "chemical" microspheres, with the radioactive tracer sticking to the myocytes in proportion to the regional tissue perfusion. The agents in use are thallium-201, technetium-99m MIBI, technetium-99m tetrofosmine and N-13 ammonia in PET imaging. Perfusion measurements based on bolus tracking methods are currently being evaluated in MR imaging, but the same methods were used in conjunction with radioactive tracers in the late 1970s and 1980s, and were abandoned due to their complexity. Some perfusion information can also be obtained with microbubbles in echocardiography.

Technetium-99m MIBI

(Tc-MIBI) (technetium-99m isonitrile [TE]) scintigraphy is the most widely used myocardial perfusion imaging technique. In this method, the patient is first subject to ergometric or pharmacological stress (dipyridamole or adenosine) which is terminated using standard criteria. Just before termination, approximately 800 MBq of Tc-MIBI is injected. Its myocardial uptake represents the stressed perfusion state as the agent sticks to the myocardium in proportion to the instantaneous regional perfusion. The patient is then asked to wait for approximately 60 minutes, drinking some milk after about 30 minutes which permits the biliary excretion of excess agent, preventing interference of high liver activity with myocardial activity during subsequent

scanning. Scanning is typically done using SPECT imaging techniques, recently in conjunction with simultaneous acquisition of transmission data, so as to correct for variable absorption in the chest. The SPECT images are reformatted so that they represent images perpendicular to the long and short axes of the heart. The injection of Tc-MIBI and scanning is repeated the next day at rest, and the stress and rest scans are compared regarding discordant or concordant activity deficits. A discordant deficit represents a region of ischaemia, while a concordant deficit is either an infarct or represents a region of hibernating myocardium.

Thallium myocardial perfusion imaging

The technique and image interpretation are similar to those described for Tc-MIBI studies. Tl as its K analogue, however, redistributes relatively rapidly. Hence, the stress study has to be performed quickly after the injection of approximately 80 MBq of Tl-201. The patient then is sent away for 4 - 5 hours during which Tl redistributes and eventually shows the rest perfusion pattern, which is recorded. Tl is reduced in concentration in myocardial areas of ischaemia because of both a reduction in perfusion and a reduction in function of the Na-K-pump. This permits Tl to be used as a marker for the identification of hibernating myocardium.

N-13 ammonia perfusion imaging

Uses the short lived N-13 isotope (9 minutes). Approximately 800-1 200 MBq of N-13 ammonia is injected under the PET camera. It is quantitatively incorporated into the cellular nitrogen pool upon first pass (transformation of glutamate to glutamine via the enzyme glutamate synthetase). A rest study is initially performed. Stress is achieved by the use of a pharmacological stress protocol, as treadmill exercises in the PET scanner are impractical. Data acquisition for stress can typically occur 30 minutes after the rest study because after 3 half-lives the resting activity has virtually disappeared. Dynamic data acquisition permits the quantitative assessment of myocardial perfusion and since the sizeable blood volume in the heart chambers is imaged, arterial input sampling is not necessary.

While the sensitivities of Tc-MIBI and Tl studies are in the low 80 % range, that of N-13 ammonia is above 90 %.Myocardial perfusion imaging in MR imaging is still experimental. Because there are no MR contrast agents which localize in to the myocardium in proportion to its perfusion, bolus tracking methods have to be used which are much more difficult to perform and evaluate than the microsphere wash-in methods used in nuclear imaging. Whether the better spatial resolution of MR imaging will outweigh the benefits of wash-in imaging in nuclear imaging remains to be seen.

Radioisotope studies of the heart are performed primarily for the evaluation of cardiac perfusion. Isotopes of thallium are injected intravenously and the myocardial blood flow is recorded with the patient at rest and while exercising (thallium stress test). This technique is most useful for separation of patients with atypical chest pain into cardiac and noncardiac origin categories.

Cardiac magnetic resonance imaging

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 spin echo images 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.

Cardiac MR imaging techniques

Multislice electrocardiographically gated spin echo imaging acquires multiple adjacent images through the heart with a repetition time (TR) equal to the RR interval of the electrocardiogram and a short echo delay time (TE = approximately 15–25 msec). One tomogram is acquired at each anatomical level and each adjacent slice is acquired at a slightly later time in the cardiac cycle. These images are used for evaluating morphology. ECG-referenced multiphasic gradient echo (GRE) images (cine GRE imaging) acquires multiple images (frequently n = 16) evenly spaced throughout the cardiac cycle at each anatomical level. Retrospective electrocardiographic gating is used. This technique is used to evaluate cardiac morphology and function and to display abnormal blood flow, usually jet flow, such as occurs with valvular stenosis or regurgitation.

Cine MR imaging, also called breathhold cine MR imaging, provides a set of GRE images at multiple phases of the cardiac cycle at each anatomical level in a short acquisition time (about 13–18 secs). Fast or snapshot GRE images, which acquires sequential images at one to two second intervals during the first passage if MR contrast

media through the cardiac chambers is used to evaluate myocardial perfusion. This sequence is done at baseline and after administration of a vasodilator in order to detect regional myocardial ischaemia.

Velocity-encoded cine MR imaging or flow-velocity mapping is a flow-sensitive technique which provides phase contrast images at multiple phases of the cardiac cycle. From the phase contrast images flow velocity can be measured at specified sites.

Echoplanar imaging (EPI) is the fastest MR imaging technique providing images in 40–50 msec. This technique can be used to evaluate ventricular function and myocardial perfusion.

Anatomic considerations

For an appreciation for 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 roentgenogram of a chest (heart) in a direct projection. In a direct projection on the right contour - two arches. Bottom it is formed by the right atrium, top an ascending aorta (sometimes vena cava superior). Between arches there is a corner named right atriovascular the corner. The left contour of a cardiovascular shadow is displayed as four arches. Top it is formed by a shadow of an arch and the beginning of a descending part of an aorta. The arch second from above - a trunk and the left branch pulmonary artery, (nonconstantly) the arch the left atrium below settles down. Closes the left contour of heart an arch left ventricle. The corner between an arch the left atrium and an arch pulmonary artery refers to left atriovascular a corner. The distance from an arch left ventricle up to left medial clavicle lines should make not less than 1,5-2 cm, and from an average line up to the most acting point of the top arch of the left contour - 3-4 cm. The top contour of a cardiovascular shadow will defend from the horizontal line connecting brest bone-clavicle joints, on 1,5-2 cm. Lengths of arches of a trunk pulmonary artery and the left auricle should be about 2 cm everyone. The height of an oval of actually heart shadow is equal norm an oval of a vascular shadow. Structure of a shadow of normal heart and large vessels usually homogeneous, without additional inclusions.

In the first slanting projection (under a corner 45^{o} to the screen the right side) a forward contour from below is submitted left ventricle, the second reflects the department right ventricle (pulmonary outflow tract). The arch generated by the ascending department and the beginning of an arch of an aorta lieis theirs above. The arch of the right atrium from below enters into a back contour, there is an arch of the left atrium above. A condition of the left atrium judge on position an esophagus with contrast agent. In norm the shadow of an esophagus at a level of an atrium is located as the crow flies. The increase in the sizes of an atrium conducts to a pushing off of an esophagus. Allocate a pushing off of an esophagus on an arch of the big and small radiuses. The arch of the big radius is more, and small it is less 6 cm.

In the second slanting projection (under a corner 45o to the screen the left side) heart and vessels also have two contours - forward and back. The forward contour is formed from below right ventricle, the arch of the right atrium is above. The uppermost arch is formed by an ascending aorta. A back contour make: from below - the arch left ventricle, is higher - an arch of the left atrium.

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. 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. 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.

The form of heart and large vessels.

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

Mitral the form of heart is allocated with the following attributes. The length and camber of the arches formed by a trunk and the left atrium increase. Decreases left atriovascular the corner, right atriovascular the corner is displaced upwards.

Aortical the form. The following attributes are characteristic:

1. Emphaticalness of a waist (expressiveness, dredging on the left contour between an arch of an aorta and an arch left ventricle, therefore the distance between atriovascular corners seems small).
2. Increase in the arch formed left ventricle.
3. Lengthening an arch and expansion of a shadow in a projection of an ascending aorta (the right contour, the top arch).

4. Lengthening the top arch of the left contour generated by an arch and the descending part of an aorta.
5. Displacement downwards right atriovascular a corner.

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

Pathologic considerations

There are many ways to classify cardiac disease. A popular classification uses two large categories, congenital and acquired cardiac disease. Congenital cardiac disease is further subdivided into cyanotic and acyanotic types. Most books on cardiology prefer this method. For the non-cardiologist, a physiologic approach affords an understandable and useful basis for dealing with congenital and acquired heart disease. In addition to this physiologic approach, it is preferable to evaluate patients with cardiac disease on the basis of their age. This discussion will focus on the plain film evaluation of these patients, since that is the type of imaging examination you will order first and with which you will be most familiar. Once you have an idea of what kind of cardiac disease you are dealing with you can order more sophisticated imaging procedures, such as echocardio-graphy, angiography, or MR, to make a definitive diagnosis.

Adult Patients

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. Admixture

C. 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, 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. Pulmonary vascularity may be normal, decreased, or increased. 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 cardiomyo-pathy, 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 (Wester-mark sign), emphysema, or scarring with rearrangement of vessels in a lung. A unilateral decrease in vascularity without changes in the cardiac size may result from either hypoplasia of a lung or the Swyer-James syndrome, a rare condition caused by diffuse, unilateral bronchitis.

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. This indicates increased left atrial pressure. Severe PVO is manifest 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.

Pediatric Patients

Cardiac disease in pediatric patients is usually congenital. However, rheumatic heart disease is an important form of acquired disease that may occur in this age group.

Before beginning an analysis of the pulmonary vascularity in pediatric patients, it is important to know whether or not they are visibly cyanotic. The presence of visible cyanosis changes the physiologic state of the patient and the category of disease. It is also important to know whether the cyanosis was present at birth (as in transposition of great vessels) or developed later (as in tetralogy of Fallot). Plain film analysis will be discussed in the acyanotic and the cyanotic patient.

Furthermore, edema may be present from another (noncardiac) etiology such as heroin intoxication, inhalation of noxious fumes, or drowning. In these conditions, the heart is usually normal in size.

Pleural fluid is another nonspecific finding that may be present in patients with congestive heart failure. If the fluid collects along a fissure, a pseudotumor may result. In the lateral view, the borders are generally tapering, and the collection of fluid is oriented in a slanted configuration. These densities disappear after successful therapy.

Pericardial Effusion

Pericardial effusion must always be considered when evaluating a patient with an 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 engorgement, should suggest a pericardial effusion. Occasionally, the pericardium will be demonstrated in normal patients as a thin, dense line separated by layers of subepicardial and mediastinal fat. In patients with pericardial effusion, this line, which never should measure more than 2 mm, is thickened.

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 a cardiac arrhythmia, a scarred myocardium, or an 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. Ultrasonic shadows reflected off the pericardial and myocardial surfaces will demonstrate an abnormal collection of fluid in the pericardial sac.

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.

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/uzzy. You should remember, however, that a supine radiograph in a large patient may simulate this appearance. With this in mind, you should make every effort to obtain an erect film. When this fails, and 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, ischaemic papillary muscle rupture or traumatic papillary muscle rupture, degenerative cordal rupture, mitral valve prolapse, mitral annular calcification, and congenital lesions such as parachute mitral valve and atrioventricular septal defect.

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.

Imaging

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. Right-sided chamber enlargement may be caused by pulmonary arterial hypertension or concurrent tricuspid regurgitation. In the absence of associated aortic valve decrease, the ascending aorta is inconspicuous. The left atrial appendage is usually enlarged in rheumatic mitral regurgitation but may not be recognizable with nonrheumatic aetiologies. Acute mitral regurgitation such as occurs with ruptured papillary muscle may cause severe alveolar pulmonary oedema, sometimes with a normal heart size.

The M-mode echocardiogram shows abnormal mitral leaflet closure patterns depending upon the type of mitral regurgitation. These consist of incomplete closure, mitral valve prolapse, ruptured chordae tendineae or flail mitral valve. The flail valve shows erratic systolic motion into the left atrium.

Two-dimensional echocardiography (V:2) 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/m2 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. 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 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 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 of age. 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.

Imaging features

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 the stenosis is caused by rheumatic heart disease. In isolated mitral stenosis the left ventricle is not enlarged. 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.

Two-dimensional echocardiography shows thickened and relatively immobile leaflets, doming of the valve, chordal foreshortening and thickening. 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, employing the modified Bernoulli equation. 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. Spin-echo and gradient-echo imaging are effective for demonstrating left atrial thrombus.

Aortic stenosis

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.

Imaging

Plain radiography varies from entirely normal to severe cardiomegaly and pulmonary oedema. The radiograph of neonates with critical aortic stenosis shows pulmonary oedema or pulmonary venous hypertension and cardiomegaly. In both older children and adults 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.

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. The peak velocity across the stenotic valve recorded by Doppler echocardiography is used in the following formula: peak pressure gradient = 4 · peak velocity2. 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.

Myocardial infarction

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 [CV] , 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).

Imaging

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 99mTc sestamibi perfusion imaging shows a decrease in size of the perfusion defect and can confirm the effectiveness of thrombolytic agents or acute catheter interventions.

Computed tomography (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. Electron beam CTand 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 spin echo images and greater contrast enhancement on T1 spin echo images of the acutely infarcted myocardium compared with normal myocardium. Serial cine MRI studies can be used to monitor left ventricular remodelling after acute infarction.

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. Interestingly, 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.