THE CHEST RADIOGRAPH AND TWO-DIMENSIONAL ECHOCARDIOGRAPHY IN THE EVALUATION OF ACQUIRED HEART DISEASE

THESIS SUBMITTED IN PARTIAL FULFILMENT
FOR THE DEGREE OF M. D.
(RADIODIAGNOSIS)

BY

SALAH M. KORAYEM
(M. B. B. Ch.)
(M. Sc. RADIODIAGNOSIS)

SUPERVISED BY

PROFESSOR DR. MOHAMED HAFEZ SHERIF
PROFESSOR OF RADIODIAGNOSIS
AL AZHAR UNIVERSITY
CAIRO

DR. S. K. MORCOS
CONSULTANT RADIOLOGIST
LODGE MOOR HOSPITAL &
NORTHERN GENERAL HOSPITAL
SHEFFIELD

FACULTY OF MEDICINE
AL AZHAR UNIVERSITY

1988

ACKNOWLEDGEMENTS

I would like to acknowledge my very grateful appreciation and thanks to Professor Dr. Mohammed Hafez Sherif, Head of the Radiology Department of Al-Azhar University, for his invaluable time, help and guidance which he has given me over the years. I remain greatly indebted to him for his encouragement and support.

My deepest thanks and gratitude to Dr. Sameh K. Morcos, Consultant Radiologist, Lodge Moor Hospital and Northern General Hospital, Sheffield, England for his tremendous help and guidance throughout the preparation of my thesis. My thanks also for the hospitality he extended to me during my stay in Sheffield.

My sincere appreciation and thanks to Professor R.G. Grainger, Professor of Radiodiagnosis, Sheffield University, England for all the kindness, help and advice he has given me.

My thanks to the staff of the x-ray department at Lodge Moor Hospital for their kind help and cooperation.

My special thanks to the staff of the Medical Photography Department of the Northern General Hospital, Sheffield for the special quality of their work.

Last, but not least, I am indebted to Miss Susan Tingle for her extreme patience in the typing of this thesis and for the excellent quality of her work.

INDEX

Page

INTRODUCTION AND AIM OF WORK

Diagnostic ultrasound is playing an important and indispensable role in the management of many patients in a wide variety of clinical situations. Its impact in diagnostic imaging is increasing daily and new approaches are being embarked on to include almost every part of the human body.

Echocardiography is one of the very important applications of ultrasound and it is now playing an increasingly significant role in the evaluation and management of patients with all forms of cardiac disease. In several instances it provides sufficient diagnostic data to obviate the need for cardiac catheterization.

Echocardiography, which is now in wide clinical use, was introduced in 1953 and was first implemented by Hertz and Edler. Unfortunately it has been avoided by many radiologists because they believe it is difficult to perform and interpret and feel it is better to leave it to the cardiologists and to the radiologists with a special interest in this field.

This study presents a simple approach to two-dimensional echo-cardiography. The parasternal long axis, apical and subcostal 4 chambers are the only views used to examine the heart. The anatomy of these planes is simple and the technique is not difficult to perform. These planes are adequate to cover a wide range of acquired heart diseases. The only structure which cannot be directly visualized by this approach is the pulmonary valve, which is uncommonly affected in patients with acquired heart disease. The examination is carried out with real-time sector scanner which is available in many radiology departments. The 3.5 MgH probe is

optimal for this application. Doppler ultrasound or time-motion mode is not used in this approach.

The contribution of this practice to the management of patients in a general hospital is the main purpose of this study. The full potential of echocardiography in the assessment of heart disorders has not been explored in the patients of this study, nor has any attempt been made to present specialized or sophisticated knowledge of this technique.

The aim of this thesis is to present a simple approach to echocardiography, to discuss its value in a general hospital and to compare the echo findings with the information acquired from the chest radiograph.

The first part of this thesis covers the different aspects of cardiac imaging with special emphasis on echocardiography and plain chest radiography. A comprehensive review of the literature is also presented.

The remaining part includes materials and methods, results, case reports, discussion and abstract.

This work has been carried out from a generalised point of view and does not present a specialized experience in this field. It is believed that the two-dimensional echocardiographic examination which is presented in this study, using basic real-time sector scanning equipment, is very useful and is possible in any general radiology department with this facility.

It is hoped that this work will encourage more radiologists with experience in ultrasonography to start to implement this technique in their departments when assessing the hearts of patients with cardiac disorders. It is advised that complicated and difficult cases should be referred to specialized units in cardiology for further assessment.

NORMAL RADIOGRAPHIC ANATOMY

NORMAL RADIOGRAPHIC ANATOMY

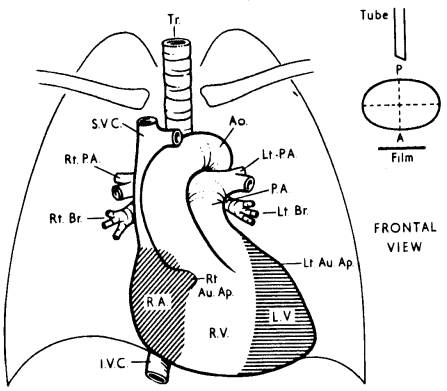
POSTERO-ANTERIOR CHEST FILM (Fig. 1):

The right upper mediastinal border is normally formed by the superior vena cava (s.v.c.) and medial to it lies the ascending aorta. As the ascending aorta dilates in older patients, the superior vena cava may be overshadowed by it. Where the superior vena cava joins the right atrium, just below the right hilum, there is a slight change in the contour of the heart shadow below it where it becomes more convex. This point indicates the upper limit of the right atrial border which then curves down to the diaphragm. The inferior vena cava can sometimes be seen as a small triangular shadow where the right atrium meets the right diaphragmatic dome.

The left upper mediastinal border is formed by the aortic knuckle; below this is the main pulmonary artery lying medial to the left hilum. Below the left hilum the left ventricular border forms the left cardiac contour, and the apex of the heart usually lies just above and lateral to the point where the left ventricular border meets the diaphragmatic dome.

In a small number of patients the tip of the left atrial appendage may form a small segment of the left cardiac border just below the main pulmonary artery segment from which it is separated by the left main bronchus. (Steiner, 1980).

Fig. 1



Diagrammatic representation of the appearance of the heart shadow and great vessels in the postero-anterior position

Abbreviations

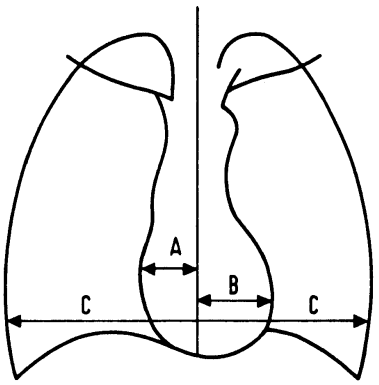
S.V.C.	=	Superior Vena Cava
Rt. P.A.	=	Right Pulmonary Artery
Rt. Br.	=	Right Bronchus
R.A.	=	Right Atrium
I.V.C.	=	Inferior Vena Cava
Tr.	=	Trachea
Ao.	=	Aorta
P.A.	=	Main Pulmonary Artery
Lt. P.A.	=	Left Pulmonary Artery
Lt. Br.	=	Left Bronchus
L.V.	=	Left Ventricle
Lt. Au. Ap.	=	Left Atrial Appendage
Rt. Au. Ap.	=	Right Atrial Appendage
R.V.	=	Right Ventricle

THE HEART SIZE (Fig. 2):

Heart size can be expressed as the ratio of the maximum transverse diameter of the heart to the maximum transverse diameter of the thoracic cage (the cardio-thoracic ratio). When the thoracic diameter is measured, it is of importance to take the end points at the edge of the internal chest wall.

The maximum transverse diameter of the heart is the summation of the maximum distances of the right and left cardiac borders from the mid line. These measurements are assessed at different levels as the maximum transverse diameter of the heart is rarely horizontal.

Fig. 2



Diagrammatic representation of the measurement of the cardio-thoracic ratio

A + B = maximum transverse diameter of the heart

C = maximum transverse diameter of the chest

In adolescents and adults a cardio-thoracic ratio below 1:2 or 50% is considered normal on an adequate inspiratory postero-anterior chest film.

In small children this ratio does not apply and is often greater. (Steiner, 1980).

NORMAL VARIATION IN HEART SIZE

There is considerable variation in heart size with the patient's age and body build and to a lesser extent with sex.

(a) In small infants the heart shadow may appear much larger than the normal cardio-thoracic ratio even when the film is taken on deep inspiration.

(b) Similarly, in the very old, the heart shadow may be larger than that of patients of similar build in the younger age group.

(c) In plump adults with a high diaphragm and a short vertical chest dimension, the cardio-thoracic ratio may be greater than 50%.

(d) In patients with a very long, narrow chest a small cardiac silhouette is common, and this may also apply to the patient with very large volume lungs and a low position of the diaphragmatic dome.

(e) In athletes the heart shadow may be a little larger than normal.

(f) A physiological difference in heart size on films taken in systole and diastole can sometimes be seen.

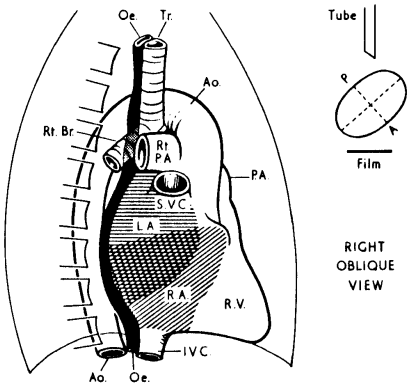
RIGHT ANTERIOR OBLIQUE PROJECTION

FIRST ANTERIOR OBLIQUE PROJECTION (Fig. 3):

Anteriorly the right ventricle forms the cardiac borders and merges above into the main pulmonary artery, which curves backwards until it divides into the right and left main branches. Behind the main pulmonary artery lies the ascending aorta which crosses the pulmonary artery and extends up into the mediastinum to form the aortic arch, which is foreshortened in this projection.

Posteriorly the heart shadow is formed by the left atrium above and by the right atrium below. The I.V.C. is sometimes seen to enter the right atrium posteriorly, forming a small triangular shadow just above the diaphragmatic dome. Enclosed within the concavity of the aortic arch - "the aortic window" lies the right main pulmonary artery, the carina and posteriorly the oesophagus. The barium filled oesophagus can be seen extending down in the posterior mediastinum; it is first impressed by the aortic knuckle and below by the pulmonary artery, and lastly by the left atrium. All these impressions are on the anterior aspect of the oesophagus.

Fig. 3



Diagrammatic representation of the heart shadow and great vessels in the right anterior oblique position

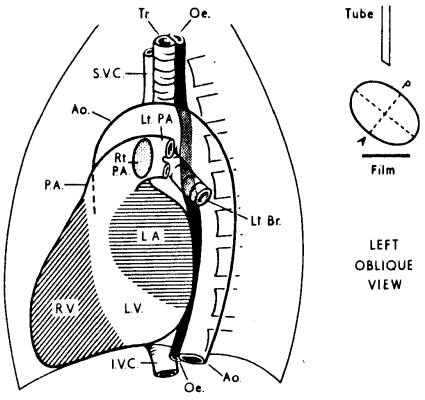
Oe. = Oesophagus

THE LEFT ANTERIOR OBLIQUE POSITION

THE SECOND ANTERIOR OBLIQUE POSITION (Fig. 4):

The anterior cardiac border is formed by the right ventricle, which merges into the pulmonary artery and aorta above. The pulmonary artery then slightly curves backwards and divides into the right and left main branches. The right main branch can appear as a distinct opacity. The ascending aorta is arising from the left ventricle within the cardiac shadow and lying posteriorly to the pulmonary artery. The ascending aorta becomes visible as it crosses the main pulmonary artery, curving upwards and slightly backwards to form the aortic arch. Contained within the arch concavity lies the "aortic window". The aortic arch, which is seen best in this projection, then curves posteriorly and descends, merging with the descending thoracic aorta, which is slightly overlapping the spine. The posterior cardiac border is formed by the left atrium above and below by the left ventricle.

Fig. 4

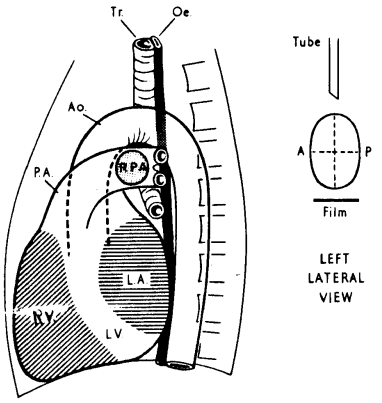


Diagrammatic representation of the heart shadow and great vessels in the left anterior oblique position

THE LATERAL POSITION

The right ventricle forms the anterior cardiac border, merging with the pulmonary artery above. The ascending aorta becomes visible where it crosses the pulmonary artery; it then curves backwards to form the aortic arch. The posterior cardiac border is formed by the left atrium above and below by the super-imposed right atrium and left ventricle. As in the oblique positions the oesophagus is seen in the posterior mediastinum in close proximity to the aortic arch, the pulmonary artery and right main bronchus and the left atrium from above downwards. The left anterior oblique and lateral projections are of particular value since they separate the right from the left ventricle and thus make it possible to assess chamber size. They are also helpful in the assessment of the main pulmonary artery, the ascending aorta, the aortic arch, the descending aorta and structures within the aortic window. (Steiner, 1980).

Fig. 5



Diagrammatic representation of heart shadows and great vessels in the lateral position

CHEST RADIOGRAPH IN THE NEONATE

Information which may be gained from the chest radiograph of the neonate is of much less value than in older children or adults. The neonatal heart at birth reflects the function of the foetus in utero. In the foetus, and consequently at birth, the right ventricle is large and as thick as the left ventricle. The main pulmonary artery is large and the left ventricle and ascending aorta are rather small. The result is that at birth the neonatal heart is relatively large and cardiothoracic ratio up to and exceeding 60% are common in the normal neonate and with a non-specific shape associated with a large right ventricle. The main pulmonary artery is not usually seen on the frontal radiograph in the neonate as both it and the aortic arch are concealed by the large thymus. The lung vessels in the neonate are difficult to assess. At birth they always appear small, presumably because they still retain arteriolar musculature which serves to increase pulmonary vascular resistance at birth. (Raphael, 1986).

This difficulty in cardiac assessment is compounded if there is poor inspiratory effort or slight movement blur, or slight rotation or a large thymus. In addition slight degrees of over or under-exposure of the radiograph can reduce or enhance the visibility of the pulmonary vessels.

The anatomical and functional adaptations of the cardiac chambers to the cardiac derangement that may be present at birth have not had sufficient time to develop.

In spite of these limitations chest radiography and echocardiography are vital in the symptomatic neonate in helping to make the crucial distinction between cardiac and pulmonary causes for the symptoms, as both heart and lung disease may present in the neonate with tachycardia, tachypnoea and cyanosis. Radiography contributes to the management in three ways:-

(1) It can help exclude lung disease as the cause of symptoms. It can exclude gross intrapulmonary pathology such as pneumothorax, congenital lobar emphysema, diaphragmatic hernia or the grosser examples of hyaline membrane disease. This is valuable evidence in pointing away from the lungs and towards the heart as the cause of symptoms.

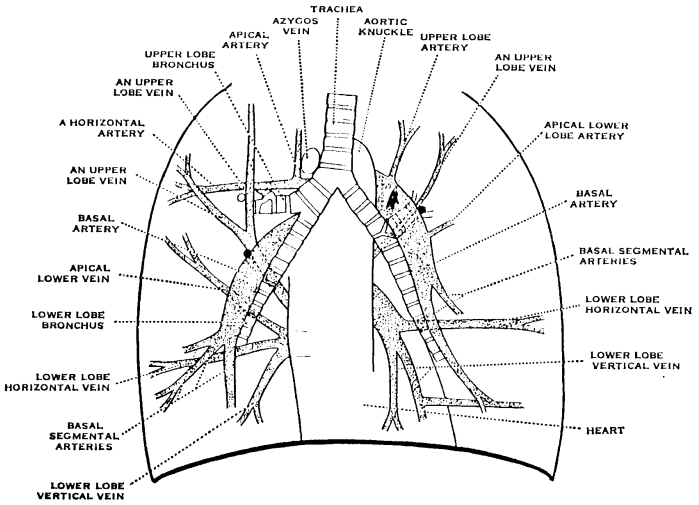
(2) It can make a positive diagnosis of heart disease. The demonstration of gross cardiac enlargement, of abnormal shape, of a grossly abnormal pulmonary vasculature or possibly even pulmonary oedema, will all point to heart disease as the cause of symptoms.

(3) A specific cardiac diagnosis may occasionally be possible. The diagnosis of heart disease usually means that echocardiography and (possibly) cardiac catheterization is indicated for further investigation of those patients who are cyanosed; or whose symptoms do not remit on medical treatment. Specific features will often aid the catheterization in the full elucidation of the anatomical problem. (Raphael, 1986).

THE RADIOGRAPHIC ANATOMY OF THE PULMONARY ARTERIES AND PULMONARY VEINS (Fig. 6):

The middle segment of the left mediastinal border is formed by the main pulmonary artery. The right main pulmonary artery is longer than the left and passes behind the ascending aorta and superior vena cava to enter the right lung anterior to the bronchus. The artery divides immediately before reaching the hilum into the upper lobe and the inter-lobar branches. The inter-lobar branch in turn gives off the middle and lower lobe branches. The left main pulmonary artery is short, arches to the left and downwards, enters the lung slightly from above and behind the main bronchus and branches into the upper and inter-lobar arteries. (Proto, 1984).

Fig. 6



THE SEGMENTAL PULMONARY ARTERIES:-

The main pulmonary arteries are closely related to the corresponding bronchi and have a similar distribution:-

Right Upper Lobe	:	apical, anterior and posterior
Right Middle Lobe	:	medial and lateral
Right Lower Lobe	:	apical, retrocardiac, anterior basal, lateral basal and posterior basal
Left Upper Lobe	:	apical, posterior and anterior arteries

The left inter-lobar artery divides into lingular and left lower lobe artery. The left lower lobe arteries are very similar in distribution to those of the right lower lobe.

THE SEGMENTAL PULMONARY VEINS:-

The main pulmonary veins run an independent course and do not closely accompany the pulmonary arteries or bronchi.

Right Upper Lobe	:	apical, posterior and anterior branches
Right Middle Lobe	:	medial and lateral segmental veins; the upper and middle lobe veins form the superior pulmonary vein
Right Lower Lobe	:	superior and inferior basal veins unite to form the common basal vein; the medial basal vein and the apical vein join the common basal vein and together form the inferior pulmonary vein
Left Upper and Lower Lobe	:	the pulmonary veins are very similar in distribution to those of the right lung

Some anatomical features of the pulmonary blood vessels:-

Variation in the venous pattern are common, particularly in the lower lobes.

Pulmonary arteries and veins can be identified by their various anatomical patterns on plain chest films and particularly on tomograms.

1. Pulmonary veins usually present a less clear-cut shadow than the arteries and appear thicker than the corresponding arteries.

2. All arterial shadows diminish gradually in calibre in proportion to their distance from the hilum.

3. The tributary vessels are always smaller in calibre than the primary vessels.

4. Under normal conditions arterial shadows are either straight or gently curved, but never tortuous. The shadows are of moderate density with well defined sharp edges.

5. Seen end on, they may be round or elliptical and closely related to the accompanying branches.

BRONCHIAL ARTERIES

They are of such small calibre that they are invisible on the plain chest film or tomography. They arise from the upper part of the descending aorta, cross the posterior mediastinum to enter the lungs at the hilum, where they accompany the bronchi very closely, providing their systemic blood supply.

PULMONARY LYMPHATICS

They are too small to be visible on normal chest films, but can become visible in certain diseases when there is obstruction of lymphatic drainage of the lung, e.g. in lymphangitis carcinomatosa, or they may distend when the pulmonary venous pressure is sufficiently elevated to cause interstitial pulmonary oedema.

The inter-lobular septa, so called septal lines, can be identified as short, straight lines perpendicular to the pleura. They are best seen at the costophrenic angles (Kerley's B lines). The deep lymphatics may also be seen (Kerley's A lines) as long, thin angled lines running towards the hila. (Steiner, 1980).

THE DIFFERENT TECHNIQUES OF CARDIAC IMAGING

THE CONVENTIONAL PLAIN RADIOGRAPHY OF THE HEART

The standard projections for the radiological examination of the heart are the postero-anterior film (PA) and the lateral film. Occasionally the right and left anterior oblique projects are needed.

Satisfactory inspiration is essential. Examinations carried out during inadequate inspiration may give a wrong impression of cardiac size due to a high position of the diaphragm, producing a transverse lie of the heart.

The over-penetrated postero-anterior film will help to demonstrate structures behind the heart and also show up an enlarged left atrium as a double contour along the right cardiac border. (Steiner, 1980).

FLUOROSCOPY

The examination should be restricted to those patients where screening is of real value. Fluoroscopy can be helpful in positioning of the patient for radiography when oblique or localized views are to be taken.

It can also provide information about:-

(1) Pulsation of the heart and great vessels.

(2) Calcified shadows within the heart (especially mitral and aortic valve) calcification.

(3) Shadows in the mediastinum close to the heart.

General cardiac pulsation may be diminished in the presence of a pericardial effusion, in constrictive pericarditis or in a grossly dilated heart.

There may be expansile paradoxical pulsation over ischaemic areas of the heart with systolic expansion of the affected part, which may be aneurysmal.

Vascular pulsation in the region of the aorta and pulmonary artery is normally seen, and this may be excessive with a hyperdynamic circulation.

When patients are fluoroscoped, a mouthful of thick barium should be given to outline the oesophagus and to its relationship to the posterior surface of the heart and to other mediastinal structures.

TOMOGRAPHY

Tomography is of value in the demonstration of pericardial and intracardiac calcification. The mitral and aortic valves are roughly in the midline in the postero-anterior projection and overlie the dorsal spine. Tomography in the lateral or oblique projection is of great value in demonstrating calcification of the valves.

Further use for tomography is the demonstration of the lung hila, the large pulmonary vessels and the smaller peripheral vascular branches.

Multi-section tomography is better than the single film technique to reduce radiation to the patient. (Steiner, 1980).

MACRORADIOGRAPHY

Macroradiography is the production of an enlarged image by x-ray magnification. The divergence of the beam from a small source is used to magnify the image geometrically. Successful macroradiography is governed by two conflicting requirements:-

(1) A fine focus tube

(2) A short exposure time

This technique has been suggested by some authors in the investigation of neonatal heart disease. (Hungerford, 1975; Cremin, 1972).

RADIOISOTOPE SCANNING OF THE HEART

Radioisotope scanning of the heart plays an important role in the assessment of heart diseases, especially in ischaemic heart diseases. The developments in the management of coronary artery disease have brought the need for an objective method of measuring regional myocardial perfusion and of detecting myocardial ischaemia. The following techniques represent the different methods of studying the heart with radioisotopes:-

(A) ACUTE MYOCARDIAL INFARCTION SCINTOGRAPHY

The most useful complex is $^{99}Tc^{m}$ pyrophosphate. The optimum time for scanning is 90 minutes after intravenous injection of 10 mCi of $^{99}Tc^{m}$ pyrophosphate.

Anterior views of the precordium are essential and may be supplemented by left anterior oblique and lateral views.

In the normal there is symmetrical activity about the midline over the sternum and ribs. The activity is increased over an infarct and the size of the abnormal area is related to that of the infarct. Abnormal activity can be detected within twelve hours of infarction and is maximal during the next seven days, then returns slowly to normal. Persistent or increased activity suggests an extension of the infarct. Unexpected positive scans may be obtained from sternal and rib fractures, multiple skeletal metastasis and carcinoma of the breast.

The main applications of this form of scanning are:-

(1) In the assessment of obscure chest pain

(2) Suspected myocardial infarction where ECG is normal and serum enzymes are elevated for other causes

(3) In assessment of unstable angina

(Davies, 1980).

(B) MYOCARDIAL PERFUSION SCANNING

The compounds used are analogues of potassium such as thallium-201. It is concentrated in normal myocardium. Myocardial activity is flow dependent and a normal pattern is a uniform horse-shoe shape with a central zone of diminished activity representing the left ventricular cavity. A myocardial infarction creates a corresponding defect. In the normal, myocardial blood flow increases uniformly during exercise, but in coronary artery insufficiency the increase in blood flow is much less, especially when the lumen of the arteries is reduced to 50%. If the scan is repeated after exercise, myocardial ischaemia is shown by deficient zones which were not present on the rest scan. Deficient zones present at rest and after exercise indicate infarction.

These tests help to distinguish myocardial pain due to infarction from that due to ischaemia and from chest pain that is not myocardial in origin. (Davies, 1980).

(C) OTHER METHODS OF RADIOISOTOPE IMAGING

These are further examples of radioisotope studies of the heart. The details of these examinations are beyond the scope of this thesis.

(1) Coronary artery perfusion scanning.

(2) Flow studies using xenon-133 or krypton ($^{81}Kr^{m}$).

(3) Multiple gated images. Data acquired and analysed by computer can be reconstituted to form cine film of cardiac cycle.

(4) Electrocardiogram-linked myocardial function studies.

(5) Rapid sequence isotope cardiography. The quality of the images obtained by this technique is good enough to identify individual heart chambers and valuable information can be obtained about the flow of blood within the heart.

(6) Determination of left ventricular mass using single-photon emission computed tomography:-

Single-photon emission computed tomography could actually determine left ventricular mass in humans. The measurement of viable left ventricular mass may have important prognostic significance in patients with coronary artery disease and cardiac hypertrophy. (Wolfe, et al., 1985).

Radioisotope studies of the heart are useful in the following circumstances:-

(a) In myocardial disease:-

True myocardial disease can be distinguished from other diseases with similar symptoms.

(b) To monitor the progress of the disease both before and after surgery.

(c) As a screening test. In the newborn to distinguish suspected congenital heart disease from respiratory disorders.

In adults to differentiate aneurysm from solid tumours in cases of mediastinal enlargement. (Davies, 1980).

MAGNETIC RESONANCE IMAGING (MRI) OF THE CARDIOVASCULAR SYSTEM

MRI promises to be of great value in the studies of cardiovascular abnormalities.

Gated images when they become routinely available will allow the blood column in a vessel with rapid flow to be visualized without the necessity for injection of contrast material. With improvements in imaging and perhaps with the use of the three-dimensional display method, it will be possible to map out congenital and acquired abnormalities of the heart and great vessels.

The CT scan shows the outward extension of aortic aneurysm, but MR image shows both the outer edges of the blood vessels and the blood column. Multiple images will allow more exact measurements of cardiac chamber volumes, ejection fraction and cardiac wall motion.

One great hope is that MRI can be used for visualization of myocardial ischaemia and infarction. One can imagine that a complete MRI angiogram will be possible. (Kundell, et al., 1983).

GATED MRI OF CARDIAC AND PARACARDIAC MASSES

Angiocardiography, echocardiography and computed tomography are currently used to evaluate cardiac and paracardiac masses. A recent paper has described the potential usefulness for magnetic resonance imaging (MRI) in the evaluation of pericardial abnormalities.

ECG-gated MRI clearly distinguishes cardiovascular structures from adjacent non-vascular structures in the mediastinum. This technique should be ideal for the evaluation of masses located within or adjacent to the heart.

The studies were performed to evaluate the capability of MRI in defining the primary origins of the masses, their local extent and other features of clinical significance. (Amparo, et al., 1984).

PARAMAGNETIC CONTRAST ENHANCEMENT IN ISCHAEMIC HEART DISEASE

Although there is no need for contrast medium to delineate the blood tissue interface of the cardiovascular system with MRI, it has been thought that the use of intravenous paramagnetic substances might improve the specificity in evaluating myocardial ischaemia. The first paramagnetic contrast agent for MRI of ischaemic heart was the bivalent manganese ion. Because of its toxicity, most interest has centred on gadolinium (Gd)-DTPA as a potential MRI contrast agent. (Brasch, et al., 1984). (Weinmann, et al., 1984). The use of paramagnetic contrast agents as myocardial perfusion markers demonstrates myocardial ischaemia without infarction. (Revel, Higgins, 1985).

COMPUTED TOMOGRAPHIC IMAGING OF THE HEART

A high-speed transaxial scanner system with a multiple x-ray source and the Dynamic Spatial Reconstructor, is undergoing evaluative studies.

Accurate quantitative imaging of the heart by CT is technically difficult for three major reasons:-

(1) The heart undergoes continuous non-reproducible motion and change of shape.

(2) Since the magnitude of motion and change of shape of the heart are directly related to cardiac function, these parameters must be quantitated.

(3) Cardiac function involves a complex interaction of multiple aspects of cardiac anatomy. (Ritman, et al., 1980).

The role of computed tomography was assessed in nine patients with hypertrophic cardiomyopathy. The ventricular septum was demonstrated in all patients and shown to be thickened. The results agreed with those obtained by echocardiography, except in two patients in whom computed tomography showed preferential thickening of the mid portion of the ventricular septum. The ventricular free wall was not clearly seen.

Computed tomography may prove a valuable technique in the assessment of patients with hypertrophic cardiomyopathy. (Stone, et al., 1984).

THE DEMONSTRATION OF MYOCARDIAL INFARCTION BY CT

Acute myocardial infarction causes a characteristic defect in the myocardial enhancement on CT scans (made during intravenous injection of contrast medium). The diminished contrast-medium uptake, attributed due to hypo-perfusion, usually involves the endocardium and may extend transmurally to the epicardium.

CT has successfully demonstrated acute and chronic myocardial infarctions and their sequelae such as localized ventricular dilation, wall-thinning, calcification, aneurysm or false aneurysm and thrombus deposition. Electrocardiographically-gated CT has shown focal changes in wall motion and systolic wall thickening. CT, however, is not suitable for routine diagnosis of myocardial infarction because:-

(1) The diagnosis almost can be made clinically by ECG and enzymatically.

(2) A patient with suspected infarction should not be separated from emergency support by being moved to the scanner.

(3) Patients with myocardial infarction are particularly vulnerable to the adverse cardiac effects of contrast agents. (Godwin, et al., 1984).

ELECTRON-BEAM CINE-CT SCANNER

The recent introduction of a multisecond CT scanner specifically designed for cinematographic real-time imaging of the heart is a significant development. Its introduction could parallel the impact that cineangiography had on cardiac diagnosis.

A major advantage of cine-CT is that central catheterization for angiographic purposes is avoided and relatively small amounts of intravenous contrast medium provide excellent CT contrast enhancement of the blood pool and cardiac structures.

The initial results are encouraging. It can play an essential role in the assessment of acute and chronic myocardial infarction, measurements of left ventricular mass and volume and the assessment of coronary artery bypass graft potency. Exercise and pharmacologic stress testing can also be carried out. (Lipton, et al., 1984).

ANGIOCARDIOGRAPHY

The use of contrast media to examine the heart and great vessels is called angiocardiography. The procedure is almost invariably combined with cardiac catheterization so that essential physiological measurements may be made. Radio-opaque contrast medium is injected through a cardiac catheter at the site appropriate for the demonstration of the presumed abnormality - selective angiocardiography.

The method of choice for introducing catheters into the vascular system is the percutaneous Seldinger technique using the femoral artery and the femoral vein for left and right heart catheterization respectively. With special needles and catheters the technique can be used on patients as small as the newborn. Some cardiologists, however, still prefer an open venotomy from the arm and an open brachial arteriotomy for right and left heart catheterization respectively. The trans-septal approach to the left atrium, in which a very long curved needle is passed from the leg up the inferior vena cava and into the right atrium to puncture the interatrial septum and gain access to the left atrium, is now little used.

Angiocardiography may be used to study cardiac anatomy and cardiac function. When anatomical knowledge is required the highest possible concentration of contrast medium should be obtained, even if the fast injection precipitates ventricular ectopic activity. As a rough guide, a bolus of 1 ml/kg of body weight should be injected within one to two seconds. To study cardiac function cardiac rhythm must remain largely undisturbed and a much slower injection rate is chosen for the left ventricle, even if the resulting opacification density is relatively low. With modern x-ray apparatus a good quality left ventriculogram may be made but with an injection rate as low as 8 ml/second for as short as three seconds. A mechanical injector in which the amount delivered and the rate of delivery can be controlled is essential, and a rate of rise of pressure control is useful to lessen catheter whip. ECG triggered pulse injections, although available on many injectors, are not widely used. (Raphael, 1986).

Within the cardiac chambers and great vessels contrast medium should be injected through catheters with side holes but no end hole to avoid jet effect and reduce catheter whip. For the coronary arteries end-hole catheters are used. Contrast medium should be injected downstream of (distal to) suspected leaking valves. Such angio-cardiography will confirm the leak, estimate its severity and may indicate the cause. Contrast medium should be injected upstream of (proximal to) obstructions; it usually indicates the nature of the obstruction but is poor in assessing severity. Physiological measurements of pressure gradient across the obstruction, coupled with calculations of flow across it, will indicate the severity more effectively. Contrast medium should be injected into the "delivering" chamber adjacent to shunt defects so that it will be carried by the presumed direction of flow to indicate the site and number of communications, their origins and terminations. Angiocardiography is very good for demonstrating the presence of a shunt and its anatomy, but poor for assessing the volume-flow of the shunt. Oxygen saturation measurements, or other more complicated techniques, are more satisfactory for such quantitation. (Raphael, 1986).

RADIOGRAPHIC TECHNIQUES OF CARDIAC ANGIOGRAPHY

The method of choice for the functional study of the heart is cine radiography. Most installations use 35mm. cine attached to a high definition caesium iodide intensifier with a choice of 9 or 5 inch modes.

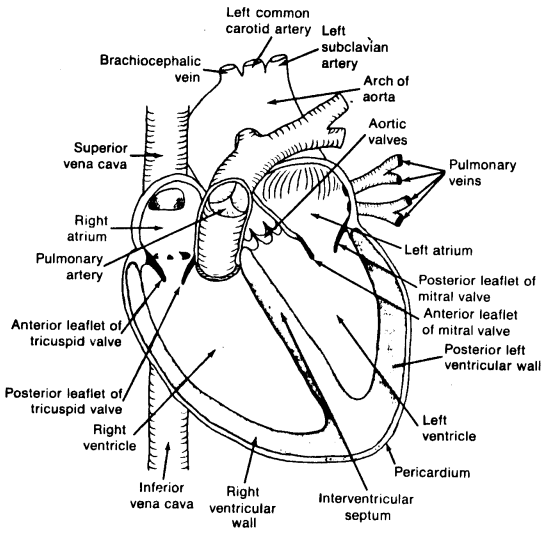
For anatomical study modern high quality cine radiography is the preferred choice for most workers, but some groups orefer 100mm. cut film apparatus, particularly for coronary arteriography where the pictures are large enough for surgeons to review in the operating theatre. For pulmonary angiography and arch aortography the large format full size rapid sequence cut film changer (AOT) is probably the method of choice. For acquired heart disease single plane filming is usually adequate.

For congenital heart disease a biplane installation is essential. Modern apparatus should not only have the facility to angle the x-ray beam around the long axis of the patient, but the operator should be able to apply angulation along the long axis of the body, providing axial tilts, especially in the investigation of the coronary arteries, (Guthaner, Wexler, 1980) and also in some types of congenital heart disease, (Elliott, et al., 1980).

A videotape recorder, with split image for biplane systems, is an essential part of the imaging system, particularly where complex congenital heart disease is studied, as the results of each contrast injection can be immediately reviewed and if necessary further injections made in the most useful way. (Judkins, et al., 1976).

NORMAL CARDIAC ANATOMY WHICH IS SEEN BY ANGIOCARDIOGRAPHY

Fig. 7



RIGHT ATRIUM

The globular shape of the right atrium is seen at angiocardiography which confirms that it forms the right border of the heart in the frontal view. The extreme thinness of the combined right atrial wall and pericardium may also be seen. The broad-based appendage protrudes upwards, forwards and then to the left. The crista terminalis is not recognizable at angiography. The right atrial appendage, contributing to the upper anterior cardiac outline, is the only part of the right atrium to appear in the cardiac silhouette in the lateral view (where it is projected over the right ventricular outflow and pulmonary valve). The anatomical features which characterize the morphological right atrium are the limbus of the

fossa ovalis on the septal aspect, the crista terminalis and the squat, broad-based appendage; only the last may be recognized at angiocardiography. (Raphael, 1986).

RIGHT VENTRICLE

Angiography demonstrates the triangular shape of the right ventricle in the frontal view and confirms that the normal right ventricle makes virtually no contribution to the cardiac silhouette in the frontal projection, though the infundibulum lies very near to the left upper heart border. In the lateral view the flattened right ventricle forms the front of the heart. The lower half of the heart is normally in contact with the back of the sternum.

Because of its obliquity, the tricuspid valve is not seen in profile in either frontal or lateral views although a slight constriction, marking the site of the tricuspid valve ring, can usually be seen in the frontal angiocardiogram even if both right atrium and right ventricle are opacified. When right ventricular emptying is obstructed (as in pulmonary valve stenosis), the tricuspid valve becomes much more obvious in the lateral view in both systole and diastole. If it is necessary to study the tricuspid valve by angiography, it can be brought into profile by a moderate degree of rotation into the right anterior oblique projection. (Raphael, 1986).

Right ventricular morphology is characterized by three features:-

(1) Coarse trabeculation of the septal aspect of the chamber.

(2) A tricuspid atrioventricular valve which is in part connected to the septum of the chamber, either by direct chordal attachment, or through a small papillary muscle of the conus.

(3) A muscular conus or infundibulum separating the entry atrio-ventricular valve from the exit semilunar valve by a ring of muscle.

Only this last may be recognized with any degree of confidence by angiocardiography. (Raphael, 1976).

LEFT ATRIUM

Angiography demonstrates the left atrium to lie at the upper posterior aspect of the cardiac shadow. It gives no definite contribution to the normal cardiac silhouette in the frontal view as the normal left atrial appendage is buried in the epicardial fat and does not produce a discreet shadow. In the lateral view, the left atrium contributes to the upper posterior border of the heart, but in the normal, the left atrium is not in contact with the air containing lung and there is no properly defined outline. The posterior border of the left atrium may, however, be located by the left lower lobe bronchus which passes obliquely downwards and to the left behind it. This feature may be seen on the well penetrated radiograph. The left atrium may be more satisfactorily located by the barium-filled oesophagus which is an immediate posterior relation.

The features which characterize left atrial morphology are an absence of those suggesting right atrial morphology, and more positively the long finger-like left atrial appendage which may be recognized by angiography. (Raphael, 1974).

LEFT VENTRICLE

Angiography in the frontal and lateral views shows that the apex of the left ventricle forms the cardiac apex in the frontal view and also forms the main left border of the heart. In the lateral view, the posterior cardiac border below the mitral valve is formed by the posterior border of the left ventricle. The position of the mitral valve, opening into the left ventricle from behind, is demonstrated during left ventriculography by non-opaque blood passing through it into the left ventricle and creating a negative shadow. The sharp inferior edge of this shadow is produced by contrast medium trapped under the posterior leaflet of the mitral valve.

The feature which best characterizes left ventricular morphology at angiography is the fibrous continuity of the entry atrioventricular valve and the exit semilunar valve, due to the lack of any conus or infundibulum. (Raphael, 1974).

CORONARY ARTERIOGRAPHY

This technique requires selective injection of contrast medium into the right and left coronary arteries and into the left ventricle and the recording of the resultant images.

The catheters may be introduced from the arm through a right brachial arteriotomy. Sones' technique using a woven dacron, stiff-bodied torque-controlled, reusable catheter with a floppy tip, with end and side holes, can also be used. The catheter is buckled against the aortic valve and its tip manipulated into each coronary artery in turn. The same catheter is usually used for both right and left coronary arteries and also for the left ventriculogram. Catheters with two lengths of tip are available and also catheters of different constructions. The catheter may be flipped through the aortic valve into the left ventricle for left ventriculography. The technique is relatively safe and in skilled hands is quick and painless.

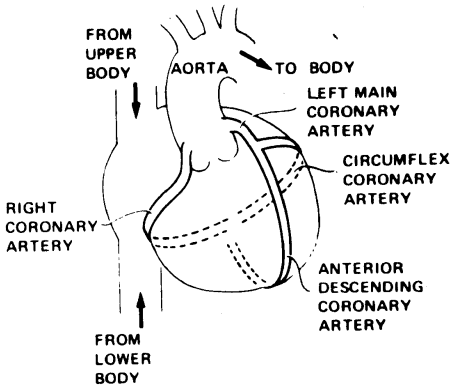
Percutaneous femoral arterial catheterization techniques which avoid open arteriotomy have been in use for many years. All require a number of disposable preshaped catheters, different for each coronary artery and also for the left ventricle. Judkin's method is most popular, but Amplatz's and Bourassa's catheters have their enthusiasts.

At least three catheters are required for each examination. These techniques give a very high success rate for good coronary arterial intubation and high quality angiography and if done carefully have a complication rate as low as the Sones' method. The percutaneous, especially the Judkin's technique, is much the more utilised method, especially by radiologists. (Raphael and Silverman, 1986).

CONTRAST MEDIUM

Non-ionic contrast media are probably the safest but are quite expensive. If ionic media are used it is essential that any medium has the correct balance of sodium and meglumine ions as a predominance of either tends to produce ventricular fibrillation. For coronary arteriography the contrast is injected by a hand-held syringe refilled from a reservoir. For left ventriculography a modern delivery rate controlled mechanical injector, preferably with a 'rise-time' control, is essential.

Fig. 8



Normal coronary anatomy

A diagrammatic depiction of the three coronary arteries

FILMING

Recording of the image is almost invariably in the magnification mode of a high definition caesium iodide image amplifier at anything from 20 to 50 frames per second on cine film. It may also be useful to have available 100mm. cut film fluorography, as the pictures obtained are large enough for the surgeon to study directly in the operating theatre. (Raphael and Silverman, 1986).

X-RAY APPARATUS

Beam direction is extremely important in recording images without overlap or foreshortening. Any equipment must not only have the facility to apply rotation of the tube around the patient, but also to angle the beam along the length of the patient, producing cranio-cardal tilts. In the modern apparatus, the patient lies still and supine during the examination and the x-ray equipment is moved around him on a U- or C-arm.

Powerful x-ray generators are important so that adequate x-ray transmission at iodine-effective kilovoltages is obtained. This produces high quality, high contrast angiograms with short exposure time (5 to 10m second) for each frame. (Raphael and Silverman, 1986).

COMPLICATIONS

(1) Most important in such an extensively performed examination is its safety. The overall mortality is 0.3% of all cases, but there are wide variations according to the type of patients studied and particularly the experience of the operator. (Raphael and Silverman, 1986).

(2) Perforation of the heart or great vessels and myocardial extravasation R.V., L.V. or S.V.C.

(3) Arrhythmias - ventricular fibrillation, asystole or other arrhythmias.

(4) Neurological complications.

(5) Pulmonary complication such as pulmonary embolism or ischaemia and oedema.

(6) Rupture of the coronary sinus.

(7) Systemic arterial embolism. Unsatisfactory examination may be performed. (Steiner, 1980). (Raphael and Silverman, 1986).

DIGITAL CARDIAC RADIOLOGY

EVALUATION OF LEFT VENTRICULAR FUNCTION IN PATIENTS WITH ISCHAEMIC HEART DISEASE

Multiple digital subtraction techniques have been developed to assess global and regional function of the left ventricle using intravenous injections of contrast material. These techniques provide useful information about left ventricular function and left ventricular wall motion abnormalities. There is a great deal of research being done in the area of digital imaging to assess the degree of myocardial hypoperfusion in patients with coronary artery disease. The details are beyond the scope of this thesis.

DIGITAL RADIOGRAPHIC EVALUATION OF CORONARY ARTERY STENOSIS

IV-DSA has the capability to verify the potency of saphenous vein bypass grafts originating from the ascending aorta. However it is less accurate in assessing stenosis of the coronary arteries which are smaller in diameter. (Newell, et al., 1985).

DIGITAL IMAGING OF THE PATIENT WITH CONGENITAL HEART DISEASE

Both intravenous and intra-arterial digital imaging of the heart have been useful in assessing congenital heart disease. The IV-DSA studies produced high quality images and accurate physiological shunt date was also obtained which compared with catheter angiography and nuclear medicine studies. Although indications for IV-DSA use are still not clear in the paediatric population with heart disease, clinically useful diagnostic information has been obtained in the following categories of paediatric patients:-

(a) Patients with suspected functional heart murmurs.

(b) Anomalies of the aortic arch, including coarction of the aorta.

(c) Suspected inadequate cardiac surgery.

(d) Simple left-to-right shunt, atrial septal defect and ventricular septal defect.

(e) Cyanotic newborns may all benefit from IV-DSA examinations.

INTRA-ARTERIAL APPLICATION OF CARDIOVASCULAR DIGITAL SUBTRACTION ANGIOGRAPHY

With the introduction of intra-arterial digital subtraction methods, it is now possible to evaluate multi-system occlusive disease. The dimensions of the catheters can now be reduced to 5Fr, with reduction in both the volume and the concentration of the contrast medium. (Crummy, et al., 1982).

In the less experienced environment and in the community hospital, the positioning of a 5Fr catheter at the base of the aorta and injection of contrast material will frequently provide adequate survey information. (Wholey, 1985).

One of the major advantages of the digital study is the immediate availability of the examination for review. This is of significant value during the ventriculogram, when qualitative functions can be observed instantly. (Tobis et al., 1983).

This is also true during the intra-aortic injection, when a small injection at the root can provide evidence of significant left main coronary artery stenosis, providing an adequate "road map" prior to the selective examination. This so-called "digital road mapping" provides immediate visual correlation during the fluoroscopic procedure and is becoming important during a variety of interventional and percutaneous angioplasty procedures. (Tobis, et al., 1985).

ECHOCARDIOGRAPHY

Echocardiography began in 1954 when Edler and Hertz of Sweden first used sonar for cardiovascular diagnosis. Since then the technique has been employed in other branches of medicine. The transducer, which consists of a cylindrical tube with a piezoelectric crystal at the end is in contact with the patient. The crystal converts electrical impulses and reverses the process, converting the returning high frequency sound impulses, or echoes, to electrical impulses. The transducer thus acts both as a transmitter and receiver. By definition ultrasound is sound with a frequency of more than 20,000 cycles/sec (20 KHZ) which means that it is above the audible range of ultrasonics.

Ultrasonic waves are a form of energy consisting of mechanical vibrations oscillating at a frequency of 1-15 MHZ (1 MHZ = 1 million cycles per second). The particles making up the medium vibrate backwards and forwards about their positions and the energy is transferred by means of this vibration, without the particles themselves moving through the medium. There is thus no transfer of matter as such so there is a minimum disturbance of the tissues. A minute amount of energy is dissipated in the form of heat, but there are no confirmed reports of injury to tissue as a result of ultrasound being used diagnostically. In adult echocardiography transducer frequencies most often used are in the range of 2.5-5.5 MHZ, whilst for children 5 MHZ transducer is preferred. The ultrasound pulses sent out are intermittent and this allows the transducer to receive those waves reflected back to the crystal during the intervals between the pulses. The time between the emission of the ultrasonic pulse and the return of the echo enables estimation of the distance of the reflecting surface as the speed of sound in tissue is known to average 1540 meters per second. (Oram, 1981).

In echocardiography the main tissues encountered are myocardium, endocardium and blood. Blood is an extremely good conductor of ultrasound, whereas bone absorbs most of it. It is poorly propagated through gases and air, thus the lung does not transmit the ultrasound beam. When the beam strikes an interface it is not totally reflected; some of it continues and it is partially reflected at the next interface when it meets. The amount of ultrasound reflected depends on the angle at which the beam meets the interface,

the nearer the angle approaches 90^{o} the greater the amount of beam is reflected. (Oram, 1981).

The loss of ultrasound as it traverses a medium is known as attenuation, which is a combination of absorption and scattering.

A term used to express the amount of absorption and attenuation of ultrasound in tissue is the half value layer or half power distance. This term refers to the distance that ultrasound will travel in a particular tissue before its energy is decreased to half its original value.

A thick muscular chest wall would offer a significant obstacle to the transmission of ultrasound. Air and lung have extremely short half-power distance and represent severe obstacles to the transmission of ultrasound. (Feigenbaum,1981).

TYPES OF ECHOCARDIOGRAPHY

ONE DIMENSIONAL ECHOCARDIOGRAPHY

A MODE:

This method is best used for locating immobile surfaces. The position is revealed by the distance from the transducer along the horizontal axis and the amplitude or height of each peak of echo proportional to its intensity, hence the term A (for amplitude) mode. Any movement of interface will be seen as to and fro motion along the horizontal axis. This mode is of limited diagnostic value in cardiology.

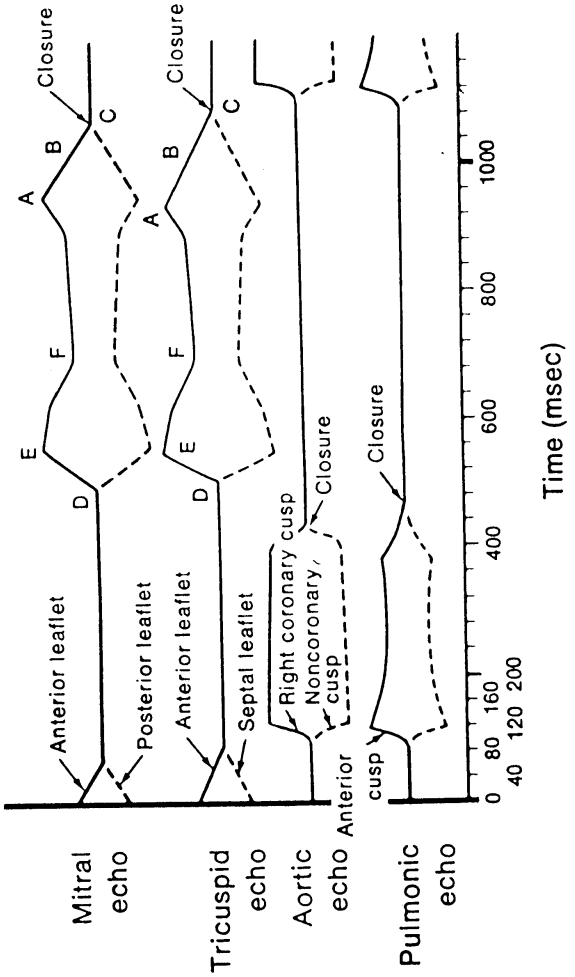
B MODE:

This is an intermediate step between the A and M modes. The echoes are shown as dots instead of peaks along horizontal axis. The amplitude of the peaks is indicated by the brightness of the dots, hence the term B (for brightness) mode. It has no diagnostic value in cardiology.

M. MODE:

This is the one usually employed in clinical echocardiography. If the B mode is moved vertically across the oscilloscope screen at constant speed, a motion photograph can be recorded to produce an M (for motion) picture. The vertical axis represents time. An electrocardiogram is recorded simultaneously. Distance is measured vertically and usually dots 1 cm. apart are provided. Time is read horizontally from left to right and dots are placed at half second intervals. For permanent records a strip chart recorder, ultra-violet or photographic prints are used. (Oram, 1981).

The M mode recording permits measurement of cardiac dimensions and detailed analysis of time relationships with other physiological variables such as E.C.G., heart sounds and pulse tracings, which can be recorded simultaneously.



TECHNIQUE

The operator holds the probe against the skin of the chest wall in the third or fourth left intercostal space using a small amount of liquid jelly to ensure a good acoustic contact with the skin. The patient is placed in the semi-sitting position.

The details of this modality are beyond the scope of this thesis. Fig. ( ) demonstrates examples of M mode recording.

A - The Mitral Valve

B - The Aortic Valve

TWO DIMENSIONAL ECHOCARDIOGRAPHY

The introduction of real-time two-dimensional echocardiography has been a major advance in non-invasive imaging of the heart and great vessels. The images are presented in a familiar format comparable to that in angiography and can be directly correlated with cardiac anatomy.

Two-dimensional echocardiography eliminates many of the limitations of M mode echocardiography; when both techniques are used, the results are extremely complementary for the assessment of cardiac anatomy and function. (Seward and Tajik, 1980).

In the literature, real-time two-dimensional echocardiography is referred to by a variety of other alternative names:-

cross sectional echocardiography

real-time B-mode echocardiography

real-time sector scanning

multiple crystal or multiscan echocardiography

Each term is in some way descriptive of the capability or design characteristics of various systems of the two-dimensional echocardiography.

TWO-DIMENSIONAL ECHOCARDIOGRAPHIC EXAMINATION

In practice four transducer positions are commonly utilized in the two-dimensional echocardiographic examinations; (Fig. 10A):-

(a) Parasternal position

(b) Apical position

(c) Subcostal position

(d) Suprasternal position

Fig. 10A

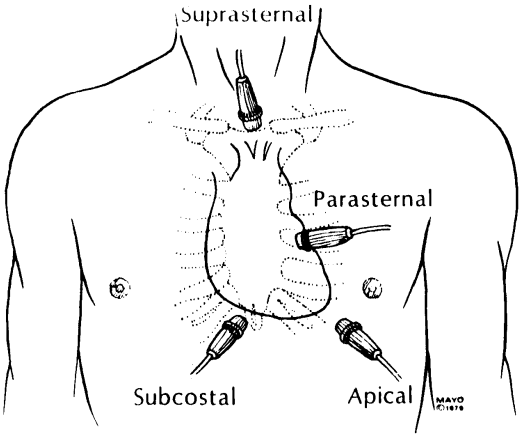
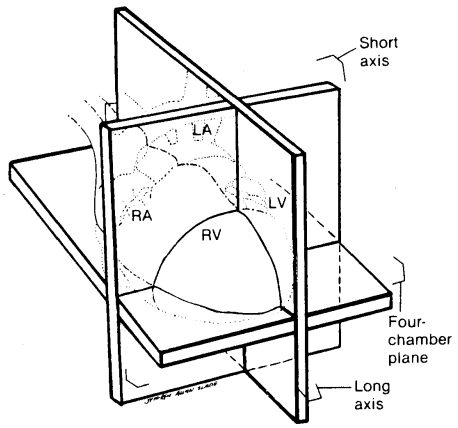


Illustration of four standard transducer positions:-

parasternal; apical; subcostal; and suprasternal ultrasound windows for visualization of the heart and great vessels.

Tomographic sections of the heart relative to its long and short axes can be obtained from each of the first three transducer positions and those of the great vessels - (aorta; pulmonary and superior vena cava) - from the suprasternal notch position. The various tomographic sections are presented in Table I (Fig. 10B). (Tajik, et al., 1978).

Fig. 10B



Imaging planes for the two-dimensional study include the short axis; long axis; and four chamber plane.

TABLE I

TRANSDUCER POSITION AND CORRESPONDING CARDIAC TOMOGRAPHIC SECTIONS

POSITION	VIEW	TOMOGRAPHIC SECTION
Long Axis	Left Ventricle	1
	Right Ventricular Inflow	2
	Right Ventricular Outflow	3
	Right Ventricular and Left Ventricular Inflow	4
PARASTERNAL Short Axis	Left Ventricular Apex	5
	Papillary Muscles	6
	Mitral Valve	7
	Left Ventricular Outflow	8
	Great Arteries	9
	Pulmonary Trunk Bifurcation	10
APICAL	Four-chamber View	11
	Four-chamber and Aortic Valve	12
	Right anterior oblique equivalent	13
SUBCOSTAL	Inferior Vena Cava and Hepatic Vein	14
	Right Ventricle and Left Ventricular Inflow	15
	Left Ventricle-Aorta	16
	Right Ventricular Outflow	17
SUPRASTERNAL NOTCH	Long Axis Aorta Short Axis Rt. P.A.	18
	Short Axis Aorta Long Axis Rt. P.A.	19
	Long Axis Aorta and SVC	20

(Mayo Clin., Proc. May, 1978; Vol. 53 : 275).

PARASTERNAL POSITION

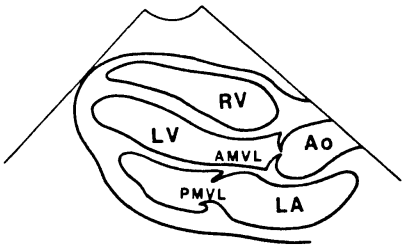
Examination is begun by placing the transducer in the left parasternal region, usually in the third or fourth left intercostal space. From this position a sector image of the heart along its long and short axes can be obtained.

LONG-AXIS VIEWS (Fig. 11)

LONG AXIS VIEW OF THE LEFT VENTRICLE - TOMOGRAPHIC SECTION (1)

The ultrasonic beam is parallel to a line joining the right shoulder to the left flank. The image thus obtained represents a section through the long axis of the left ventricle. (Fig. 11).

Fig. 11



Parasternal long axis view of the heart

Ao. = Aorta
LA = Left Atrium
AMVL = Anterior Mitral Valve Leaflet
PMVL = Posterior Mitral Valve Leaflet
LV = Left Ventricle
RV = Right Ventricle

The long-axis view of the left ventricle allows visualization of the aortic root and aortic valve leaflets. The aortic valve leaflets appear thin and they coapt during diastole in the midline of the aortic root. With the onset of systole the leaflets open abruptly and come to lie nearly parallel to the aortic walls. The right ventricular outflow tract is anterior to the aortic root. The chamber behind the aortic root is the left atrial cavity. Immediately posterior to the lower part of the left atrium the left inferior pulmonary vein, appearing as a rounded structure, can also be seen. This view allows good visualization of the anterior and posterior leaflets of the mitral valve along with their chordal and papillary muscle attachments. The anterior leaflet appears longer and larger than the posterior leaflet. Both are thin and usually produce uniform echoes. With the onset of diastole the distal third of the anterior mitral leaflet approximates the left ventricular septal surface, but it moves posteriorly to lie in a mid-open position during mid diastole and reopens with atrial systole. The posterior leaflet demonstrates a similar although oppositely directed (mirror image) motion and has a much smaller excursion compared with the anterior leaflet.

During systole the mitral valve leaflets coapt at a point inferior (toward the left ventricular apex) to the left atrio-ventricular groove. The coronary sinus may appear as a small, circular echo-free structure in the region of the posterior atrio-ventricular groove. The left ventricular outflow tract, bounded by the ventricular septum anteriorly and the anterior leaflet of the mitral valve posteriorly, is well seen and is normally widely patent during systole. The ventricular septum can usually be visualized in its entirely and its motion characteristics at different levels can readily be appreciated. In normal patients the septum moves posteriorly during systole. The normal anatomic relationships of septal-aortic and mitral-aortic continuity are best evaluated in this view. This view allows good visualization of the left ventricular cavity in the long axis throughout various phases of the cardiac cycle.

USEFULNESS OF THE LONG-AXIS VIEW OF THE LEFT VENTRICLE:

(1) Evaluating aortic root pathology (enlargement, dissection).

(2) Evaluating aortic valvular pathology (calcification, stenosis, vegetations, bicuspid valve).

(3) Evaluating subvalvular left ventricular outflow obstruction (muscular or membranous stenosis).

(4) Evaluating left ventricular chamber dimensions and performance.

(5) Determining ventricular septal and posterior wall motion, excursion, thickness and function.

(6) Visualizing ventricular septal defect and septal aneurysm.

(7) Evaluating ventricular septal-to-aortic continuity or discontinuity, and mitral-to-aortic fibrous continuity.

(8) Evaluating structural and motion abnormalities of mitral valve and of its supporting structures, for example:-
stenosis; subvalvular; valvular or annulus calcification; prolapse; flail leaflets; vegetations; diastolic flutter; systolic anterior motion.

(9) Evaluating enlargement of coronary sinus as seen in patients with anomalous systemic venous or pulmonary venous connection to coronary sinus.

(10) Evaluating left atrial dimension and detecting intra-atrial masses (thrombus, myxoma) or membrane cortriatriatum.

LONG AXIS VIEW OF RIGHT VENTRICULAR INFLOW - TOMOGRAPHIC SECTION (2)

With the transducer in the same interspace (third or fourth) and with inferomedial tilt and slight clockwise rotation of the transducer, a long-axis view of the right ventricle and right atrium is obtained. The image orientation of this view is such that the chest wall is

anterior, the right atrium is on the right and posterior and the right ventricular visualization of the right atrial cavity, the tricuspid valve and right ventricular inflow up to the apex of the right ventricle. The position and motion of the tricuspid leaflets are usually well demonstrated in this view. As with the anterior leaflet of the mitral valve, the anterior leaflet of the tricuspid valve is a relatively larger and longer structure when compared with the posterior and septal components of this valve. The excursion of the anterior leaflet is large during diastole, whereas there is comparatively less excursion of the posterior and septal leaflets.

LONG-AXIS VIEW OF THE RIGHT VENTRICULAR OUTFLOW TRACT - TOMOGRAPHIC SECTION (3)

The long axis of the right ventricular outflow tract can be recorded with the transducer in the same parasternal location but with further clockwise rotation and slight superior tilting of the transducer so that the beam is now nearly parallel to the true sagittal plane. The image orientation of this view is such that the right ventricle is anterior and the left ventricle is posterior. In this section the pulmonary valve is recorded to the right of the image, with the right ventricular outflow tract anteriorly and an oblique section of the left ventricle posteriorly. This section allows good visualization of the entire right ventricular outflow region and of the pulmonary valve and the proximal main pulmonary artery.

USEFULNESS OF LONG-AXIS VIEWS OF RIGHT VENTRICULAR INFLOW AND OUTFLOW TRACTS

(1) Evaluating right atrial and right ventricular enlargement.

(2) Detecting mass lesions in the right atrium and ventricle.

(3) Evaluating right ventricular outflow tract.

(4) Evaluating tricuspid valvular abnormalities.

(5) Evaluating tricuspid atresia.

(6) Diagnosing Ebstein's anomaly.

(7) Evaluating pulmonary valvular pathology.

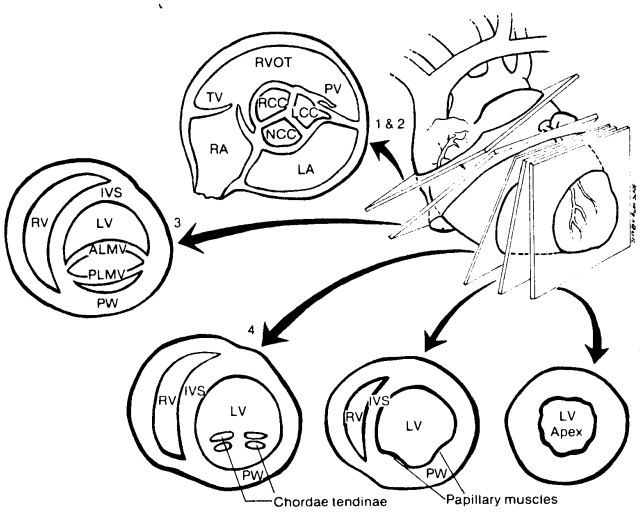
LONG-AXIS VIEW OF RIGHT AND LEFT VENTRICULAR INFLOW TRACTS - TOMOGRAPHIC SECTION (4)

This view is best recorded with the transducer usually in the fourth interspace in the parasternal region and often slightly more laterally placed. This view records the four chambers of the heart. Both ventricles appear slightly foreshortened, but the inflow tracts of both ventricles are readily seen. The image is oriented so that the atria are displayed on the right and posterior and the ventricles are on the left and slightly anterior. The right atrium and right ventricle are anterior, closer to the transducer, whereas the left atrium and left ventricle are posterior. In this view the comparative dimensions of both atrial chambers can usually be easily delineated. The long axis of the atrial septum can be visualized with occasional drop-out or thinning in the region of the fossa ovalis. The motion characteristics of the atrial septum can also be evaluated. Normally, the atrial septum bulges towards the right atrium during ventricular systole. In patients with tricuspid regurgitation, a reversal of the atrial septal motion can be seen. The anatomic inter-relationships of the anterior leaflet of the mitral valve and the septal leaflets of the tricuspid valve can be well outlined in this view.

SHORT-AXIS VIEWS - Fig. (12)

The short-axis view of the heart is obtained by rotating the transducer clockwise so that the plane of the ultrasound beam is approximately perpendicular to the plane of the long axis of the left ventricle. The beam is roughly parallel to a line joining the left shoulder to the right flank. With the transducer pointed directly posteriorly, a cross section of the left ventricle at the level of the mitral leaflets is obtained (section 7). From this position the transducer is tilted inferiorly towards the left ventricular apex, so that a transverse section of the ventricular apex is obtained. (Section 5).

Fig. 12



The short axis plane may be used to evaluate cardiac structures at the level of:-

(1) Pulmonary Valve - PV
Right Ventricular Outflow Tract - RVOT
Tricuspid Valve - TV
Right Atrium - RA
Left Atrium - LA
Right Coronary Cusp - RCC
Left Coronary Cusp - LCC
Non-coronary Cusp - NCC

(2) As above, only better visualization of the aortic cusps.

(3) Right Ventricle - RV
Intervening Septum - IVS
Left Ventricle - LV
Anterior Leaflet of Mitral Valve - ALMV
Posterior Leaflet of Mitral Valve - PLMV
Posterior Wall - PW

(4) Right ventricle, interventricular septum, left ventricle, chordae tendineae, papillary muscles and posterior wall.

Short-axis at the level of the left ventricular apex can also be obtained by placing the transducer directly over the point of maximum (apical) impulse - (Tomographic Section (5)).

Short-axis at the level of the papillary muscles - (Tomographic Section (6)).
This can be obtained by superior tilting of the ultrasound beam from the previous position. The papillary muscles, namely antero-lateral and postero-medial, project into the left ventricular cavity at approximately the 3.0 and 8.0 o'clock positions respectively.

Short-axis at the level of the mitral valve leaflets - (Tomographic Section (7)).
In this view the mitral anterior and posterior leaflets are transected in cross section and appear like a fish-mouth during diastole. Portions of the right ventricle, tricuspid valve, ventricular septum and left ventricular outflow tract are visualized anterior and to the left of the image. The tricuspid valve orifice, when transected in full, has a round-edged triangular shape during diastole. The moderator band and the anterior papillary muscle become readily recognized, especially if the right ventricle is hypertrophied and enlarged.

Short-axis at the level of the left ventricular outflow tract - (Tomographic Section (8)).
This section allows visualization of the anterior mitral leaflet; the outflow tracts of both ventricles and the boundaries of the left ventricular outflow tract. The right atrium and the right ventricle, with a portion of the anterior tricuspid leaflet, are seen on the left of the image. The left atrium is seen directly posteriorly.

Short-axis at the level of the great arteries - (Tomographic Section (9)).
The aorta appears as a circle with a tri-leaflet aortic valve that has the appearance of the letter Y during diastole. The right ventricular outflow tract crosses anterior to the aorta from the left to the right of the image, wrapping around the aorta, and in cross section it forms a sausage-like appearance anterior to the

circular aorta. The pulmonary valve is observed anterior to and to the right of the aortic valve. The other structures recorded in this section include:-

the left atrium (posterior to the aorta);

the left atrial appendage (to the right);

the right atrium (to the left of the image).

The atrial septum is interposed between the left and right atrial chambers. The origins of the right and left main coronary arteries can be seen in this view also. The anterior leaflet of the tricuspid valve may be seen on the left of the image. Occasionally, the inferior vena cava and the coronary sinus can also be seen.

Short-axis at the level of the main pulmonary artery and its bifurcation (Tomographic Section (10)).
This section is obtained if the transducer is rotated slightly clockwise and tilted superiorly beyond the pulmonary valve.

USEFULNESS OF SHORT-AXIS VIEWS:-

(1) Determining right and left ventricular chamber dimensions.

(2) Evaluating global and regional left ventricular performance.

(3) Estimating mitral and tricuspid valve orifice.

(4) Locating and determining the number of papillary muscles in the left ventricle.

(5) Evaluating atrial dimensions and detecting intra-atrial masses.

(6) Evaluating atrial septum for motion and defects.

(7) Determining spatial orientation of great arteries.

(8) Evaluating abnormalities of aortic, pulmonic, mitral and tricuspid valves.

(9) Visualizing right and left main coronary arteries.

(10) Evaluating aortic root pathology.

(11) Evaluating main pulmonary artery and proximal right and left branches for size and presence of clots.

(12) Evaluating enlargement and drainage of coronary sinus.

APICAL POSITION

This view is obtained with the patient turned in the left semi-lateral decubitus position. The apical impulse is localized and the transducer is placed at, or in the immediate vicinity of, the point of maximum impulse.

For the four-chamber view, the ultrasound beam is directed superiorly and medially towards the patient's right scapula - (Tomographic Section 11 - Fig. (13)).
In this position the beam transects the heart from the apex to the base. This view displays all four chambers of the heart, the ventricular and atrial septa and the crux of the heart. The image orientation is such that the apex is on the top and the atria are on the bottom. The right atrium and right ventricle are on the right and the left atrium and left ventricle are on the left of the image.

Fig. 13

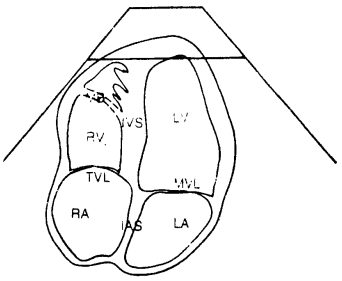


Diagram of the apical 4-chamber plane image systole

This apical four-chamber view allows evaluation of the inter-relationships of the ventricular and atrial septa and of both atrio-ventricular valves. Normally with this projection the ventricular and atrial septa do not appear to join each other in a straight apex-to-base line. Instead, the atrial septum appears displaced slightly to the left of the ventricular septum. The left (mitral) atrio-ventricular groove is normally slightly higher than the right (tricuspid) atrio-ventricular groove. The anterior leaflet of the mitral valve inserts in the left atrio-ventricular sulcus and near the cephalic end of the membranous septum, whereas the septal leaflet of the tricuspid valve inserts near the mid portion of the membranous septum. Therefore, the insertion of the septal leaflet of the tricuspid valve is somewhat inferior to the insertion of the anterior mitral leaflet. (5 - 10mm in the hearts of older children and adults). This is an important anatomic distinction because it can be useful in identifying ventricular chambers.

In this view the atrial septum can usually be seen in its entirety. However, drop-out of echoes does occur in its mid portion, which is the region of fossa ovalis.

With the same apical transducer position, in order to obtain tomographic section (12), the transducer is tilted further anteriorly to record the aortic root and valve in addition to the four-chambers. The aortic root occupies the region where the crux of the heart was recorded in the previous section.

Right anterior oblique view of the left ventricle - (Tomographic Section (13)).
The beam is directed in a plane nearly parallel to the ventricular septum. In this view the apex of the left ventricle is displayed at the top of the projected image, the aorta is at the bottom and the left atrium lies directly posterior. In this view the antero-lateral and inferior walls of the left ventricle are imaged. (Fig. 14).

Fig. 14 a,b

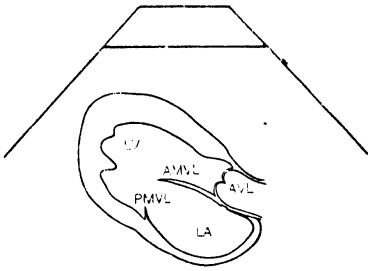


Fig. a

Diagram of the apical long axis image diastole

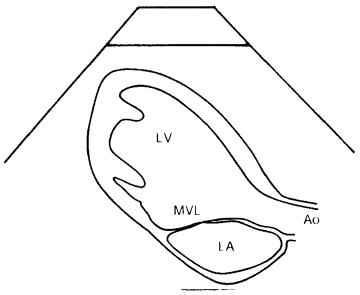


Fig. b

Diagram of the apical long axis imaging systole

USEFULNESS OF APICAL VIEWS

(1) Evaluating ventricular and atrial dimensions.

(2) Detecting and localizing intracardiac masses.

(3) Evaluating septal defects.

(4) Detecting malalignment of atrial and ventricular septa.

(5) Detecting displacement of atrio-ventricular valve leaflets and orifice as in Ebstein's anomaly.

(6) Detecting structural abnormality of the atrio-ventricular valves and of their tensor apparatus.

(7) Evaluating left ventricular function.

(8) Evaluating left ventricular apex and detecting left ventricular aneurysm.

(9) Determining aortic override of the ventricular septum.

(10) Visualizing the right and left inferior pulmonary veins draining into the left atrium.

SUBCOSTAL POSITION

In certain patients, especially those with chronic obstructive lung disease and emphysema, the usual precordial ultrasonic window may become obliterated because of hyper-inflated lungs. This situation necessitated a search for other locations for imaging the heart and led to the discovery of the subcostal region as a good ultrasonic window in such patients. With the two-dimensional ultrasound system, it was observed that the subcostal position provides good imaging of the heart not only in emphysematous patients but also in the majority of normal adults, children and infants. The views recorded from the subcostal position are unique, for they allow better definition of

certain structures from this location than can be obtained from the precordial positions.

The subcostal examination is begun by placing the transducer in the midline or slightly to the patient's right. The transducer head is tilted inferiorly and slightly towards the patient's right and with this position the liver parenchyma, hepatic vessels and short-axis view of the inferior vena cava are obtained. With slight superior tilt of the transducer, the drainage of the hepatic veins into the inferior vena cava can be identified. The supine position is sometimes preferred for subcostal examination.

For obtaining a four-chamber view (Tomographic Section 15 - Fig. 15) the transducer is tilted further superiorly so that it points roughly between the patient's suprasternal notch and the left supraclavicular fossa. A tomographic view of the heart is thus obtained, which is nearly similar to the four-chamber view obtained from the parasternal position, except that in this view the apices of the two ventricles can be visualized by tilting the transducer head slightly towards the patient's left. In this section the two atria, and especially the atrial septum, are best visualized. Whereas dropout of echoes of the atrial septum in the region of the fossa ovalis may be noted from parasternal and apical transducer positions, the atrial septum can be seen in its entirety from the subxiphoid position, and it appears intact in almost every examination without any persistent dropout. Atrial septal motion can again be well evaluated in this view. The right atrium and right ventricle are displayed as anterior chambers closer to the transducer, and the left atrium and left ventricle are displayed posteriorly. A portion of the hepatic parenchyma is interposed between the transducer and the cardiac silhouette.

Fig. 15

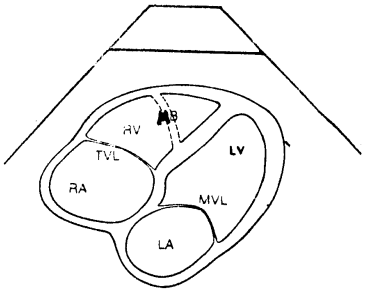


Diagram of the subcostal 4-chamber plane image systole

From this position the transducer is rotated clockwise and tilted slightly superiorly to visualize the aorta and its relationship to the mitral valve and left ventricle. In this tomographic section (number 16), a foreshortened view of the left ventricular long aixs is recorded. Both leaflets of the mitral valve and the aortic leaflets, as well as the left ventricular outflow tract, can usually be well visualized. The tricuspid valve can usually be seen directly anterior in this view.

Tomographic section 17 is obtained by further clockwise rotation and superior tilting of the transducer, which results in a cardiac cross section. In this view the left ventricle is visualized in the short axis with portions of the mitral valve in its cavity. More importantly this view is utilized to show the long axis of the entire right ventricular outflow tract.

USEFULNESS OF SUBCOSTAL VIEWS:-

(1) Detecting dilatation or obstruction of the inferior vena cava.

(2) Determining inferior vena cava-to-atrium connections.

(3) Determining visceral and atrial sinus.

(4) Evaluating atrial dimensions and detecting atrial masses.

(5) Evaluating atrial septal motion.

(6) Detecting and locating atrial septal defects.

(7) Detecting right and left mitral valve abnormalities.

(8) Detecting pulmonary valvular abnormalities.

(9) Determining right ventricular wall thickness.

(10) Evaluating right ventricular apex and outflow tract.

(11) Determining number, origin and spatial relationship of the great arteries.

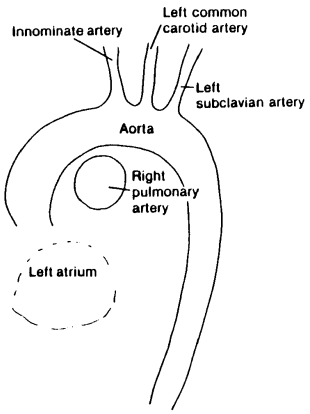
(12) Evaluating left ventricular size, shape, performance and wall dynamics.

(13) Evaluating ventricular septum for thickness, dynamics and defects.

SUPRASTERNAL NOTCH POSITION (FIG. 16)

For visualization of the aortic arch in the long axis - (Tomographic Section 18), the transducer head is positioned in the suprasternal notch, with the long axis of the transducer to the left and parallel to the trachea. With this transducer position, the ascending aorta, aortic arch, origin of the brachiocephalic vessels and descending thoracic aorta are visualized. Occasionally, leaflets of the aortic valve can also be seen in the aortic root. Posterior to the ascending aorta and beneath the aortic arch, the right pulmonary artery is visualized in its short axis. Inferior to the right pulmonary artery, the left atrium can be recorded.

Fig. 16



Schematic cardiac structures visualized in the suprasternal view

The short axis of the aortic arch (Tomographic Section 19) is obtained by rotating the transducer clockwise. In this view the cross section of the ascending aorta appears superior and the right pulmonary artery in its long axis appears inferior. Occasionally, the first bifurcation of the right pulmonary artery can be visualized to the left of the image. With slight clockwise rotation of the transducer, and by tilting it towards the patients left and slightly anteriorly, the distal main pulmonary artery can be visualized. From this position, with tilting of the transducer posteriorly and to the left, the left pulmonary artery may occasionally be seen.

Inferior to the pulmonary artery, the left atrial cavity is seen. Immediately beneath the distal part of the right pulmonary artery the right superior pulmonary vein connecting to the left atrium can be seen. Furthermore, in this view, the superior vena cava can also be recorded, appearing as an echo-free space alongside the aorta on the left of the image. The left innominate vein can also be visualized traversing superior to the aorta to its junction with the superior vena cava. The right innominate vein can be equally well visualized joining the superior vena cava. With slight counter-clockwise rotation and anterior tilt of the transducer, the long axis of the superior vena cava can be recorded alongside the long axis of the ascending aorta (Tomographic Section 20). In this view the superior vena cava can be scanned to its junction with the right atrium.

USEFULNESS OF THE SUPRASTERNAL NOTCH POSITION:

(1) Determining the dimensions of various parts of the aorta and detecting dissection of the aorta.

(2) Evaluating arch abnormalities.

(3) Detecting and localizing coarctation of the aorta.

(4) Determining the size of the right main and occasionally left pulmonary arteries.

(5) Evaluating superior vena caval abnormalities.

(6) Evaluating the Waterston and Glenn anastomoses.

CARDIAC IMAGING WITH ULTRASOUND : RIGHT SIDE UP OR UPSIDE DOWN?

The apical four chamber projection was described by Silverman and Schiller in 1978. With the echocardiographic equipment they used the heart appeared inverted on the oscilloscopic screen. When the transducer was placed at the cardiac apex, they presented the material in the same manner. Bierman, Fellows and Williams convinced the manufacturer of their ultrasound equipment to install a simple switch to allow "flipping" the image 180°, so that when imaged from the apical or subcostal areas the heart would appear in the anatomical position with the atria on top and the apex at the bottom. This instrumentation became available in 1979 and so facilitated evaluation of complex congenital heart abnormalities.

In 1980 the guidelines of the American Society of Echocardiography recommended the anatomic projection as "Option 1", but acknowledged the upside down image as a second option. (Gutgesell, 1985).

DETECTION OF CORONARY ARTERIES BY TWO-DIMENSIONAL ECHOCARDIOGRAPHY

Weyman has shown the ability of two-dimensional echocardiography to identify the proximal part of the left main coronary artery. More recent studies have indicated that other segments of the coronary arteries may also be visualized.

Although the possibilities are intriguing, it is uncertain at present what role, if any, two-dimensional echocardiography will have in the direct identification of atheromatous disease of the coronary arteries. (Weyman, et al., 1976).

CONTRAST ECHOCARDIOGRAPHY

The development of contrast echocardiography has made it possible to derive information from non-invasive procedures which was available only from cardiac catheterization.

Injections of indocyanine green, dextrose urografin, blood or saline will all produce excellent echo-targets, which appear downstream from the injection sites progressing through the right heart in several cardiac cycles. They are then completely cleared by the capillary bed of the lung. Since peripherally injected contrast is usually cleared by the lung in the pulmonary capillary bed, the appearance of contrast in the left heart is abnormal and usually signifies a right-to-left shunt.

In patients with right-to-left shunts, contrast may appear in the left chambers when the degree of the shunt is as small as 5 per cent. The location and timing of the appearance of contrast in the left heart will depend upon the level of the shunt and the relative intracardiac pressures in the right and left heart.
(Pieroni, et al., 1973).

DOPPLER ULTRASOUND

QUANTITATIVE FLOW MEASUREMENT

The widespread availability of Doppler Ultrasound as an additional facility on modern real-time scanners marks the latest and one of the more dramatic refinements in ultrasound imaging. In its simplest application pulsed Doppler ultrasound offers a directional stethoscope for the cardiologist. (Burns and Jaffe, 1985).

In moving blood it is the red cells which give rise to the returning Doppler-shifted echo. The cells are much smaller than the wavelength of ultrasound and the process by which they give rise to an echo is known as Ray Leigh Tyndall scattering.

The size of this echo is small compared with that produced by specular reflection from solid tissue interfaced and this is evidenced by the echo-free appearance of blood-filled structures on ultrasound images. (Burns and Jaffe, 1985).

INSTRUMENTATION

In continuous-wave Doppler instruments, two (usually adjacent) transducers are used, one to transmit and the other to receive. Their beams are arranged to overlap so as to form a sensitive volume defined by their spatial product. The continuous-wave system will be sensitive to a moving target lying within this volume. In the heart this means that blood flow signals are quite likely to be swamped by the much stronger Doppler-shifted echoes from moving solid structures in their immediate vicinity. (Burns and Jaffe, 1985).

The subcostal window has been reported as a new approach for recording continuous wave Doppler aortic flow velocities. (Ramirez, et al., 1985).

In pulsed Doppler short bursts of ultrasound are transmitted at regular intervals and the echoes are demodulated to create a Doppler shift signal. (Burns and Jaffe, 1985).

DOPPLER-ECHOCARDIOGRAPHIC ASSESSMENT OF CARDIAC OUTPUT

Doppler echocardiography has recently been shown to provide an alternative, non-invasive method for measuring volumetric flow at specific locations within the heart and great vessels. Because Doppler studies are non-invasive and non-toxic, they may be repeated as often as necessary, providing information concerning patient status and response to interventions on a heat-to-heat basis. In addition, because patients are examined at rest and unsedated, these studies provide information about basal hemodynamics, which may be altered by invasive procedures.

The ability of the Doppler method to measure flow through the mitral, aortic, pulmonary and tricuspid valves offers the potential not only to determine the cardiac output, but also to quantitate shunt flow and regurgitant volumes based on comparison of flow through individual valves. (Ascah, et al., 1985).

DOPPLER DIAGNOSIS OF VALVULAR AORTIC STENOSIS IN PATIENTS OVER SIXTY YEARS OF AGE

Twenty-five consecutive elderly patients with suspected aortic stenosis underwent continuous-wave Doppler echocardiography followed by cardiac catheterization. Doppler-derived calculation of peak and mean aortic valve gradients were compared with catheterization-derived values of peak-to-peak, peak and mean gradients.

The best correlation was found between Doppler-and-catheterization-derived mean gradients. A Doppler-derived measure of the timing of peak aortic flow velocity (modified time-to-peak velocity/modified left ventricular ejection time) successfully separated those with gradients above or below 50mm.Hg and also helped to avoid over or under-estimation of aortic valve gradients by Doppler. (Agatston, et al., 1985).

SUMMARY OF CLINICAL APPLICATION OF DOPPLER ULTRASOUND

(a) BASELINE AND SERIAL ASSESSMENT OF CARDIAC OUTPUT

Changes in the stroke volume and outnut are expected to be determined by Doppler ultrasound.

(b) ASSESSMENT OF CARDIAC SHUNT

The magnitude of pulmonary-systemic shunts can be quantitated by determining the ratio of pulmonary to systemic blood flow. This can be obtained by Doppler.

(c) QUANTITATION OF REGURGITANT FLOW

A number of Doppler methods for quantifying valvular regurgitation have been reported. (Goldberg and Allen, 1985). (Ascah, et al., 1985).

(d) Evaluation of left ventricular diastolic filling in children with systemic hypertension. (Williams and Lahovitz, 1985).

THE LIMITATION OF ECHOCARDIOGRAPHY

Echocardiography has its problems as well as its advantages. Ultrasound travels poorly through ribs, sternum, thick chest wall and very poorly through the lungs. Ultrasound examination, therefore, of the heart may be very difficult (even impossible) in certain individuals. The lung can be considered as a barrier to cardiac examination by ultrasound. Patients with emphysema may occasionally be impossible to examine. (Felner, et al., 1982).

THE LIMITATION OF M-MODE

The most important limitation (disadvantages) of M-mode echocardiography is the lack of lateral projection of the heart.

The entire left ventricle, right atrium and all leaflets of the tricuspid valve cannot readily be evaluated with M-mode techniques. In addition intracardiac structures are displayed in an unfamiliar format which bears little resemblance to cardiac anatomy, and the spatial orientation of intra-cardiac structures cannot be readily appreciated. It is not possible to measure the long axis by M-mode, so left ventricular size is derived only from measurement of the minor axis.

Limitations of M-mode echocardiography are also caused by a variety of technical factors such as lack of the examiner's experience and inadequate understanding of the clinical considerations in individual patients. (Felner, et al., 1982).

THE LIMITATION OF TWO-DIMENSIONAL ECHOCARDIOGRAPHY

Compared with M-mode echocardiography, which utilizes sampling of 1,000 impulses/sec., two-dimensional echocardiography utilizes sampling rates of 30-60 frams/sec. The fram repetition frequency, therefore, of two-dimensional echocardiography is too low to record very rapid events such as abnormalities of ventricular wall motion, valve opening and closure and fluttering. Here the M-mode has a distinct advantage. (De Maria, et al., 1980; Felner, et al., 1982).

Anatomic analysis is a major disadvantage of two-dimensional echocardiography images. These images can be confusing since the anatomic planes which are recorded may be unfamiliar and the variable image presentation makes standardization more difficult. In addition a variety of planes are needed to describe the anatomy, and if small changes in the scanning planes are made during the examination, it obscures the images. (Felner, et al., 1982).

The difficulty in continuously defining the end-cardial perimeter of the left ventricle with two-dimensional echocardiography has presented a significant limitation in the evaluation of left ventricular size and contractile pattern. (DeMaria, et al., 1980).

Furthermore, near structures which occupy the narrow projection of the fan-shaped image, are stretched to accommodate the rectangular format of strip chart recorders. Resolution in the lateral fields constitutes a major consideration in the development and application of two-dimensional echocardiography. (DeMaria, et al., 1980). Additional limitations of two-dimensional echocardiography include:-

(1) Greater physician/technician interaction needed for best results.

(2) We cannot get an accurate quantitation of cardiac dimensions and functions.

(3) The quality and accuracy of the technique depends on the skills and experience of the operator. (Felner, et al., 1982).

LIMITATIONS OF PULSED DOPPLER ECHOCARDIOGRAPHY

Although pulsed Doppler evaluation is fairly simple to perform, some caution should be exercised in interpreting the results, Much of the information depends on the fairly subjective interpretation of audio output, especially in the evaluation of mild turbulence. The examination of the normal patient may lead to loud clicks and low-pitched sounds, originating from the valve leaflets and heart walls passing through the sample volume that seems to overlap the slightly harsh sounds of mild turbulence.

There are also quantitative limitations owing to lack of knowledge about angulation, orifice size and flow profile, but considerable useful information is still obtained from the current system. (Felner, et al., 1982).

THE CHEST RADIOGRAPH IN CARDIOVASCULAR DISEASES

THE CHEST RADIOGRAPH IN CARDIOVASCULAR DISEASE

Alveolar air provides an excellent radiographic contrast medium against which the cardiac silhouette, the great systemic vessels and the pulmonary vessels may be identified and assessed. The chest radiograph is therefore a prime method of investigation of the heart and the pulmonary circulation. (Grainger and Fleming, 1982).

HEART SIZE AND SHAPE

Radiology has effectively replaced clinical examination as a method of assessing heart size. Standard films are more accurate than clinical estimates of heart size but minor changes are still difficult to detect with certainty and a normal film does not exclude severe disease. Extensive coronary artery disease, for instance, may not produce cardiac enlargement. The cardio-thoracic ratio is a useful index of the heart size and the heart is normally less than 50% of the transverse chest diameter.

Conditions selectively affecting specific parts of the heart produce characteristic appearances in the standard film. The radiographic findings are most useful when considered in conjunction with all the clinical findings and may be misleading if taken in isolation. Serial films are useful to detect progression of enlargement and can be interpreted in growing children with the help of percentile charts. (Humer, 1979).

The radiological cardiac shape and size reflect the position, shape and size of the individual chambers and great vessels. Conventional Radiography cannot distinguish the heart wall from the chamber cavity or pericardial fluid, for which angiocardiography and/or echocardiography will be required. (Grainger, 1982).

CARDIAC ENLARGEMENT

The effects of abnormal circulatory function on the heart may be reflected by an overall increase in cardiac size or by selective chamber enlargement or both.

Three methods of assessing the size of the heart on the plain radiograph are currently in use. The simplest is the measurement of the transverse cardiac diameter, followed by the measurement of the cardio-thoracic ratio; finally the calculation of the overall volume of the heart. (Raphael, 1986).

TRANSVERSE CARDIAC DIAMETER

This is the simplest possible measurement of cardiac size. In 90% of normal adult males the transverse cardiac diameter, as measured on the standard six foot postero-anterior radiograph, is less than 13.5cm. In 90% of normal adult females it is less than 12.5cm. The transverse cardiac diameter is most accurate in assessing cardiac enlargement when related to the patient's height and weight.

All methods of assessing heart size from the frontal radiograph alone may be misleading; in some individuals with a deep chest the heart may appear normal in the frontal view, but a lateral view will reveal an increase in the antero-posterior depth of the heart suggesting that significant cardiomegaly may be present. Measurement of the transverse cardiac diameter enables comparisons to be made of the heart size in the same patient on different films. In normal individuals this does not vary by more than 1.5 cm. at different examinations and a change of more than 2 cms. is therefore likely to represent a significant change in heart size. (Simon, 1968).

In enlarged hearts much smaller differences may represent a real change. The simplest way to assess change in heart size is direct superimposition of one radiograph on another. (Raphael, 1986).

CARDIOTHORACIC RATIO

This is the most popular method of assessing cardiac enlargement. The upper limit of normal for the cardiothoracic ratio is generally held to be around 50%, although a bigger ratio is permissible, up to 55% for Blacks and Asians. The method is difficult to apply in individuals with a transversely lying heart, which simulates cardiac enlargement, and in vertically lying hearts which may conceal cardiac

enlargement. In addition it may be difficult to measure the transverse cardiac diameter if the true left heart border is concealed by a large pericardial fat pad.

The cardio-thoracic ratio may be increased by non-standard radiographic techniques:-

incomplete inspiration;

radiographs exposed supine, prone, antero-posterior or with a short tube-film distance.

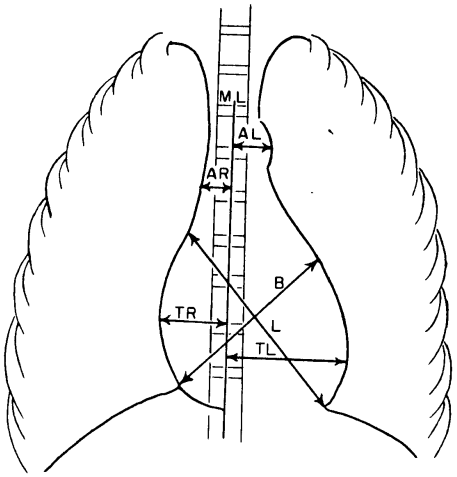
It is much influenced by the build and age of the patient and may exceed 60% in the normal neonate. (Raphael, 1986).

CARDIAC VOLUME (FIG. 18)

The total cardiac volume may be calculated from paired frontal and lateral radiographs. The method consists of measuring the three axes (long diameter L; broad diameter B; depth D), multiplying them together with the appropriate constant to obtain the volume, taking into account a further constant which compensates for the radiographic magnification.

The calculation of cardiac volume is time-consuming and outside of Scandinavia it has not found great clinical application. (Raphael, 1986).

Fig. 18



A diagram of PA chest radiograph to demonstrate the long (L) and the broad (B) diameter of the heart

L +	Long Diameter	This line extends from the junction of the superior vena cava and right atrium to the cardiac apex.
B +	Broad Diameter	This line extends from the junction of the right atrium and the diaphragm to a point on the left heart border at the junction of the pulmonary artery and left atrial appendage.
D +	Depth	Represents the greatest horizontal depth of the cardiac shadow as seen on the lateral chest radiograph.

SELECTIVE CHAMBER ENLARGEMENT

Selective chamber enlargement may be recognized on the plain radiograph by the changed appearance it produces:-

(1) The affected chamber will expand, displace and deform that part of the cardiac silhouette which it forms.

(2) The affected chamber may appear in the cardiac silhouette in a view in which it is not normally seen.

(3) The enlarged chamber may cause recognizable displacement of contiguous structures. Although the signs of selective chamber enlargement are well known, difficulties in interpretation may occur:-

(a) When a normally sized chamber is displaced, it may appear to be enlarged.

(b) If two chambers resemble each other in their rings of enlargement.

(c) When radiographic signs resembling chamber enlargement may be brought about by other structures.

(d) When the appearance of selective chamber enlargement may be produced by thoracic deformity. (Raphael, 1986).

SELECTIVE LEFT ATRIAL ENLARGEMENT

The left atrium (LA) lies at the upper part of the posterior aspect of the heart immediately in front of the oesophagus and in the angle of the carina. Because of its position, it is the easiest and most specific chamber in which to detect selective chamber enlargement. It must be enlarged two and a half times the normal for plain film evidence of selective enlargement to become apparent. (Levin, 1972).

Enlargement may involve primarily the body of the left atrium or both the appendage and the body. Enlargement of the left atrial appendage is strongly suggestive of a rheumatic aetiology and the grosser examples of its enlargement are almost entirely confined to rheumatic mitral valve disease.

THE FOLLOWING ARE THE PLAIN RADIOGRAPHIC SIGNS OF SELECTIVE LEFT ATRIAL ENLARGEMENT:-

(1) Posterior displacement of the barium filled oesophagus as seen in the lateral view

Left atrial enlargement is manifest as a localized displacement of the barium-filled oesophagus, often recognizably limited below by the atrio-ventricular groove, after which it leaves contact with the left atrium to make contact with the left ventricle before passing through the diaphragm. In most cases the enlarged left atrium will push the oesophagus to the right and backwards, but in a few patients the oesophagus slips to the left instead of to the right.

(2) Elevation of the left main bronchus and splaying of the carina

The trachea, carina and main bronchi should be visualized on a well penetrated frontal chest radiograph. The position of the left atrium, immediately under the carina, means that enlargement in an upward direction will first elevate the left main bronchus and when enlargement is gross it may splay the carina. Very gross enlargement leads not only to splaying of the carina but to compression of bronchi with possible lobar collapse, especially on the left.

(3) A double shadow seen through the heart

This sign requires an over-penetrated film for its demonstration. The normal left atrium does not project beyond the cardiac outline as a discrete structure. With selective left atrial enlargement posteriorly, the body of the left atrium bulges backwards. Where it is in contact with the lung, at the right and left sides of the heart, the interface between the left atrium and the lung will demarcate the outline of the left atrium in the frontal view, producing the effect of the double shadow.

Double shadows may also be produced by:-

(a) the root of the aorta; however this should be seen to be in continuity with the ascending aorta;

(b) normal sized left atrium;

(c) rarely the pulmonary veins join each other before joining the left atrium and produce a shadow behind the heart mimicking left atrial enlargement.

(4) Displacement of the right border of the heart to the right

The right border of the enlarged left atrium can be seen to be displaced to the right. The pericardium restrains gross enlargement of the left atrium and the right border of the moderately enlarged left atrium tends to coincide with the right border of the right atrium. Eventually left atrial enlargement may be sufficiently marked that the right border of the left atrium comes to bulge the pericardium and to project beyond the border of the right atrium to form the right heart border.

(5) Displacement of the descending aorta to the left

A localized displacement of the lower descending thoracic aorta to the left is very frequently seen on well penetrated frontal radiographs.

(6) Enlargement of the left atrial appendage

The left atrial appendage is usually concealed in the tissues of the left heart border and its selective enlargement leads first to a filling in of the concavity of the left heart border, which is normally present below the pulmonary bay and above the bulge of the left ventricle. Further left atrial enlargement produces a discrete bulge below the level of the pulmonary bay and below the left bronchus, and the bulge may well reach quite large proportions.

(7) Aneurysmal dilatation of the left atrium

Extreme enlargement of the left atrium so that the right border of the left atrium approaches the right rib cage, and the left border approaches the left rib cage, is termed aneurysmal dilatation. (Raphael, 1986).

CAUSES OF SELECTIVE LEFT ATRIAL ENLARGEMENT

The commonest cause, particularly when enlargement is gross, is rheumatic mitral valve disease. Lesser enlargement of the left atrium occurs in non-rheumatic mitral disease and from other forms of outflow obstruction such as left atrial myxoma.

OTHER CAUSES OF LEFT ATRIAL ENLARGEMENT

(1) Obstruction to left ventricular emptying either from aortic valve disease, hypertension or aortic coarctation.

(2) Hypertrophic cardiomyopathy.

(3) Volume overload situations with left to right shunts at ventricular or aorto-pulmonary levels, but not with inter-atrial septal defects.

(4) Long standing atrial fibrillation.

Apparent selective left atrial enlargement, affecting mainly the appendage, may be seen in the congenital absence of the left pericardium with herniation of a normal atrial appendage through the defect. Idiopathic enlargement is extremely rare and a pathological cause should be sought. (Raphael, 1986).

SELECTIVE LEFT VENTRICULAR ENLARGEMENT

The left ventricle (LV) normally forms the left border and apex of the cardiac shadow in the frontal radiograph. In the lateral view it forms the posterior aspect of the heart below the level of the atrio-ventricular ring, carrying on to form the inferior surface in contact with the diaphragm.

In the left ventricle, two-processes, hypertrophy and dilatation, may occur independently of each other. They both lead to left ventricular enlargement, but if this is gross it is almost always due to chamber dilatation. Enlargement of the left ventricle affects all diameters, but particularly the long axis.

The following are the plain film radiographic signs of left ventricular enlargement:-

(a) Rounding of the heart apex

This may be the only sign and may be seen before overall cardiac enlargement has occurred. It may be due to muscular hypertrophy and may be recognized before cavity dilatation of any significance has occurred.

(b) Elongation of the long axis of the left ventricle

The earliest sign of left ventricular cavity dilatation is an elongation of its axis which occurs mainly to the left and in a downward direction. The apex of the heart may be carried below the diaphragm but it may be difficult to recognize a definite cardiac apex.

(5) In the lateral view:-

The dilatation of the left ventricular cavity may be recognized when the soft tissue shadow of the heart muscle bulges behind the barium-filled oesophagus. The posterior part of the left heart border contributed by the left ventricle may be seen to bulge behind the entry of the inferior vena cava into the heart shadow as seen in the true lateral view. (Chikos, 1977). This may occasionally be the only sign of left ventricular enlargement as the heart shadow may appear normal in the frontal view.

A large right ventricle may displace a normal left ventricle posteriorly, behind the barium-filled oesophagus. Plain film radiography is not a sensitive method of detecting selective left ventricular enlargement, nor of recognizing it in the presence of enlargement of other chambers. (Raphael, 1986).

CAUSES OF SELECTIVE LEFT VENTRICULAR ENLARGEMENT:-

(1) Aortic regurgitation is the commonest cause of very gross left ventricular enlargement and this is commonly associated with a dilated ascending aorta.

(2) Obstruction to left ventricular emptying such as aortic stenosis, (aortic valve calcification and post-stenostenotic dilatation), hypertension (aortic enlargement) or co-arctation (rib notching and post-coarctation aortic dilatation).

(3) Congestive or hypertrophic cardiomyopathy.

(4) Ischaemic heart muscle disease.

(5) Volume overload of the left ventricle such as ventricular septal defect or aorto-pulmonary communication. (Raphael, 1986).

SELECTIVE RIGHT ATRIAL ENLARGEMENT

The right atrium (RA) forms the right border of the heart and in the frontal view, but its atrial appendage, which passes forwards and to the left, may contribute to the upper anterior cardiac silhouette in the lateral view. The right atrium is the most difficult chamber to assess for selective enlargement, particularly if only a single radiograph is available.

The plain radiographic signs of right atrial enlargement are:-

(a) Displacement of the right heart border to the right.

(b) Increase in the radius of the curvature of the right heart border. Thus the right heart border may be more curved than usual.

(c) A step-like angle between the right atrium and the superior vena cava may be seen.

(d) Filling in the space between the sternum and the front of the upper part of the cardiac silhouette in the lateral radiograph and increase in the application of the cardiac outline to the sternum.

In this way it may resemble right ventricular enlargement. (Raphael, 1986).

CAUSES OF ENLARGEMENT OF THE RIGHT ATRIUM (RA)

Selective right atrial enlargement is usually due to abnormalities of the tricuspid valve. In acquired heart disease it is usually due to rheumatic tricuspid incompetence or stenosis. It may also be due to pulmonary hypertension. The right atrium may become large in any situation where there is difficulty in emptying into the right ventricle.

In congenital heart disease enlargement may occur in any left to right shunt in which the increased flow passes through the right atrium, in particular atrial septal defect and also in communications from the left ventricle to the right atrium, or the aorta to the right atrium.

Some of the grossest examples of selective right atrial enlargement occur in Ebstein's malformation of the tricuspid valve. (Raphael, 1986).

SELECTIVE ENLARGEMENT OF THE RIGHT VENTRICLE

The right ventricle (RV) is a flattened, triangular shaped structure occupying the front of the heart. It forms the front surface of the heart in the lateral projection, but contributes no part of the normal cardiac outline in the frontal projection. The normal heart shadow is in contact with the sternum only in the lower half of its extent in the lateral view. With progressive enlargement of the right ventricle the area of contact between the front surface of the heart and the sternum increases until eventually the whole heart lies in contact with the sternum.

This is the earliest and the most sensitive radiological sign of right ventricular enlargement. The sign must always be interpreted in relation to the shape of the chest. A depressed sternum will increase the area of contact with the normal heart and a bowed sternum will fail to make contact with an enlarged right ventricle. Abnormalities of the chest shape are common in congenital heart disease and so reduce the value of this sign.

Dilatation of the right ventricle frequently becomes so gross that the left border of the right ventricle may move so far to the left to make up the left heart border and thus in the frontal view the cardiac

outline takes the triangular shape of the right ventricle. In particular the infundibulum of the right ventricle may dilate and fill in the concavity of the left heart border between the pulmonary bay and the apex, leading to a triangular shape or even a marked bulge.

Dilatation of the right ventricle usually involves the whole of the chamber and as this is curved round the septal aspect of the left ventricle, this dilatation results in a tilting up and a posterior displacement of the left ventricle, if this is normal in size. Thus in the frontal view right ventricular enlargement results in an overall increase in the cardiac shadow, which adopts a triangular configuration whose long axis is elongated in a downward direction, and there may be elevation of the apex of the heart, which is usually formed by the left ventricle. (Raphael, 1986).

Other radiographic evidence pointing to selective right ventricular enlargement may frequently be obtained by studying the main pulmonary artery, which is usually enlarged, and the peripheral pulmonary vessels, which may be increased or decreased and thus point to the presence or cause of some right ventricular abnormalities.

The characteristic elevation of the apex of the heart "coeur en sabot", the appearance of right ventricular enlargement in the tetralogy of fallot and in pulmonary atresia, is probably due to the large right ventricle rotating the heart clockwise around its vertical axis, displacing the normally sized left ventricle posteriorly and tilting it up. (Raphael, 1986).

CAUSES OF ENLARGEMENT OF THE RIGHT VENTRICLE

The right ventricle is not enlarged in the majority of cases of pulmonary valve stenosis as there is no cavity dilatation until right ventricular failure occurs. The hypertrophy which occurs is not apparent on the plain film.

Enlargement occurs in the following cases:-

(1) Severe obstruction to emptying as in critical pulmonary stenosis in the neonate.

(2) Severe long-standing pulmonary hypertension, particularly when this is complicated by pulmonary incompetence.

(3) Tricuspid incompetence or pulmonary incompetence.

(4) Left to right shunts, typical in atrial septal defect.

(5) Ebstein's malformation. The distal portion of the right ventricle will be enlarged. (Raphael, 1986).

BIVENTRICULAR ENLARGEMENT

In the majority of adult patients with acquired heart disease the disease process, although beginning on the left side of the heart, will eventually come to involve the pulmonary circulation and the right heart chambers. In this situation, which appears late in most forms of rheumatic heart disease, all chambers are likely to be involved and it is not possible with plain radiography to determine the contribution of each chamber, other than the left atrium. The heart with all chambers enlarged takes on a globular shape. (Raphael, 1986).

SKELETAL ABNORMALITIES

Skeletal abnormalities may result from cardiac disease (e.g. the bowing of the sternum in ventricular septal defect) or may lead to cardiac disease as in kyphoscoliotic heart disease, or may be part of the generalized disorder that has progressed to heart disease as in Marfan's syndrome. (Raphael, 1986).

DEPRESSED STERNUM

The only condition which produces radiological appearances which suggest heart disease is depressed sternum. This, in its fully developed form, results in a heart which is displaced to the left, appears enlarged, has a straightened or even bulged left border and a dilated main pulmonary artery. The appearances suggest right ventricular enlargement secondary to an abnormality of the pulmonary valve or pulmonary circulation. The unusual disposition of soft tissue in the anterior chest wall may cause an ill-defined hazy opacity adjacent to and obscuring the right heart border.

The condition should be suspected in the frontal view because of the cardiac displacement and abnormality of shape, particularly when the intervertebral discs of the lower dorsal spine can be seen with increasing clarity through the heart shadow above the diaphragm,and when the abnormal downwards and forwards slope of the anterior ends of the ribs can be recognised. A lateral view may show the gross appearances of depressed sternum with the anterior ends of the ribs bulging forwards beyond the sternum and reduction in space between the sternum and the spine, so marked that the heart may be obviously compressed and this may explain the radiological appearances.

Appearances in the frontal view may be quite florrid, yet in the lateral view appearances may not suggest gross sternal depression. Minor degrees of apparent cardiac abnormality may be seen in the frontal view in those patients where the dorsal spine is straighter than normal with no dorsal kyphosis. There is also a slightly reduced anteroposterior diameter of the thorax in rather tall and slender patients. These variants have been termed "straight back syndrome". (Raphael, 1986).

PULMONARY VASCULARITY IN HEART DISEASE

Most cardiac diseases cause alterations in the pulmonary vascularity. In acquired heart disease the abnormality of vascularity is generally pulmonary venous congestion or hypertension. Since the most frequently encountered congenital heart diseases involve some type of shunt, the abnormality of vascularity in congenital heart disease is generally pulmonary arterial over-circulation or under-circulation. Pulmonary arterial hypertension is frequently associated with and caused by both pulmonary venous hypertension and pulmonary arterial over-circulation. (Higgins and Lipton, 1986).

Fig. 19

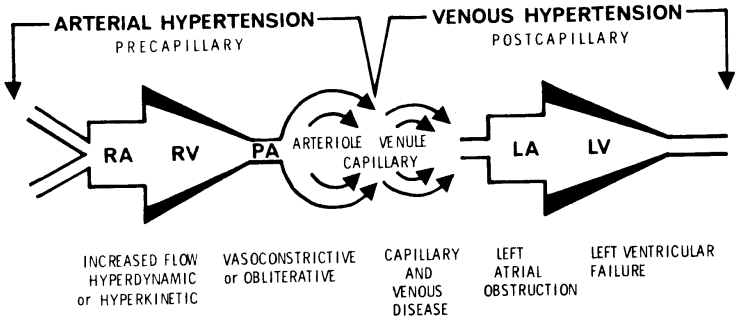


Diagram showing possible sites of obstruction to pulmonary blood flow and their relationship to the different types of pulmonary hypertension

PULMONARY VENOUS HYPERTENSION

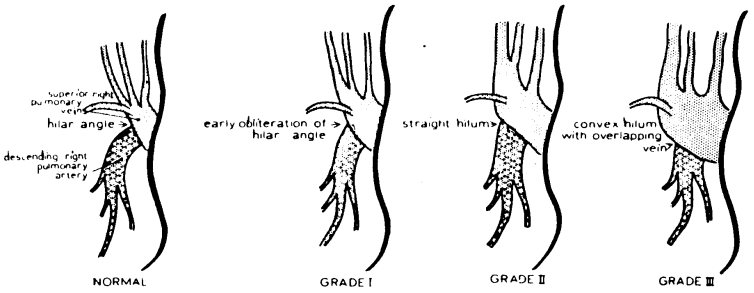
Pulmonary venous hypertension is caused by increased resistance to blood flow in the pulmonary veins, within the left atrium, at the mitral valve or within the left ventricle as a consequence of reduced compliance (diastolic relaxation) of the ventricle. The lesions that can cause pulmonary venous hypertension are listed in Table 2. (Higgins and Lipton, 1986).

Pulmonary venous hypertension (PVH) is typically divided into three grades of severity. (Jefferson and Rees, 1973).

GRADE I PVH

It is characterized by reversal of the normal gravity dependent gradient of pulmonary blood flow (diameter of lower lobe vessels greater than upper lobe vessels). The vessels in the upper zone are either equivalent in diameter or wider than the lower zone vessels in Grade I PVH. Comparison of the size of the upper and lower zone vessels is visually facilitated on the lateral chest radiograph. The right descending pulmonary trunk is generally the most prominent vessel on the frontal radiograph. Attenuation or loss of visibility of the mid and distal portions of this vessel is a clue to the presence of pulmonary venous hypertension. Because of enlargement of the right superior pulmonary vein in pulmonary venous hypertension, the hilar angle (lateral concavity) formed by the intersection of this vein and the descending pulmonary artery is effaced. (Doppman and Lavender, 1963).

Fig. 20



The right hilar angle is constricted by the intersection of the right upper pulmonary vein and the descending trunk of the right pulmonary artery. The hilar angle is progressively obliterated with increasing severity of pulmonary venous congestion.

GRADE II PVH

It is characterized by signs of interstitial pulmonary oedema and pleural effusion. The most reliable signs of interstitial oedema are Kerley B lines (septal lines) caused by fluid accumulation in the interlobular septa and/or lymphatics. These are short horizontal lines with a thickness of 1-4 mm which are located at the costophrenic angles and occasionally extend as high as the middle lung zones. They lie perpendicular to and usually on the pleural surface. The accumulation of fluid in the interstitial space also causes indistinctness of vessel margins; increased opacity of all or portions of the lung (pulmonary clouding); perihilar clouding; peribronchial thickening; reticulated parenchymal pattern in the lower lung zone; and subpleural oedema. The presence of Kerley A lines generally indicates a more acute or advanced degree of Grade II PVH.

The Kerley A lines represent distended lymphatic channels within oedematous septa coursing from the peripheral lymphatics towards the central hilar lymph nodes. Kerley lines usually disappear after reduction of pulmonary venous pressure. However, they may be persistent due to the development of fibrosis or haemosiderin deposition in the interlobular septa. Persistent septal lines are sometimes seen in non-cardiac diseases such as lymphangitic carcinomatosis, pneumoconiosis or central lymphatic obstruction by tumour or irradiation.

GRADE III PVH

It is signified by an alveolar filling pattern. The confluent air space density of pulmonary oedema may be generalized but more frequently has a perihilar or lower zone distribution. In chronic lung disease the pattern of oedema is frequently asymmetric or atypical.

Asymmetric pulmonary oedema may ensue from a variety of factors, (Table 3), but the most frequent factor is gravity. Oedema predominantly of the right lung is commonly caused by the preference of cardiac patients to recline on their right side due to

disturbing cardiac pulsation when lying on the left side. Another important factor is the presence of underlying lung disease which has caused non-uniform obliteration of the pulmonary vascular bed. Occasionally, this finding may be a clue to obstruction of a pulmonary vein or veins by left atrial tumour or thrombosis. (Higgins and Lipton, 1986).

Since pulmonary alveolar oedema is most frequently caused by some form of cardiac disease, the chest radiograph usually reveals left atrial enlargement or cardiomegaly or both. An exception to this rule is the pulmonary oedema caused by an acute myocardial infarction, (Higgins and Lipton, 1980). With cardiac causes of pulmonary oedema, the upright chest radiograph shows enlarged vessels in the upper lung zones. Consequently, pulmonary oedema in the absence of these signs suggests a non-cardiogenic cause. (Table 4). Frequently encountered causes of non-cardiogenic oedema are the adult respiratory distress syndrome, drug overdose, idiosyncratic reaction to drugs and drowning. (Higgins and Lipton, 1986).

TABLE 2

LESIONS AT VARIOUS ANATOMICAL LEVELS
WHICH CAUSE PULMONARY VENOUS HYPERTENSION

PULMONARY VEIN

Pulmonary venous occlusive disease.
Partial or total anomalous pulmonary venous connection with obstruction.
Thrombosis.
Extrinsic compression of pulmonary veins.
Mediastinal Tumours.
Fibrosing Mediastinitis.
Granulomatous Mediastinitis (histoplasmosis).
Ostial obstruction by atrial baffle after Mustard procedure.

LEFT ATRIUM

Cor Triatriatum.
Left Atrial Myxoma.
Left Atrial Thrombus.
Reduced capacity after intra-atrial surgery.

MITRAL VALVE

Congenital Hypoplasia or Stenosis.
Mitral Stenosis.
Mitral Regurgitation.
Thrombosis of prosthetic valve.

LEFT VENTRICLE

Myocardial Failure.
Congestive Cardiomyopathy.
Ischaemic Cardiomyopathy.
Decompensated Heart Disease.
Reduced LV Compliance.
Acute Myocardial Infarction.
Severe Left Ventricular Hypertrophy.
Hypertrophic Cardiomyopathy.
Restrictive Cardiomyopathy.
Constrictive Pericarditis.

Discrepancies between the radiographic severity of PVH and simultaneous measurement of pulmonary artery wedge pressure in individual cases are frequent. These discrepancies can be referred to as the diagnostic phase lag and the therapeutic phase lag. The diagnostic phase lag is the time between elevation of pulmonary venous pressure and the appearance of the radiographic abnormality. The therapeutic phase lag is the time between the fall in pulmonary venous pressure and resolution of the radiographic signs. These temporal lags occur because most of the radiographic signs of PVH are due to alterations in extravascular lung water. Fluid fluxes in the lung ensue over minutes to hours, whilst changes in venous pressure may be instantaneous. Pulmonary venous pressure within the normal range is characteristic of non-cardiogenic pulmonary oedema. (Higgins and Lipton, 1986).

PULMONARY ARTERIAL OVERCIRCULATION (PLETHORA)

Pulmonary arterial overcirculation is caused by left to right shunts, bi-directional shunts and high output states. Pulmonary plethora may also be apparent in normal teenagers, in well-conditioned athletes, and in pregnancy.

Pulmonary overcirculation is indicated by enlargement of the central (hilar) pulmonary, lobar and segmental pulmonary arteries. The arteries that course from ventral to dorsal produce prominent nodular vascular shadows on the frontal chest radiograph and have been referred to as "shunt vessels". The upper zone vessels show increase in calibre, but in contra-distinction to PVH, the lower lobe vessels are also prominent. Semi-quantitative guides for the recognition of overcirculation in infants include the diameter of the right inferior pulmonary artery exceeding the diameter of the trachea and the diameter of an en face pulmonary vessel exceeding the diameter of its companion bronchus. The visualization of en face vessels below the level of the tenth posterior rib and prominence of vessels situated below the crest of the diaphragm on the frontal view also suggests pulmonary overcirculation. The prominence of the hilar vessels on the lateral view may be particularly helpful in deciding whether pulmonary overcirculation exists.

Very large volume shunts may cause minor pulmonary venous hypertension in addition to overcirculation. This circumstance is indicated by indistinctness of the vascular margins, perivascular cuffing, peribronchial thickening, interstitial and eventually alveolar oedema.

Shunts causing a pulmonary to systemic flow ratio of greater than 2:1 can be reliably detected radiologically. Shunts of lesser severity cannot be consistently detected on the chest radiograph. (Higgins and Lipton, 1986).

TABLE 3

FACTORS CAUSING ASYMMETRIC PULMONARY OEDEMA

Gravitation (ipsilateral)
Lung disease (COPD)
Pulmonary embolism (contralateral)
Unilateral obstruction of pulmonary veins (ipsilateral)
Cardiac Tumours
Mediastinal Tumours
Fibrosing Mediastinitis
Left Atrial Thrombus
Re-expansion of pneumothorax (ipsilateral)
Post-thoracocentesis
Lung Infarction (ipsilateral)

(Higgins and Lipton, 1986)

TABLE 4

CAUSES OF NONCARDIOGENIC PULMONARY OEDEMA

Adult respiratory distress syndrome
Drowning
Asphyxia
Upper airway obstruction (usually with cardiomegaly)
High altitude
Increased intracranial pressure
Post-ictal
Noxious gases
Smoke
Nitrous dioxide
Sulphur dioxide
Nitrogen mustard

DRUGS

Aspirin
Valium; Librium; Barbiturates
Narcotics (heroin, methadone, morphine)
Beta adrenergic drugs
Contrast media
Colchicine
Fluoresccin
Hydrochlorthiazide
Nitrofurantoin
Propoxyphene

POISONS

Parathion
Transfusion reactions
Renal transplantation
Bone Marrow transplantation
Fat embolism
Pancreatitis

(Higgins and Lipton, 1986)

PULMONARY UNDERCIRCULATION (OLIGAEMIA)

Diminution of pulmonary blood flow must be severe for reliable radiographic detection. This is indicated by small hilar, lobar and segmental pulmonary arteries and particularly by a concave or absent main pulmonary artery segment. The lateral chest radiograph may be particularly useful by indicating a decrease in size and density of the hilar vessels and diminutive vessels coursing to the lower lobes.

Because it is difficult to reliably distinguish between normal and diminished pulmonary vascularity, apparently normal vascularity in the centrally cyanotic patient is considered to be practically equivalent to pulmonary oligaemia and indicative of a right to left shunt. (Higgins and Lipton, 1986).

PULMONARY VASCULARITY IN SPECIFIC ACQUIRED CARDIAC LESIONS

The severity of some forms of heart disease is reflected by pulmonary venous hypertension. This is particularly so in mitral stenosis and with myocardial or pericardial processes which reduce left ventricular compliance. (Newell, et al., 1980). The abrupt onset of severe regurgitant lesions (such as acute mitral regurgitation from papillary muscle or chordal rupture and acute aortic regurgitation from bacterial endocarditis or trauma) typically cause severe pulmonary oedema.

MITRAL VALVE DISEASE

Mitral stenosis, if haemodynamically significant, nearly always causes PVH (Simon, 1972). The severity of stenosis is roughly related to the grade of PVH. Recurrent episodes of pulmonary oedema in patients with mitral stenosis causes interlobular septal fibrosis (persistent Kerley B lines), haemosiderosis and ossific nodules, which are generally confined to the lower lung fields. The signs of PVH are the most important gauge of the severity of mitral valve obstruction available from non-invasive evaluation. The signs of PVH are commonly accentuated with the onset of atrial fibrillation. (Higgins and Lipton, 1986).

Besides PVH the radiograph may show signs of elevated pulmonary vascular resistance and pulmonary arterial hypertension. These are manifested on the radiograph by enlargement of the main pulmonary artery segment, right and left pulmonary arteries and right inferior pulmonary artery.

Chronic mitral regurgitation causes less severe signs of PVH but more prominent left atrial enlargement than does mitral stenosis. In chronic regurgitation there is not a close correlation between severity of regurgitation and PVH. The heart size (cardiothoracic ratio) and left atrial size are more reliable gauges of the severity of chronic mitral regurgitation. However, severe long-standing regurgitation occasionally does cause interstitial or even alveolar oedema.

Acute mitral regurgitation causes severe PVH and florrid oedema, which initially occurs in the absence of left atrial enlargement or cardiomegaly.

Acute onset of pulmonary oedema may be caused by abrupt or critical reduction in the mitral orifice by a left atrial thrombus or myxoma or by fibrosis and thrombosis of a mitral prosthetic valve. (Higgins and Lipton, 1986).

AORTIC VALVE DISEASE

Pulmonary vasculature is usually normal during all or most of the course of chronic aortic valve disease. Pulmonary venous hypertension occurs infrequently with aortic stenosis but may ensue from reduced compliance caused by left ventricular hypertrophy, or from subendocardial ischaemia resulting from imbalance in myocardial ratio of blood supply to oxygen demand. The development of myocardial failure is accompanied by signs of PVH. (Higgins and Lipton, 1986).

Pulmonary venous hypertension and oedema are absent in chronic aortic regurgitation in spite of substantial cardiomegaly. The severity of regurgitation is reflected more by the degree of

cardiomegaly than by pulmonary vasculature. (Samuels, et al., 1979). Prominent signs of PVH or pulmonary oedema indicate the presence of myocardial failure. Acute aortic regurgitation causes severe PVH; this usually occurs in the absence of substantial cardiomegaly. (Higgins and Lipton, 1986).

ACUTE MYOCARDIAL INFARCTION

Nearly 50% of patients show radiographic signs of PVH within the first 24 or 48 hours after uncomplicated acute myocardial infarction. This is due to an acute decrease in left ventricular compliance which resolved completely or partially within the first week after infarction. The chest radiograph generally reveals PVH without cardiomegaly or left atrial enlargement. There is a good correlation between the presence and severity of PVH and short (30 days) and long term (6 to 12 months) survival after acute infarction. (Battler, et al., 1980).

Severe and persistent pulmonary oedema may be caused by rupture of an infarcted papillary muscle or of an infarcted ventricular septum. The alveolar oedema caused by acute ventricular septal defect frequently masks signs of pulmonary arterial overcirculation.

HYPERTROPHIC CARDIOMYOPATHY

Hypertrophic cardiomyopathy in the non-obstructive or obstructive form commonly causes PVH due to reduced ventricular compliance. Increase in severity of PVH or pulmonary oedema may ensue when the atrial kick is lost with the onset of atrial fibrillation.

CONGESTIVE CARDIOMYOPATHY

Congestive cardiomyopathy causes blood flow redistribution to the upper lung zone, interstitial oedema and alveolar oedema, which vary in relation to clinical status. Radiographic signs of PVH tend to under-estimate the level of pulmonary venous pressure; radiologically evident alveolar oedema has been found in less than one third of patients when pulmonary venous pressure was greater than 25 mmHg. (Higgins and Battler,1981). The degree of cardio-

megaly is a more sensitive and consistent gauge of severity of disease; a cardiothoracic ratio greater than 0.60 is associated with a poor prognosis. (Dash, et al., 1980).

PERICARDIAL DISEASE

Pericardial effusion is suggested by an enlarged 'cardiac' silhouette in the absence of PVH. Whilst this can be a helpful diagnostic sign, pericardial effusions frequently are caused by cardiac decompensation and chronic renal disease, which may themselves produce PVH. Moreover, the entity of effusive constrictive pericarditis causes enlargement of the cardiac silhouette due to the effusion and PVH due to cardiac constriction by the visceral pericardium. (Hancock, 1971). Pericardial tamponade causes attenuation of the pulmonary vasculature.

Constrictive pericarditis causes PVH of varying severity. This occurs in the presence of normal cardiac size or only mild cardiomegaly. (Higgins and Lipton, 1986).

HIGH CARDIAC OUTPUT STATES

The pulmonary circulation in high cardiac output states reflects the increase in blood volume and the hyperkinetic blood flow. This situation is observed in anaemia, arterio-venous fistulae, thyrotoxicosis and beri-beri. Liver disease, especially portal cirrhosis and Paget's disease, may be accompanied by substantial arteriovenous shunting resulting in a high cardiac output state. (Higgins and Lipton, 1986).

Hyperkinetic circulation is reflected in pulmonary plethora with enlargement of central and segmental pulmonary arteries in both the upper and lower lung zones. In extreme cases interstitial or alveolar oedema may be superimposed. There is frequently increase in heart size. The increased blood volume causes a concurrent prominence of the superior vena cava, azygos vein and/or brachiocephalic veins. Systemic venous engorgement may cause widening of the superior mediastinum. (Pistolesi, et al., 1982).

PULMONARY VASCULARITY IN CONGENITAL HEART DISEASE

LEFT TO RIGHT SHUNTS

The hallmarks of the left to right shunts are acyanosis and pulmonary arterial overcirculation.

In the premature infant large volume left to right shunts are caused by patent ductus arteriosus. Prominent pulmonary arterial over-circulation in the first year of life is most often caused by ventricular septal defect.

It is common for the radiograph to demonstrate asymmetric pulmonary vascularity; the over-circulation frequently appears more severe in the right lung. This finding may suggest contra-lateral branch pulmonary artery stenosis but is usually a flow phenomenon and occurs in the absence of stenosis.

The pattern of pulmonary vascularity itself does not usually distinguish amongst the various left to right shunt lesions. However, there is a tendency after infancy for atrial septal defects to cause a greater degree of enlargement of the main pulmonary artery segment and particularly the right pulmonary artery. A vascular shadow pursuing an unusual course through the lungs or disappearing into the superior mediastinum (right or left superior vena cava) may indicate partial anomalous pulmonary venous connection (PAPVC). A vertically oriented vascular shadow enlarging in diameter as it disappears into the right diaphragm indicates PAPVC to the inferior vena cava. This finding is usually, but not invariably, associated with cardiac dextroposition, hypoplasia of the right lung and other bronchovascular anomalies.

The Eisenmenger stage of left to right shunts, as evidenced by enlarged main and hilar pulmonary arteries and abrupt narrowing of the distal portion of the segmental pulmonary arteries, occurs with ventricular septal defects and aortic level shunts (patent ductus arteriosis, aorticopulmonary window, etc.). This complication almost never occurs with secundum ASD in childhood and is even unusual in adults with ASD. The diagnosis of the

Eisenmenger stage of ASD from the chest radiograph is not very reliable but is strongly suggested by massive central pulmonary arteries and pulmonary arterial calcification. (Higgins and Lipton, 1986).

RIGHT TO LEFT SHUNTS

Most right to left shunts have severe obstruction to pulmonary blood flow, manifested on the chest radiograph by pulmonary oligaemia. The radiological distinction between decreased pulmonary vascularity and normal may be difficult and is particularly unreliable in infancy. The normal vascularity in the presence of cyanosis should be considered equivalent to decreased vascularity and indicative of a right to left shunt.

Diminished pulmonary blood flow usually indicates obstruction to pulmonary blood flow and an intracardiac defect. This is usually caused by pulmonary outflow tract stenosis or atresia with a ventricular septal defect. Less frequently it is caused by severe valvular stenosis, tricuspid atresia or hypoplastic right ventricle, each in association with an inter-atrial communication. Because the functional portion of the right ventricle is greatly reduced in volume, Ebstein's anomaly also frequently causes reduced pulmonary blood flow. (Higgins and Lipton, 1986).

BIDIRECTIONAL SHUNTS

The hallmark of these lesions is pulmonary arterial over-circulation (plethora) in association with cyanosis. The most frequent cause of this complex of findings is complete transposition of the great vessels. It is also caused by truncus arteriosus, total anomalous pulmonary venous connection, tricuspid atresia with a large VSD or transposition, double outlet right ventricle, single atrium and single ventricle. (Higgins and Lipton, 1986).

PULMONARY ARTERIAL HYPERTENSION

Pulmonary arterial hypertension is defined as an elevation in mean pressure above 20 mmHg and an elevation in peak systolic pressure above 30 mmHg. There are four major mechanisms which cause elevation in pulmonary artery pressure - namely:-

(1) EXCESSIVE PULMONARY BLOOD FLOW

(Hyperkinetic pulmonary hypertension)

(a) Left to right shunts including:-

atrial septal defect
partial anomalous pulmonary venous connection
ventricular septal defect
patent ductus arteriosis
other aortic level shunts

(b) Bidirectional shunts including:-

transposition of the great vessels
truncus arteriosis
total anomalous pulmonary venous connection
single ventricle and many forms of tricuspid atresia

(c) Surgically created left to right shunts such as:-

Potts
Waterston

(d) High output states such as:-

thyrotoxicosis
arteriovenous fistulae
severe anaemia

(2) OBLITERATION OF PULMONARY VASCULATURE

(a) Arteriolar obliterative disease secondary to excessive blood flow (Eisenmenger reaction)

(b) Pulmonary embolic disease

(c) Schistosomiasis

(d) Parenchymal lung disease including:-

chronic obstructive pulmonary emphysema
cystic fibrosis
broncho-pulmonary dysplasia
advanced pneumoconiosis
granulomatous disease

(e) Primary pulmonary hypertension

(f) Pulmonary arteritis including:-

polyarteritis
disseminated lupus erythematosis
scleroderma
Takayasu's arteritis

(g) Bronchogenic carcinoma

(3) EXCESSIVE PULMONARY VASOCONSTRICTION

(a) Persistent fetal circulation

(b) Hypoxia including the effect of high altitude

(c) Response to certain drugs including acetylcholine

(4) SECONDARY TO PULMONARY VENOUS HYPERTENSION

(a) Pulmonary veno-occlusive disease

(b) Cor triatriatum

(c) Mitral stenosis and left atrial myxoma

(d) Left ventricular failure

(e) Decreased left ventricular compliance resulting from:-

left ventricular hypertrophy
acute myocardial infarction
restrictive cardiomyopathy
hypertrophic cardiomyopathy

(f) Constrictive pericarditis

(Higgins and Lipton, 1986).

RADIOGRAPHIC FEATURES OF PULMONARY ARTERIAL HYPERTENSION

The hallmark of pulmonary arterial hypertension is enlargement of the main and central pulmonary arteries. Dilatation of the main and central pulmonary arteries is most pronounced in the ASD Eisenmenger syndrome. Frequently but not invariably there is disproportion in the size and prominence of the central and segmental pulmonary vessels compared to the more peripheral vasculature. In advanced cases of pulmonary hypertension, calcification of atherosclerotic plaques in the main or hilar pulmonary arteries can be discerned on the chest radiograph.

In pulmonary hypertension caused by excessive pulmonary blood flow (hyperkinetic) the chest radiograph displays that visible peripheral pulmonary arteries are increased in number and diameter as well as enlargement of the main and central pulmonary arteries. The heart size is enlarged in proportion to the volume of the left to right shunt and thereby to the prominence of the pulmonary vasculature. With the development of the Eisenmenger reaction, peripheral

pulmonary vascularity diminishes and the main and central pulmonary arteries enlarge further whilst overall heart size decreases and the right ventricle becomes more prominent.

In the arterial obliterative form of pulmonary hypertension, the main pulmonary artery is enlarged. The pulmonary arterial enlargement is generally less severe when hypertension is due to thromboembolic disease, pulmonary parenchymal disease, pulmonary arteritis and primary pulmonary hypertension than that observed with the Eisenmenger syndrome. Indeed in acute pulmonary arterial hypertension due to pulmonary embolism, the main pulmonary artery is frequently normal in size. Likewise, in the pulmonary vasoconstrictive form of pulmonary hypertension, the degree of enlargement of the main and central pulmonary arteries is only mild or non-existent.

In pulmonary arterial hypertension secondary to venous hypertension, radiographic signs of pulmonary venous hypertension are usually evident. However, this is not invariable since in the presence of severe pulmonary arterial hypertension or the development of right ventricular failure, signs of pulmonary venous hypertension may resolve. This form of pulmonary arterial hypertension can be associated with cardiomegaly or a normal heart size. Distinct left atrial enlargement is usually evident. Pulmonary arterial hypertension secondary to venous hypertension occurs to the most severe degree in mitral valve disease, particularly in chronic mitral stenosis. (Higgins and Lipton, 1986).

ACQUIRED VALVULAR HEART DISEASE

MITRAL VALVE DISEASE

In most patients significant mitral valve disease is probably the sequel to rheumatic fever or, much more rarely, chorea.

RHEUMATIC MITRAL VALVE DISEASE

RHEUMATIC FEVER

This is generally held to be a hypersensitivity reaction to streptococcal toxin as it usually follows one to three weeks after a streptococcal sore throat or an attack of scarlet fever. The acute illness usually occurs in children and is characterized by pyrexia, flitting arthritis and a raised ESR. Cardiac involvement is suggested by the development of murmurs, ECG abnormalities and an enlarging heart. Pericarditis may cause chest pain. About two thirds of all children with clinical rheumatic fever have evidence of valvulitis, but in the majority it disappears. Severe persisting cardiac involvement in the acute phase leads to the development of the more severe valvular lesions, especially mitral and aortic incompetence. The milder forms of the disease usually lead to valve stenosis. The mitral valve is the commonest to be affected, followed by the aortic and then the tricuspid valves. The pulmonary valve is virtually never involved directly. Subclinical cases are thought to be common, leading to chronic valve disease without preceding overt clinical rheumatic fever. (Raphael, 1986).

PLAIN RADIOGRAPHY

The chest radiograph may be entirely normal. Cardiomegaly within the first month of the illness is usually due to pericarditis with effusion, particularly if apparent change in heart size is rapid. Echocardiography will demonstrate the presence of pericardial fluid. Rheumatic pericarditis may eventually calcify but does not lead to constriction. A slow increase in heart size, usually involving all chambers and developing over several months, suggests the onset of acute carditis, often with an acute valvulitis (a pancarditis).

Persistence of cardiomegaly is a poor prognostic sign. Severe acute pancarditis may lead to cardiac failure with pulmonary venous congestion and pulmonary oedema. In most patients cardiac failure clears with appropriate treatment, but in a few patients pancarditis is progressive and fatal. In the majority the plain radiograph reverts to normal and the disease enters a quiescent phase before chronic rheumatic disease manifests clinically. (Raphael, 1986).

RHEUMATIC MITRAL VALVE DISEASE

Chronic rheumatic valvulitis leads to scarring and retraction of the valve leaflets, fusion of the commissures and thickening and shortening of the chordae tendineae, with fibrosis of the papillary muscles. Depending on the severity of the process, pure valve incompetence results if there is extensive leaflet destruction and pure stenosis if commissural fusion occurs with virtually normal leaflets, but the commonest result is a mixture of stenosis and incompetence, either of which may be worsened by chordal involvement. Valve stenosis, if present, limits the amount of incompetence, so that both cannot be severe together. In longstanding mitral valve disease either stenotic or incompetent, dystrophic valvular calcification may occur. Stenosis of the mitral valve is most frequently due to rheumatic fever.

Mitral valve disease leads to an increase in the mean pressure in the left atrium. This is transmitted back to the pulmonary veins and when severe leads first to interstitial and then to alveolar oedema. If that disease is severe and longstanding parenchymal lung changes of haemosiderosis and the formation of ossific nodules may appear. Secondary pulmonary arterial hypertension may develop, leading to pulmonary valve incompetence, right ventricular dilatation and functional tricuspid incompetence. There may also be organic involvement by the rheumatic process of the tricuspid valve, leading either to stenosis or incompetence. (Raphael, 1986).

PLAIN RADIOGRAPHIC APPEARANCES

The cardinal radiological feature of rheumatic mitral valve disease is selective left atrial enlargement. The left atrial appendage is particularly affected and when this is seen to be enlarged it

always suggests a rheumatic aetiology for the mitral valve disease. The appearances vary from a simple straightening of the left heart border to a very large bulge in the characteristic site of the appendage immediately below the left main bronchus. (Raphael, 1986).

Puzzling appearances may occur with unusual enlargement of the left atrium. The features of enlargement of the left atrium vary from only one of the characteristic signs being visible on the plain radiograph to aneurysmal enlargement when the left atrium reaches to within an inch or so of the chest wall on one or both sides of the chest. (De Sanctis, et al., 1964). These very large atria are more commonly seen in longstanding mitral incompetence with atrial fibrillation. (Raphael, 1986).

The left atrium may calcify either in its wall or in clot lining the wall. (Gedgaudas, et al., 1968). The calcification is usually curvilinear, at least in part. In the frontal view it lies fairly high up in the cardiac shadow at a higher level than that seen in pericardial calcification. The lateral radiograph locates the calcification in the upper posterior aspect of the heart at the position of the left atrium and this may be confirmed by fluoroscopy.

Fluoroscopy is also the best method of detecting dystrophic calcification, which may occur in the mitral valve in long-standing rheumatic valve disease. (Lachman and Roberts, 1978). It locates the mitral valve lying between the left atrium and left ventricle near the postero-inferior aspect of the heart in the lateral view. Fluoroscopy also shows the characteristic movement of the calcified leaflets moving downwards in relation to the heart shadow in ventricular diastole and serves to distinguish them from the 'J' or 'C' shaped calcification which occurs in or below the mitral valve ring in elderly females. (Henby, 1964). This 'C'-shaped calcification is usually of no clinical significance - the gap in the 'C' is due to the absence of ventricular wall at the mitral/aortic valve confluence.

Calcification in the mitral valve itself is strongly suggestive of a rheumatic aetiology.

The right ventricle often appears enlarged in the lateral view in rheumatic mitral valve disease, presumably because it is pushed against the sternum by the enlarged left atrium. The right ventricle may be genuinely enlarged if there is pulmonary arterial hypertension or pulmonary valve incompetence. Tricuspid valve disease may be suspected if there is right atrial enlargement.

A rise in mean pressure in the left atrium is reflected back into the pulmonary veins and produces a graded series of changes in the lung, which may be seen on the plain radiograph.

(1) Upper lobe blood diversion produces distension of the upper lobe veins ("stag's antler" appearance). This is followed by constriction of the lower lobe veins.

(2) Interstitial oedema recognized by Kerley's septal costophrenic B lines and central A lines.

(3) Pulmonary alveolar oedema appearing as confluent pulmonary shadows of perihilar distribution.

(4) Pulmonary haemosiderosis, seen as fine punctate calcifications throughout the lungs.

(5) Pulmonary ossific nodules, calcified densities of up to 1 cm. in diameter, may occur as the result of long-standing pulmonary hypertension.

(6) Pulmonary arterial hypertension, recognized by enlargement of the main pulmonary artery and central pulmonary vessels with peripheral vessel pruning. (Raphael, 1986).

Symptomatic mitral stenosis, of such severity as to require surgical treatment may, however, be present with an entirely normal chest radiograph. After mitral valvotomy with amputation of the left atrial appendage, there is a concavity in the left heart border below the left bronchus, which invariably gives the heart a left ventricular configuration and makes genuine left ventricular enlargement difficult to assess.

The contribution of mitral stenosis and incompetence to the clinical picture cannot be determined by the plain radiograph. Selective left ventricular enlargement, which should be associated with mitral incompetence, cannot be recognized with certainty and could also be due to any associated aortic valve lesion. Only if left atrial enlargement is very gross can mitral incompetence be suspected. (Raphael, 1986).

NON-RHEUMATIC MITRAL VALVE DISEASE

This is almost always incompetence due to disorder of the subvalvar apparatus, though incompetence due to perforation of a leaflet by endocarditis may occur.

MITRAL VALVE PROLAPSE

Elongation of the chordae tendineae associated with mucoid degeneration of the leaflets allows prolapse of the mitral valve leaflets back into the left atrium in ventricular systole. The condition may occur in the course of Marfan's disease or more commonly may be seen alone. Prolapse may occur without mitral incompetence and produce a mid or late systolic click as the chordae tense and the leaflets balloon backwards. If coaptation of the leaflets is impaired by chordal elongation, mitral incompetence occurs but the leak does not begin until after the leaflets have prolapsed into the atrium. (Iskandrian, et al., 1978). (Popp and Winkle, 1976).

CHORDAL RUPTURE AND PAPILLARY MUSCLE DYSFUNCTION

Mitral regurgitation may result from rupture of groups of chordae tendineae allowing part of a leaflet to flail so that it does not coapt in systole but permits mitral regurgitation. The murmur is mid to late systolic in onset and the condition progressive in a jerky fashion, worsening as groups of chordae rupture and improving as the heart compensates.

Mitral incompetence can also result from ventricular cavity gross dilatation as occurs in dilated cardiomyopathy or ischaemic cardiac failure. Regurgitant flow in this situation is usually not gross.

Mitral regurgitation may also result from papillary muscle dysfunction. (Raphael, 1986).

PLAIN FILM RADIOGRAPH

The appearances depend on the duration of the mitral regurgitation and on any associated heart disease. Quite severe non-rheumatic mitral incompetence may be present with pulmonary oedema, but with

a virtually normal heart shadow. After an interval the heart is usually compensated with the development of left ventricular enlargement, which may be quite marked. Selective left atrial enlargement may be absent, slight or moderate and the left atrial appendage is usually not enlarged. Pulmonary vascular changes reflect the haemodynamic derangement and the effects of treatment. (Raphael, 1986).

CALCIFIED MITRAL RING

The calcification occurs in a layer of clot in the angle between the LV wall and the mitral valve cusps. The calcification is readily recognized on the chest radiograph as a C-shaped open ring. The gap in the ring is due to the absence of LV wall where the anterior mitral leaflet base is in contact with the posterior part of the aortic valve ring. It rarely occurs before the age of 70 years and is much commoner in women. It is of little clinical significance of itself but may be associated with left ventricular hypertrophy, either primary or secondary to hypertension or aortic stenosis. (Fulkerson, et al., 1979).

AORTIC VALVE DISEASE

AORTIC STENOSIS

CALCIFIC AORTIC STENOSIS:

This condition is generally thought to be due to the deposition of calcific masses on a congenitally deformed bicuspid valve. The condition occurs in patients over thirty years and with a male predominance. Unlike the mitral valve in mitral stenosis, where calcium deposits on an already stenosed valve, in calcific aortic stenosis it is the deposition of the masses of calcium on the cusps which obstruct flow by their very bulk as well as by stiffening the cusps; the aortic valve calcium is the stenosing agent. (Raphael, 1986).

PLAIN FILM RADIOGRAPHY:

Rounding of the cardiac apex may suggest sole left ventricular hypertrophy; however, there is usually some degree of cardiac enlargement, and this may be marked if there is any associated aortic incompetence. A localized prominence of the ascending aorta may indicate post-stenotic dilatation, but in older patients the whole thoracic aorta may appear widened. The lateral view may reveal aortic valve calcification, but this is best seen by fluoroscopy. The extent of the valve calcification bears only a rough relationship to the severity of the stenosis.

Clinically significant aortic stenosis may be present with a heart shadow which is virtually normal in the frontal view though the heart usually shows some evidence of enlargement of the left ventricle in the lateral view. There may be no visible abnormality in the aorta. (Proto and Speckman, 1979).

RHEUMATIC AORTIC STENOSIS:

Rheumatic aortic stenosis is due to inflammatory fusion of the commissures of the aortic valve cusps; it is often associated with some degree of aortic incompetence and almost invariably is associated with involvement of the mitral valve.

PLAIN FILM RADIOGRAPHY:

The appearance of rheumatic aortic stenosis is very commonly dominated by the presence of associated mitral valve disease, which produces selective left atrial enlargement and makes recognition of any left ventricular disorder very difficult. Post-stenotic dilatation of the aorta is rarely recognized in the rheumatic form of aortic stenosis, although the aorta may be dilated if there is associated aortic incompetence.

Gross calcification of the aortic valve is rare in rheumatic disease, but with good quality fluoroscopy flecks of calcium on the valve are relatively commonly seen, often with calcium in the mitral valve. The two calcifications may usually be distinguished by their position and inclination, the aortic valve calcification being above the mid-point of the heart and in a rather horizontal plane, the mitral valve calcification being lower and more posterior. Mitral valve calcification moves posteriorly in ventricular systole, whereas aortic calcification rises vertically. When valvar and ring calcifications are both gross it may be quite difficult to determine the exact situation. (Raphael, 1986).

AORTIC INCOMPETENCE:

This may be due to disease of the cusps of the aortic valve or to disease of the aortic walls. Cusp involvement may precipitate acute aortic incompetence when damaged by bacterial endocarditis or, very rarely, by trauma or as a result of acute eversion. Chronic aortic incompetence may be acquired as a result of congenital deformity or of rheumatic heart disease.

All forms of aortitis affecting the ascending aorta may involve dilatation of the valve ring and lead to aortic incompetence. Syphilis, ankylosing spondylitis, Marfan's disease and aortic dissection are amongst the commonest causes. (Raphael, 1986).

CHRONIC AORTIC INCOMPETENCE

PLAIN FILM RADIOGRAPHY:

Chronic aortic incompetence produces some of the most dramatic appearances with enlargement of the left ventricle which may be very gross indeed. The enlargement parallels the severity of the condition and progressive cardiac enlargement is one factor suggesting the need for surgical intervention. In this condition the post-operative reduction of heart size following valve surgery is often very dramatic. The whole thoracic aorta may be enlarged to a moderate degree. Valve calcification is not a marked feature, but flecks of calcium may be seen at fluoroscopy. When mitral valve disease is present as well, selective left atrial enlargement may dominate the appearances and obscure significant left ventricular enlargement. (Raphael, 1986).

ACUTE AORTIC INCOMPETENCE

PLAIN FILM RADIOGRAPHY:

Depending on the rapidity of onset the heart may be normal in size or show only minor degrees of predominantly left ventricular enlargement. If the patient is in left heart failure there will be upper lobe blood diversion or overt pulmonary oedema. Acute aortic incompetence is an important cause of pulmonary oedema in a patient with a normal sized heart. The aorta is usually unremarkable. (Raphael, 1986).

BICUSPID AORTIC VALVE:

Bicuspid aortic valve is a frequent congenital anomaly often associated with other congenital aortic lesions, especially coarctation. Only two aortic cusps are present, the larger usually being divided by a raphe.

The bicuspid aortic valve rarely causes symptoms before the third or fourth decade, when progressive and premature degenerative changes results in thickening, irregularity and calcification and causes progressive aortic valve stenosis. Plain film radiography may show signs of aortic valve stenosis as described previously. (Roberts, 1970).

PULMONARY VALVE DISEASE

The pulmonary valve is least commonly affected. Pulmonary regurgitation occurs most frequently as a sequela of endocarditis, pulmonary hypertension or operative correction of pulmonary stenosis. (Jones, et al., 1973).

Acquired pulmonary obstruction is unusual. Right ventricular hypertrophy and post-stenotic dilatation of the main pulmonary artery are to be expected if the obstruction is at the level of the pulmonary valve. Usually no enlargement of the pulmonary artery occurs with subpulmonic obstruction. (Jefferson and Rees, 1973).

A variety of conditions may cause acquired pulmonary obstruction or regurgitation. The causes are listed in tables 5 and 6.

PLAIN FILM RADIOGRAPHY:

The typical findings of acquired pulmonary regurgitation are enlargement of the right ventricle and pulmonary artery. (Smith, et al., 1979).

TABLE 5

CAUSES OF ACQUIRED PULMONARY REGURGITATION

Pulmonary hypertension

Surgical trauma

Infectious endocarditis

Rheumatic heart disease

Carcinoid syndrome

Large atrial shunts without pulmonary hypertension

Pulmonary artery syphilis

Methysergide therapy

Pulmonary arteritis and valvulitis secondary to aortic aneurysm

TABLE 6

CAUSES OF ACQUIRED PULMONARY STENOSIS

Rheumatic heart disease

Carcinoid syndrome

Vegetations of infectious endocarditis

Primary or secondary right ventricular tumours

Cyst or tumour of the pulmonary valve

Right ventricular embolus extending to the pulmonary artery

Extrinsic compression

Hypertrophic cardiomyopathy

Obstruction due to echinococcus cyst

Involvement of the pulmonary valve by far-advanced tuberculosis

Valvular atheroma

DISEASES OF HEART MUSCLE

DISEASES OF HEART MUSCLE

Cardiomyopathy is defined as a disease of heart muscle of unknown cause or association. When a cause or association is known the condition is so described, e.g. "thyrotoxic heart disease". (Raphael, 1986).

CONGESTIVE CARDIOMYOPATHY COCM

(dilated cardiomyopathy)

PLAIN RADIOGRAPHIC APPEARANCES:

The heart is usually enlarged and may be very large indeed. Evidence of enlargement may only be seen in the lateral view with the frontal view appearing normal. The configuration of the heart may be solely left ventricular, or all chambers may be enlarged, although there is usually left ventricular prominence.

Upper lobe blood diversion and a variable degree of pulmonary oedema are usually seen. The recognition of calcification in the region of the aortic valve on the lateral view may be the first indication that this is silent aortic valvar stenosis rather than congestive cardiomyopathy. (Raphael, 1986).

DIFFERENTIAL DIAGNOSIS OF SPECIFIC HEART MUSCLE DISEASE RESEMBLING DILATED CARDIOMYOPATHY

INFECTIONS:

(a) Viral myocarditis

(b) Diphtheria

(c) Chagas' disease (infection with trypanosoma cruzi. (Acquatella, et al., 1980).

COLLAGEN DISEASE:

Rheumatic fever

INFILTRATION OF THE HEART:

(a) Haemochromatosis

(b) Glycogen storage disease (Ress, et al., 1976)

(c) Sarcoidosis (Fleming and Bailey, 1981)

(d) Amyloid heart disease (Chew, et al., 1975)

(e) Hurler's syndrome (Krovetz, et al., 1965)

METABOLIC DISORDERS:

(a) Thyrotoxicosis

(b) Myxoedema

(c) Acromegaly

(d) Beriberi

(e) Cushing's syndrome

DRUGS AND POISONS:

(a) Alcoholic heart disease

(b) Beta-blocking drugs

(c) Cytotoxic drugs

MISCELLANEOUS CONDITIONS:

(a) Postpartum cardiomyopathy

(b) Neuromuscular disorders

(c) Familial congestive cardiomyopathy

(d) Endocardial fibroelastosis

(e) Hypertensive cardiomyopathy

(f) Anaemia

(Raphael, 1986)

HYPERTROPHIC CARDIOMYOPATHY (HOCM)

This condition is characterized by an excessive and inappropriate hypertrophy of the myocardium with no known responsible causative mechanical factors. The hypertrophy always involves the left ventricle and characteristically the upper interventricular septum is most affected, giving rise to one of the synonyms of the condition asymmetric septal hypertrophy. However, the whole of the left ventricular myocardium may be uniformly hypertrophied. The right ventricle may also be involved. The condition is often familial. The cardiac muscle contracts well and systolic left ventricular function is normal. However, the increased bulk of LV muscle impedes left ventricular filling which has to take place at a higher pressure. (Shah, 1979).

PLAIN RADIOGRAPHIC APPEARANCES

Appearances can vary from a heart entirely normal in size and shape to varying degrees of left ventricular hypertrophy; from simple rounding of the apex to gross apparent left ventricular enlargement by very marked hypertrophy. The appearances may be those of pure left ventricular involvement or the heart may show (very rarely) marked left atrial enlargement or even a globular appearance when all chambers are enlarged and appearances may be indistinguishable from COCM. The lungs may be normal or show upper lobe blood diversion or pulmonary oedema. Aortic valve calcification will not be seen, although mitral valve and endocardial calcification may be rarely seen in HOCM, particularly following bacterial endocarditis. The heart shadow may remain unchanged for years, but increased size occurs with clinical deterioration often in association with the onset of atrial fibrillation. The enlargement is almost always due to dilatation of chambers other than the left ventricular cavity, which only rarely undergoes a terminal dilated phase. (Chapman, et al., 1978).

Patients with this suggested diagnosis should undergo catheter investigation only if echocardiography is equivocal.

RIGHT VENTRICULAR INVOLVEMENT

This leads to a clinical finding resembling pulmonary infundibular stenosis. The chest radiograph does not show post-stenotic pulmonary artery dilatation and there is a rather non-specific heart shape. Right ventricular angiography shows infundibular narrowing, often associated with a large septum bulging into the right ventricle.

Left ventriculography will reveal the bilateral involvement and point to the correct diagnosis. (Raphael, 1986).

RESTRICTIVE CARDIOMYOPATHY

ENDOMYOCARDIAL FIBROSIS (EMF)

This condition is characterized by the deposition of a layer of fibrous tissue on the ventricular endocardium, beginning at the apex and spreading proximally to encroach on the atrioventricular valve. Either ventricle or both may be affected. Thrombus may deposit on the endocardium and become organized, resembling a ventricular tumour.

The functional derangement is that of a restrictive type of cardiomyopathy. The fibrosis does not initially affect contractile function severely and the ventricles empty reasonably well, but the fibrosis prevents them from expanding fully in diastole. Valve involvement leads to atrioventricular valve incompetence. (Cockshott, 1969).

PLAIN RADIOGRAPHIC APPEARANCES:

In right heart involvement the heart is enlarged with a right heart or globular configuration. The right atrium may be very large. There may be a pericardial effusion. The lungs may be clear if there is no left heart involvement. Pleural effusions may be seen. Some cases may have a normal heart or some left ventricular and left atrial enlargement plus pulmonary congestion. Very rarely curvilinear calcification or endocardial calcification may be seen.

Angiography is usually characteristic. The fibrosis effaces and amputates the apices of the involved ventricles and smooths their cavitary outline. Atrioventricular valve incompetence may be seen into an enlarged atrium. Tumour-like masses of organized thrombus may cause confusion. (Cockshott, 1969).

TUMOURS OF THE HEART

Involvement of the heart by secondary malignant disease, usually from breast or bronchus, is relatively common, the majority of cases being discovered only at necropsy. The diagnosis is usually obvious when clinical evidence of cardiac involvement, arrhythmias or (haemorrhagic) pericardial effusion develop in a patient with a known primary neoplasm. Intrathoracic extracardiac tumours, benign or malignant, may produce cardiac symptoms and signs by compression of the heart and great vessels and mimic obstructive lesions of these vessels. Cardiac tumours, ventricular and aortic aneurysms may resemble each other radiologically.
(Abrams, et al., 1971).

PRIMARY CARDIAC TUMOURS

Primary tumours of the heart are rare and the majority are benign, the malignant tumours being sarcomas.

LEFT ATRIAL MYXOMA

This is the commonest single variety of primary intracardiac tumour. It has three types of presentation:-

(a) OBSTRUCTIVE SYMPTOMS

The patient presents with shortness of breath and signs of mitral valve disease. The physical signs suggest mitral stenosis, but often with some regurgitation. The signs may vary from day to day and with the position of the patient. The symptoms may appear excessive in relation to the apparent severity of the mitral valve disease. All these features, combined with sinus rhythm, raise the possibility of a myxoma. (O'Neill, et al., 1979).

(b) EMBOLIC PHENOMENON

Peripheral embolism is a commonly presenting symptom. The emboli may be minor, appearing as splinter haemorrhages in the fingers or major, leading to limb or cerebral ischaemia or involving other organs.

(c) SYSTEMIC MANIFESTATIONS

Fever, anaemia, a raised ESR and sometimes finger clubbing may suggest the possibility of infective endocarditis. (Read, 1980).

PLAIN FILM RADIOGRAPHY:

The heart is commonly enlarged. There is often evidence of selective left atrial enlargement, although rarely is there a large appendage, which would suggest rheumatic heart disease. Upper lobe blood diversion, pulmonary oedema, or even pulmonary arterial hypertension may all be seen. The appearances of the chest film may be normal. The rare calcified myxoma may be seen at fluoroscopy, moving forwards through the mitral valve in ventricular diastole. (Raphael, 1986).

OTHER CARDIAC TUMOURS

(a) Rhabdomyomas

(b) Fibroma

(c) Sarcomas

(d) Hydatid disease

PLAIN FILM RADIOGRAPHY

Plain film radiography usually shows the heart shadow to be enlarged, often markedly so. Selective chamber enlargement occurs only if obstruction to emptying is present. The appearances may be those of a pericardial effusion or, more rarely, a localized tumour mass may be seen. (Raphael, 1986).

DISEASES OF THE PERICARDIUM

DISEASES OF THE PERICARDIUM

CONGENITAL ANOMALIES OF THE PERICARDIUM

(1) Absence

(2) Pericardial cyst

(3) Pericardial diverticulum

PERICARDIAL NEOPLASM

(1) Malignant tumours (mesothelioma)

(2) Metastatic tumours (usually from lung or breast carcinoma, melanoma or lymphoma).

(Steiner and Rao, 1986).

PERICARDITIS AND PERICARDIAL EFFUSION

Inflammation of the pericardium can result from many diseases and may present with serous or exudative effusion, fibrinous production or cellular proliferation with adhesions or constriction, either alone or in varying combinations. (Ellis and King, 1973).

PERICARDIAL EFFUSION

Pericardial effusion is the major manifestation of pericarditis. The fluid may be serous with a clear transudate or exudate, varying in nature with the underlying cause. Transudate hydropericardium will occur, for example, in congestive heart failure, uraemia, myxoedema and collagen diseases. Haemopericardium may be due to trauma, aortic or cardiac rupture or metastatic carcinoma. (Soulen, et al., 1968). Chylopericardium is rare, resulting from injury or obstruction to the thoracic duct. Pericardial effusion may resolve completely, or considerable pericardial thickening may remain, particularly in haemopericardium.

PLAIN FILM DIAGNOSIS

An abrupt symmetrical increase in heart size, without specific chamber enlargement, not accounted for by intrinsic cardiac disease, should suggest the diagnosis of pericardial effusion. Encroachment on the retrosternal space, effacement of the normal cardiac borders, a flask or water bottle cardiac configuration and bilateral hilar overlay (the 'overlay sign') by the heart are characteristic findings in pericardial effusion.

If there is decreased pulmonary vasculature in spite of cardiac enlargement, or if the superior vena cava and azygos veins are dilated, tamponade should be suspected. Since acute tamponade may occur with small effusions, the enlargement may be difficult to detect on plain films. Careful comparison with previous films will be helpful in detecting subtle changes in cardiac contour. (Steiner and Rao, 1986).

A pericardial stripe wider than 2mm. is diagnostic of pericardial disease. Since there is abundant fat between the pericardium and the pleura at the cardiac apex, a positive epicardial fat pad may also be seen in the frontal projection, usually as an elliptical stripe paralleling the lower left heart border. This line may migrate towards or away from the left heart border with changes in the amount of pericardial fluid. A positive epicardial fat pad sign is present in pericardial effusion in up to 65% of patients. (Carsky, et al., 1980).

Fluoroscopy has limited value in the diagnosis of pericardial effusion.

PERICARDITIS

Pericarditis may be fibrinous, purulent, adherent or constrictive, with or without calcification. Acute pericarditis with pericardial effusion may either be local, such as viral pleuritis, or part of a more generalized disease process such as lupus erythematosus. Table 7. (Hudson, 1978).

TABLE 7

CAUSES OF CONSTRICTIVE PERICARDITIS

(1) Idiopathic

(2) Infection

- (a) Bacterial including actinomycosis and nocardia
- (b) Myobacterial - particularly tuberculosis
- (c) Fungal - particularly histoplasmosis
- (d) Viral - especially Coxsackie B
- (e) Parasitic - especially amoebic pericarditis

(3) Connective tissue disorders

- (a) Rheumatoid arthritis - most common collagen disease
- (b) Lupus erythematosus - less common
- (c) Polyarteritis
- (d) Drug related disease

(4) Metabolic - Uraemia

(5) Trauma

- (a) Haemopericardium
- (b) Surgery

(6) Radiation

(7) Neoplastic

- (a) Mesothelioma
- (b) Metastatic carcinoma
- (c) Lymphoma

(Steiner and Rao, 1986)

CONSTRICTIVE PERICARDITIS

When fibrotic, calcified or involved with diffuse neoplasm, the pericardium may become rigid, limiting diastolic expansion and filling of both ventricles. Obstruction to flow from the inferior and superior vena cava, the pulmonary veins, the main pulmonary artery and the atrioventricular valves also occurs. In most cases constriction originates as pericarditis with effusion. As the effusion resolves the visceral and parietal pericardial layers fuse causing scarring with or without calcification. (Hirschmann, 1978).

AETIOLOGY OF PERICARDITIS

(1) Acute non-specific, idiopathic or iatrogenic

(a) Post-myocardial infarction syndrome
Post-pericardotomy syndrome (Dressler's syndrome)

(b) Asbestosis

(c) Radiation induced pericarditis

(d) Drugs

(2) Connective tissue disease including rheumatoid disease, rheumatic fever, disseminated lupus erythematosus, scleroderma and Takayasu arteritis

(3) Penetrating or non-penetrating trauma

(4) Cardiovascular

(a) Dissecting aortic aneurysm

(b) Acute myocardial infarction

(c) Congestive heart failure

(5) Infection

(a) Tuberculosis

(b) Fungal infections, particularly histoplasmosis

(c) Pyogenic infections; blastomycosis

(d) Viral infection; Coxsackie B; ECHO; influenza

(e) Amoebiasis

(6) Neoplasm

(a) Primary malignant

(b) Metastatic including lymphoma; leukaemia

(7) Metabolic

(a) Myxoedema

(b) Uraemia

(Steiner and Rao, 1986).

CHEST RADIOGRAPHY IN CONSTRICTIVE PERICARDITIS

CALCIFICATION

Calcification is seen in about one third of cases of constrictive pericarditis. (Daves, 1981). This finding is indicative of chronic pericarditis but may not be associated with significant constriction. Calcification is characterized on the plain films as a shell-like line or plaque, more common in inferior and posterior pericardial surfaces and the atrioventricular groove. The calcification is often thick and irregular with intermittent fine line distribution. Pericardial calcification can be distinguished from other forms of cardiac calcification by its distribution not conforming to chamber or valve configuration. The lack of a tramway sign also differentiates pericardial calcification from coronary artery calcification.

Pericardial calcification is usually best seen on lateral chest films. Because it is often caudal in location it may be missed on an under-penetrated frontal film, especially in a patient with ascites or pleural effusion and may be then seen on computed tomography or fluoroscopy. The heart size is generally normal or smaller than normal with evidence of elevated pulmonary venous pressure implying congestive failure. When the heart is persistently enlarged pericardial effusion, in addition to pericardial thickening, may be present. A common feature of constrictive pericarditis is flattening of the right atrial border in spite of enlargement elsewhere of the cardiac silhouette. (Steiner and Rao, 1986).

COMPUTED TOMOGRAPHY (CT)

Computed tomography is very helpful in demonstrating the extent and distribution of pericardial calcifications. It will help to distinguish pericardial calcifications and pericardial thickening from cardiomyopathy. CT will document dilatation of the cava and azygos venous system and the presence of pleural effusion and ascites. (Moncada, et al., 1981).

ISCHAEMIC HEART DISEASE

There are two clinical syndromes associated with ischaemic heart disease. When the ischaemia is transient and reversible the term 'angina pectoris' is used. When ischaemia is irreversible and leads to necrosis then 'myocardial infarction' develops, healing by scar formation if the patient survives. (Raphael and Silverman, 1986).

ANGINA PECTORIS

Angina pectoris itself has no plain radiographic manifestations, though evidence of coronary artery disease or previous myocardial infarction may be seen. (Donaldson and Ell, 1981). The definitive investigation is coronary arteriography. Thalium 201 isotope scanning is another technique which is used to investigate angina. However, it has been proven to be less specific than was at first hoped. (Maseri, et al., 1975). (Raphael and Silverman, 1986).

MYOCARDIAL INFARCTION

Myocardial infarction is a process of ischaemic necrosis of cardiac muscle which heals by fibrotic scarring.

PLAIN FILM APPEARANCES

HEART SIZE

Plain film findings are usually sparse. If there has been a previous significant myocardial infarction the heart shadow may be bulky or even overtly enlarged. The effects of the current infarct in leading to cardiac dilatation will not become evident for several weeks. A large heart carries a bad prognosis. Calcification in coronary arteries may be seen as moving flecks at image amplification fluoroscopy, or rarely as tramline calcification in the position of the coronary arteries in appropriately exposed frontal and lateral films. Myocardial calcification following infarction, other than in left ventricular aneurysm, is very rare. (Raphael and Silverman, 1986).

LUNG CHANGES

The haemodynamic disturbances will be reflected in the lungs. Upper lobe blood diversion and interstitial or overt alveolar oedema suggest extensive infarction. If they persist they are prognostically bad. Pulmonary oedema, other than in cardiogenic shock, usually clears rapidly. It may be clinically silent but should always be treated early by diuretics if demonstrated radiographically. (Raphael and Silverman, 1986).

BASAL LUNG SHADOWS

Within two or three days of the myocardial infarction ill-defined basal lung shadows may be seen, more commonly on the right, evolving into broad band shadows resembling plate atelectasis, before clearing. They do not represent pulmonary infarction but are probably caused by some lung collapse due to splinting of the diaphragm by pain. (Tudor, et al., 1973).

DRESSLER'S SYNDROME

At about ten to thirty days after infarction or surgery some patients develop chest pain associated with a high ESR - Dressler's syndrome. This is characterized by the triad of pleuritis (small pleural effusion), pneumonitis (ill-defined basal lung shadows) and pericarditis which may progress to a substantial pericardial effusion and even on rare occasions to tamponade. The syndrome may remit but may then relapse over a period of weeks or months. It should not be confused with extension of the myocardial infarction or with pulmonary embolism with infarction. It usually responds dramatically to aspirin or steroids, which is a useful distinguishing feature. (Dressler, 1959).

COMPLICATIONS OF MYOCARDIAL INFARCTION

RUPTURE OF THE HEART

This is the commonest complication. The myocardium ruptures and pericardial tamponade leads rapidly to death. There is little opportunity for radiology. Very rarely the rupture is subacute leading either to pericardial effusion with tamponade, or the rupture may be walled off within the pericardium producing a false or pseudo-aneurysm. (Raphael and Silverman, 1986).

LEFT VENTRICULAR ANEURYSM

This is the commonest mechanical complication encountered in routine practice. The definition of aneurysm varies according to different groups of workers giving different incidences of symptoms and prognosis. (Donaldson, et al., 1976). The pathological definition is of a large, thin walled, fibrous sac bulging not only the lumen of the left ventricular cavity, but also from the external surface of the heart and usually clearly demarcated from normal myocardium. Such a large sac is usually easily resectable.

Left ventricular aneurysm develops in a small percentage of patients with a large antero-septal myocardial infarction. The clinical findings are those of left ventricular failure, angina, persisting arrhythmias or, rarely, peripheral embolism. Plain radiographic findings vary from an apparently normal heart to an apparent simple left ventricular enlargement or an obvious localized bulge from the left ventricle. Curvilinear calcification in the wall of the aneurysm occurs only after several years. There may be evidence of cardiac failure in the lungs. After resection of the aneurysm the heart may revert to near normal size and shape and the lungs may clear of oedema. (Raphael and Silverman, 1986).

THE DIFFERENTIAL DIAGNOSIS OF A LEFT VENTRICULAR ANEURYSM INCLUDES THE FOLLOWING:

(a) Ischaemic cardiomyopathy.

(b) Congestive cardiomyopathy.

(c) Silent aortic stenosis.

The calcified aortic valve may be detected on the plain film.

ESSENTIAL HYPERTENSION

The heart shadow in minor degrees of hypertension may appear relatively normal or there may be minimal enlargement of the left ventricle and of the aorta. In the majority of patients, however, left ventricular hypertrophy is the first reaction and this is soon followed by left ventricular enlargement. The aorta too dilates and the degree of this depends partly on the level of systemic pressure.

RADIOGRAPHIC APPEARANCE

Left ventricular enlargement will soon be noticed and if there is evidence of left ventricular failure, left atrial enlargement due to functional mitral incompetence will also be seen. With adequate management and lowering of the blood pressure the radiological changes of cardiac enlargement soon regress and the heart can return to a normal size. When hypertension is not controlled, left ventricular enlargement progresses steadily.

The ascending aorta, aortic arch and descending thoracic aorta dilate and elongate and become more and more prominent. They eventually form part of the right and left mediastinal border projecting beyond the normal confines and bulging slightly into the right and left lung field. This aortic dilatation and elongation is sometimes referred to as "aortic unfolding". The degree of aortic dilatation largely depends on the severity of co-existing atherosclerosis. The aortic impression on a barium swallow becomes much more pronounced and widened. As the aorta further dilates it tends to become tortuous, this being clearly demonstrated on the oblique and lateral films. The barium filled oesophagus may also take a rather tortuous course, tending to follow the thoracic aorta.

Long-standing and severe hypertension may lead to left ventricular failure. (Steiner, 1980).

DISEASES OF THE THORACIC AORTA

NORMAL ANATOMY

The thoracic aorta is shaped like an inverted 'J' and is composed of four parts:-

the aortic bulb or root

the ascending aorta

the aortic arch

the descending aorta

The aortic bulb extends from the aortic valve to the top of the sinuses of Valsalva and forms an angle of 15 to 30° with the longitudinal axis of the body. It lies at the level of the 8th and 9th thoracic vertebrae. (Steiner, et al., 1986).

The aortic bulb lies entirely within the pericardial cavity as does the proximal portion of the ascending aorta, and these aortic structures are not usually seen on plain radiographs. However, in about half of the patients part of the intra-pericardial portion of the aorta may be visible because of pericardial fat. (Abrams, 1983).

The ascending aorta is approximately 2.5 to 3.5 cm. in diameter and extends cephalad and to the right from the sinuses of Valsalva towards the innominate artery, a distance of 4 to 5 cms.

The ascending aorta joins the aortic arch at the plane joining the disc between the fourth and fifth dorsal vertebrae and the angle of Louis. The arch begins in the right anterior mediastinum and courses to the left upwards and posteriorly in front of the trachea and the oesophagus.

The descending aorta descends, moving slightly to the right within the posterior mediastinum, partially overlying the anterior aspect of the spine. It reaches and penetrates the diaphragm at the level of the 11th or 12th thoracic vertebra. (Steiner, et al., 1986).

Although the aorta can be clearly imaged throughout its length using computed tomography (Moncada, et al., 1983), magnetic resonance imaging or contrast aortography, with plain films the normal thoracic aorta is appreciated as a separate structure only where it is border-forming with the lung or where it is calcified. The aortic valve, the ascending aorta and the proximal arch are not visualized as separate structures but blend in with other mediastinal structures. The descending aorta is seen lateral to the left paravertebral line. In the 15^{o} left anterior oblique projection the width of the upper part of the ascending aorta and the arch is seen in profile. The transverse section of the aortic arch is outlined by lung on one side and by the barium-filled oesophagus on the other. (Cooley and Schriber, 1980).

CONGENITAL ANOMALIES OF THE AORTIC ARCH

The three most common anomalies of the aortic arch and its branches are:-

(1) Left sided aortic arch with aberrant right subclavian artery.

(2) Mirror image right aortic arch.

(3) Right aortic arch with aberrant left subclavian artery. (Steiner, et al., 1986).

COARCTATION OF THE AORTA

Coarctation consists of a local stenosis or obstruction of the aorta in the area of the aortic isthmus. It usually occurs at or immediately distal to the left subclavian artery and proximal or distal to the ductus arteriosus. (Slater and Sanctis, 1980). The more frequent type of coarctation is the ductal or post-ductal form.

The less common preductal coarctation is usually discovered in infancy and may present as complete atresia of the thoracic aorta distal to the innominate or left subclavian artery, or may occur as a zone of tubular or localized hypoplasia or stenosis proximal

to the left subclavian artery. A patent ductus arteriosis is usually present. Subaortic stenosis, ventricular septal defect or transposition of the great arteries are common associations. (Steiner, et al., 1986).

Associated anomalies occur in about 75% of patients including:-

ascending aortic aneurysm
bicuspid aortic valve (which occurs in perhaps 85% of cases)
aortic dissection
patent ductus arteriosis
aortic regurgitation
congenital haemangioma
aneurysms
(Scheeweiss, et al., 1982)
ventricular septal defect (which usually occurs in the preductal form of coarctation

There is an association between coarctation, aortic stenosis and gonadal dysgenesis - Turner's syndrome.

The systolic blood pressure in the arm is at least 20 mm Hg higher than in the legs in a significant coarctation.

Collateral circulation develops with age, so that by 10 years most patients with haemodynamically significant post-ductal coarctation will show evidence of rib notching due to the pulsation of dilated and tortuous intercostal arteries conveying blood towards the aorta. Enlarged internal mammary, lateral thoracic, epigastric and anterior spinal artery collaterals also develop. If the coarctation is proximal to an aberrant right subclavian artery, rib notching will only be present on the left side. If the left subclavian artery is atretic or below the coarctation there will be unilateral right sided rib notching. If the coarctation is in the distal thoracic aorta or upper abdominal aorta, only the lower ribs will exhibit notching. (Steiner, et al., 1986). The different causes of rib notching are listed in Table (9).

TABLE 9

CAUSES OF RIB NOTCHING

ARTERIAL CAUSES

(1) Aortic Obstruction
- (a) Coarctation
- (b) Thrombosis of the abdominal aorta
- (c) Atherosclerosis
- (d) Subclavian artery obstruction including Blalock-Taussig procedures (unilateral)
- (e) Takayasu's syndrome

(2) Severe Pulmonary Oligaemia in Cyanotic Heart Disease
- (a) Tetralogy of Fallot
- (b) Pseudotruncus arteriosis
- (c) Extreme pulmonary stenosis
- (d) Absent pulmonary artery
- (e) Severe emphysema

VENOUS CAUSES

(1) SVS obstruction

(2) IVC obstruction

ARTERIOVENOUS CAUSES

(1) Pulmonary arteriovenous fistula

(2) Intercostal arteriovenous fistula

(3) Intercostal pulmonary artery fistula

NEUROGENIC CAUSES

(1) Neurofibromatosis

OSSEOUS CAUSES

(1) Hyperparathyroidism

(2) Extreme haematopoiesis, e.g. Cooley's anaemia

(3) Idiopathic

(4) Normal

(Steiner, et al., 1986)

RADIOLOGY

Radiological signs of obstructive coarctation in adults are often characteristic and include a high or apparently double aortic knob with an indentation on the left border below the knob in the area of obstruction. A reverse '3' pattern of a barium filled oesophagus due to prestenotic dilatation, the coarctation concavity and post-stenotic aortic dilatation,is highly suggestive of the diagnosis.

Bilateral rib notching from the third to the eighth rib is usually visible after the age of ten years with a haemodynamically significant coarctation.

The heart is normal in size although there is usually left ventricular prominence. Thoracic aortography conclusively establishes the diagnosis and demonstrates the location of the stenosis, the degree of narrowing and the length of involvement. (Steiner, et al., 1986).

THORACIC AORTIC ANEURYSM

An aneurysm is a localized dilatation of a blood vessel. True aneurysms are due to weakening of the vascular wall caused by degeneration of the medial elastic fibres and contain all three vessel layers. They are usually atherosclerotic in origin but may be due to syphilis and other infections or may be congenital. (Steiner, et al., 1986).

A false aneurysm is due to perforation of the aortic wall limited externally by the adventitia, perivascular connective tissue planes or by haematoma. False aneurysms are usually traumatic but may also be due to infection or may follow rupture of an atherosclerotic aneurysm. (Randall and Jarmolowski, 1983).

About 25% of all atherosclerotic aneurysms occur in the thoracic aorta and its branches, most commonly in the arch and descending aorta. Seventy per cent of thoracic aorta aneurysms are of atherosclerotic origin. Fifteen to twenty five per cent are of traumatic origin, less than five per cent are luetic and a few are mycotic. (Lang, 1982).

RADIOLOGY

Widening of the mediastinum and specifically of the aortic arch is the most common radiological abnormality of aneurysm.

Ascending aortic aneurysms will cause a loss of the incisura between the right atrial border and the ascending aorta. If there is associated aortic regurgitation left ventricular enlargement will occur. Large aneurysms of the upper ascending aorta or transverse arch may displace the trachea and oesophagus posteriorly and to the left.

Aneurysms often calcify, but thin rim-like calcification localized to the ascending aorta should suggest the diagnosis of lues, although atherosclerotic aneurysms should also be considered. Sternal erosion may occur in ascending aortic aneurysms. If the aneurysm originates in the aortic arch or descending aorta, displacement of the trachea and oesophagus occurs, most often anteriorly and to the right. If the outline of the aortic knob is blurred the possibility of dissection should be suggested as most aneurysms have sharp and distinct borders. Erosion of vertebral bodies or posterior rib cage may occur secondary to a pulsatile aneurysm of the descending aorta. (Steiner, et al., 1986).

Retrograde aortography and digital intercavenous aortography are very useful in confirming the diagnosis of aortic aneurysm and identifying associated conditions such as coarctation, other aneurysms and aortic valve insufficiency. (Machida and Tasak, 1980). (Steiner, et al., 1986).

AORTIC DISSECTION

Spontaneous acute aortic dissection is a catastrophic condition found in about one of 380 adult autopsies. (Slater and Sanctis,1980).

It is thought to develop because of a defect in structure or degeneration of the elastic and smooth muscle fibres of the aortic media. Reduced blood flow in the vasa-vasorum due to atherosclerosis resulting in ischaemia and necrosis of the aortic wall

may be a factor. Degeneration of elastic fibres and the development of fluid-filled cysts in the media are similar to that found in cystic medial necrosis of Erdheim.

Degeneration and haematoma of the media leads to a tear in the aortic intima, allowing blood under arterial pressure to dissect the diseased media and separate the intima from the adventitia for varying distances, both distal and proximal to the tear. It is unclear whether the haematoma precedes the tear or whether the tear leads to the development of haematoma. (Steiner, et al., 1986).

RADIOLOGY

Although mediastinal widening and enlargement of the aortic contour are the most common findings in 70% of patients, it may be difficult to appreciate this, particularly when only a portable film is available. Prominence of the ascending aorta and enlargement and haziness of the aortic knob, which occurs when reactive pleurisy or bleeding into the aortic wall has taken place, are common findings. (Steiner, et al., 1986). Tracheal and oesophageal displacement and a shift in the position of a calcified rim by greater than 6 mm. from the aortic border are valuable signs of dissection. (Cooley and Schriber, 1980).

The cardiac silhouette may enlarge due to haemopericardium, hypertension or aortic regurgitation. Pleural effusion, extrapleural apical and paramediastinal widening are other common findings. Although plain films will usually show some abnormality, it must be appreciated that a dissection can exist in a patient whose chest film appears normal. (Kaufman and White, 1980).

Aortography is the single most useful study in the diagnosis of aortic dissection. (Steiner, et al., 1986).

MYCOTIC ANEURYSMS

Mycotic or infected aneurysms are caused by invasion of the arterial wall by bacteria or by other non-luetic infective agents. (Abrams, 1983). They account for only 2.5% of all thoracic aneurysms but are important to recognize because of their rapid development and poor outcome. (Cooley and Schriber, 1980).

RADIOLOGICAL FINDINGS

Mycotic thoracic aorta aneurysms present as a rapidly expanding mediastinal mass. They usually occur in the ascending aorta or the sinus of Valsalva. The diagnosis is established by retrograde aortography. (Lang, 1982). (Wong, et al., 1981).

TRAUMATIC ANEURYSM

Aortic injury due to blunt trauma may result in aortic rupture. Although blast injuries and other non-penetrating injuries may cause rupture, the most common cause is motor vehicle (steering wheel) accidents. (Lang, 1982).

Since the clinical findings of aortic rupture are often confusing, subtle or absent, the radiological findings are essential to determine which patients should proceed to aortography or computed tomography. (Abrams, 1983).

The principle plain film findings include (in descending order of importance):-

mediastinal widening

tracheal displacement to the right

depressed left main stem bronchus

haemothorax and fracture of the clavicle or the first and second ribs

Other findings may include:-

left sided extrapleural apical cap with or without bleeding into the left or right paraspinal space

(Peters and Gamsu, 1980).

Aortography should be carried out to establish the diagnosis. (Abrams, 1983).

PLAIN CHEST RADIOGRAPHY IN CONGENITAL HEART DISEASE

There are many limitations of the plain chest x-ray in the assessment of congenital heart disease, especially in the neonates and infants. In neonates it may be impossible to assess accurately the cardiac size and shape, the pulmonary vasculature and the size of the aortic arch. A good quality radiograph is essential to assess the possibility of congenital heart disorder.

There are important features which should be evaluated in patients with heart disease:-

(1) The pulmonary vasculature

(2) The size of the heart

(3) The shape of the heart

(4) The size and position of the main pulmonary arteries

(5) The size and position of the ascending aorta and its arch

(6) Associated features such as skeletal changes

(Grainger, 1986).

The details of the above-mentioned features are beyond the scope of this thesis. However a summary of some of the important features will be presented.

THE PULMONARY VASCULATURE

Abnormalities of the pulmonary arteries, veins or the bronchial arteries may be encountered in congenital heart disease. This will include:-

(a) Pulmonary Oligaemia

The radiographic features have been discussed previously. They are impossible to assess if the chest radiograph is over-exposed. In pulmonary artery stenosis there is no obvious oligaemic change in the lungs except when the stenosis is very severe or associated with right to left shunt.

(b) Pulmonary Plethora

(c) Pulmonary Arterial Hypertension

The radiographic features have been discussed previously.

(d) Abnormalities of the Bronchial Circulation

Enhancement of the bronchial circulation may occur in association with severe obstruction of the pulmonary arterial blood supply in conjunction with right to left shunt. Pulmonary oligaemia without right to left shunt does not cause a radiologically recognisable increase in the bronchial arterial flow. (Grainger, 1986).

Radiographic Features of Enhanced Bronchial Circulation

Recognized in the central portion of the lung fields. Reticulonodular pattern with many rounded opacities are the typical features in addition to the absence of the normal vascular markings of the lungs.

Size of the Heart

Cardiac enlargement may be seen in many of the congenital heart disorders such as:-

large left to right shunt

fibroelastosis

severe valvular obstruction

The Shape of the Heart

Specific configuration of the heart and mediastinum may occur in some of the congenital heart diseases. These will include:-

(a) Egg-shaped configuration may be seen in uncorrected transposition of the great arteries.

(b) Sitting duck appearance may be seen in persistent truncus arteriosis.

(c) Boot-shaped heart may be seen in tetralogy of Fallot.

(d) Figure of eight or cottage loaf of bread is seen in supra-cardiac total anomalous pulmonary venous drainage.

Never exclude the diagnosis of any of these conditions because the shape of the heart is not characteristic.

Size and Position of the Main Pulmonary Arteries

(a) The pulmonary arteries may be high in position in certain types of persistent truncus arteriosis.

(b) Post-stenotic dilatation of the main pulmonary and left pulmonary artery is seen in pulmonary valve stenosis.

(c) Congenital absence or hypoplasia of the right or left pulmonary artery may occur and produce the radiographic features of small hilum and small lung volume.

Position, Size and Shape of the Ascending Aorta and its Arch

Right sided aortic arch may be seen in the following congenital heart diseases:-

(1) Persistent truncus arteriosis

(2) Tetralogy of Fallot

(3) Tricuspid Atresia

(4) Transposition of the great vessels

(A) Right sided aortic arch may be a normal variant and not associated with congenital heart disease.

(B) Dilated ascending aorta extending high in the mediastinum producing the appearance of a high prominent aortic arch is seen in persistent truncus arteriosis.

(C) A sharp (forward) kink may be seen along the aortic arch in cases of aortic coarctation.

ASSOCIATED FEATURES

Rib notching is an important abnormality which is seen in association with coarctation of the aorta. (Grainger, 1986).

Echocardiography can be very useful in the assessment of many congenital heart diseases. However the details of echocardiographic findings are beyond the scope of this study. (Grainger, 1986).

DIFFICULTIES, PITFALLS AND THE LIMITATIONS OF THE PLAIN RADIOGRAPHY IN THE ASSESSMENT OF HEART DISEASE

NORMAL VARIATIONS SIMULATING DISEASE

(1) Normal variability of heart size with changes in cardiac cycle in diastole and systole. (Keats, 1975).

(2) Visualization of the right border of the left atrium in a normal person. This should not be taken as evidence of left atrial enlargement in itself unless correlative evidence is present in the other projections. (Burko and Gyepes, 1965).

(3) Subpericardial fat which could be mistaken for pneumopericardium, seen in frontal and lateral projections.

(4) Lucent lung in the interval between the aorta and adjacent pulmonary vessels on the left, or juxtaposition of pulmonary arteries on the right could also be mistaken for pneumopericardium.

(5) A line may be seen through the right side of the heart which indicates the medial edge of the right lung. This should not be taken as a double contour due to left atrial enlargement. (Whalen, et al., 1973).

(6) A large thymus could simulate cardiomegaly in the frontal film, but in infants the lateral projection shows the heart to appear of normal size. The enlargement of the thymus may be unilateral or bilateral. The left lobe of the thymus could simulate enlargement of the left atrial appendage or produce an appearance suggestive of dextrocardia. (Keats, 1975).

(7) Normal variation in the size of the pulmonary artery in children and young adults. The pulmonary artery is extremely variable in size and its prominence in youth is a common finding. It should not be a source of concern in itself. (Keats, 1975).

(8) A discrete bulge may be seen on the posterior wall of the left ventricle. This could be mistaken for myocardial aneurysm. It represents a transient phase of contraction of the left ventricle late in systole and is a normal phenomenon. (Keats and Martt, 1962).

(9) Normal epipercardial fat pads. These fat collections may be confused with cysts and neoplasms. They vary in size with the weight of the patient. (Holt, 1947).

(10) The straight back syndrome, producing flattening of the heart and prominence of the pulmonary artery. Individuals with congenital absence of the normal dorsal kyphosis and narrow sagittal diameter of the chest present striking alterations in cardiac contour compounded by the coexistence of physical findings which may mimic organic heart disease. (Keats, 1975).

(11) Visualization of the pericardium at the right heart border should not be misinterpreted as calcification in the pericardium. (Keats, 1957).

(12) The mediastinal fat may produce widening of the mediastinum. This may be seen in obesity, Cushing's disease and in patients receiving steroids. (Price and Rigler, 1970). This may cause difficulty in assessing the heart size and configuration.

(13) Normal cardiac enlargement in pregnancy. In addition there may be a selective dilatation of the right atrium. (Keats and Martt, 1964).

LIMITATIONS AND PITFALLS OF THE PLAIN RADIOGRAPHY IN ASSESSMENT OF HEART DISEASE

Selective chamber enlargement may be easy to diagnose by plain radiography when the enlargement is gross, when it is confined to a single chamber of the heart or when there is supporting evidence of abnormality from the aorta or pulmonary vessels. There are many fallacies in the diagnosis of selective chamber enlargement produced either by chamber displacement mimicking enlargement or by other structures which may resemble enlarged chambers.

Left ventricular enlargement may be concealed in the frontal view, or by displacing the heart forward against the sternum may suggest that right ventricular enlargement is also present. Left atrial

enlargement may be so gross that the left atrium forms the right heart border and may closely resemble right atrial enlargement, though properly penetrated frontal views should reveal the characteristic features of left atrial enlargement and any left bronchial displacement. When there is doubt about which chamber forms the right heart border, the contribution of the right atrium can usually be identified as it is continuous with the entry of the inferior vena cava passing through the diaphragm to the right atrium.

Left atrial enlargement of even moderate degree commonly displaces the heart forward, giving the impression of simultaneous right ventricular enlargement. Right ventricular enlargement can displace the left ventricle backwards so that the apex of the heart comes to lie behind the barium-filled oesophagus suggesting simultaneous left ventricular enlargement.

Other structures may mimic selective chamber enlargement, e.g. the confluence of the pulmonary veins may mimic the double shadow of selective left atrial enlargement in the penetrated frontal view. The root of the aorta may resemble a normal left atrium but aneurysm of the ascending aorta may resemble either selective left atrial or selective right atrial enlargement. Pericardial defects may resemble left atrial enlargement when the atrium (usually the left) or its appendage herniates through the defect and appears extremely prominent on the frontal radiograph.

After closed mitral valvotomy the removal of the left atrial appendage leaves a concavity below the pulmonary bay, in the position of the previous bulge of left atrial appendicular enlargement. This leaves the heart with an axis elongated to the left and having the appearance of mainly left ventricular enlargement, whatever the underlying pathological state. Apparent abnormalities of cardiac size and shape, resembling overall and selective chamber enlargement, may result from skeletal abnormalities, particularly of the sternum or dorsal spine.

In early childhood it may be difficult to assess both total and selective cardiac chamber enlargement, but some varieties of congenital heart disease tend to develop distinctive cardiac silhouettes as the child grows older. (Raphael, 1986).

FURTHER EXAMPLES OF THE LIMITATIONS OF PLAIN RADIOGRAPHY

(1) Pericardial diseases such as thickening, small or moderate amount of pericardial effusion and tumours are not clearly diagnosed by plain radiography. (Baum, et al., 1980).

(2) Intracardiac tumours or thrombi, cardiac wall thickening or tumours, valvular thickening, vegetation or mild calcification are difficult to detect by plain chest radiography. (Carlsson, et al., 1978).

(3) Patients with ischaemic heart disease such as angina pectoris or myocardial infarction may have a normal chest radiograph. (Godwin, et al., 1984).

(4) Diseases which cause extensive lung opacification may prevent adequate assessment of the heart size and configuration. Examples of such diseases include:-

(a) Agenesis of the lung

(b) Hyaline membrane disease

(c) Consolidation of the lung, especially the lingula, on the left side and the middle lobe on the right side

(d) Large pleural effusion

(e) Post-pneumonectomy

(5) Diaphragmatic abnormalities may also cause difficulty in the assessment of the heart size such as:-

(a) Eventration of the diaphragm

(b) Tumours of the diaphragm

(c) Traumatic rupture of the diaphragm

(d) Elevation of the diaphragm due to large ascites or intra-abdominal masses as well as subphrenic abscess

(6) Skeletal deformities of the thoracic cage such as marked depressed sternum or kyphoscoliosis deformity cause distortion of the shape of the heart and misinterpretation of its size.

(7) Inadequate technique of the plain chest radiograph will lead to inaccurate interpretation. Poor inspiratory and antero-posterior projections produce false impression of cardio-megaly. The elevation of the diaphragm in expiration leads to a transverse position of the heart with apparent increase in its transverse diameter. Antero-posterior projection leads to radiographic magnification of the heart which lies anteriorly in the thoracic cavity. Wrong interpretation of lung congestion and early pulmonary oedema secondary to heart failure often occur in expiratory chest radiographs, especially if it is under-exposed.

The supine position will give the wrong impression of upper lobe blood diversion and may lead to the wrong interpretation of early left heart failure.

Rotation of the patient in the postero-anterior or the lateral chest radiograph may cause misinterpretation in the assessment of the heart size and distortion of its configuration. (Bachman, et al., 1978).

(8) Large anterior mediastinal masses may cause distortion of the cardiac outline and would interfere with accurate assessment of heart size and shape.

(9) The plain chest radiograph is very limited in the diagnosis and evaluation of many of the congenital heart disorders.

SUMMARY OF THE ABBREVIATIONS WHICH ARE USED IN THIS CHAPTER

LV - Left Ventricle

LVPW - Left Ventricular Posterior Wall

IVS - Interventricular Septum

MV - Mitral Valve

AMVL - Anterior Mitral Valve Leaflet

PMVL - Posterior Mitral Valve Leaflets

AV - Aortic Valve

LA - Left Atrium

RA - Right Atrium

RV - Right Ventricle

PE - Pericardial Effusion

AO - Aorta

Th - Thrombus

T - Tumour

Per - Pericardium

2D - Two Dimensional

TWO-DIMENSIONAL ECHOCARDIOGRAPHY IN THE ASSESSMENT OF ACQUIRED HEART DISEASES

Echocardiography plays a very important role in the assessment of a wide range of cardiac disorders. These will include:-

(A) ECHOCARDIOGRAPHIC ASSESSMENT OF CARDIAC ENLARGEMENT

Echocardiography has proved to be of great value in elucidating the cause or causes of a 'large heart'. There are a number of possibilities to be considered, including an increase in the volume of one or more chambers; an increase in left ventricular wall thickness; a pericardial effusion.

It is not uncommon for more than one of these abnormalities to be present in an individual patient. Thus aortic or mitral valve disease might cause enlargement of the left ventricular cavity and an increase in left ventricular wall thickness. In these patients left ventricular end-diastolic pressure may rise, so that left atrial size is also increased whilst associated fluid retention may lead to the development of a small pericardial effusion. (Donner and Soulen, 1986).

(1) ECHOCARDIOGRAPHIC ASSESSMENT OF LEFT ATRIAL ENLARGEMENT

Left atrial volume can be estimated by cross-sectional echocardiography from orthogonal pairs of apical views. Left atrial cavity outlines are traced manually on stop frame images and their areas calculated by Simpson's rule using a microprocessor, allowing left atrial volume to be derived. Both M-mode and two-dimensional echocardiographic estimates of left atrial cavity size have been validated by comparison with those derived from angiography. (Gibson, 1986).

(2) ECHOCARDIOGRAPHIC ASSESSMENT OF LEFT VENTRICULAR ENLARGEMENT

Measurements of left ventricular cavity size are clinically useful, not only in elucidating the cause of cardiac enlargement, but also because of their close relation to left ventricular function. To estimate LV volume from 2-D images

a variety of planes may be chosen to obtain the major and minor dimensions of the chamber and then apply formulae similar to those used in angiography. Because it requires the least assumptions the most satisfactory approach utilizes orthogonal planes and a modification of Simpson's rule. This rule is a mathematical approximation which divides the LV into a large number of slices which are summed to yield ventricular volume. The more distorted or irregular the ventricular chamber the thinner and more numerous the slices which are necessary. Computer assisted measurements and calculations make this complex technique practical. These measurements and calculations are most commonly made from an apical plane and a short axis plane view at the level of the papillary muscles. Multiple short axis views may be utilized in a distorted chamber. There is consistent under-estimation of ventricular volume in comparison with angiography, and 95% confidence limits, about individual estimates, are wide. Volume estimates based on two-dimensional echocardiography should thus be regarded as semi-quantitative only. However it is possible to use cross-sectional echocardiography to obtain an excellent idea of cavity shape and, in particular, to detect left ventricular aneurysm. (Gibson, 1986).

(3) ECHOCARDIOGRAPHIC ASSESSMENT OF RIGHT VENTRICULAR ENLARGEMENT

Right ventricular enlargement is best determined by cross-sectional (21) real-time echocardiography from one or two apical views. As with the left ventricle cavity area is plani-metered from the stop frame image and volume calculated by Simpson's rule. (Bommer, et al., 1979).

The cavity of the right ventricle can be obtained from the subcostal view, particularly in children or in adults in whom the cavity is enlarged. These are best obtained from a cross-sectional display and have been used very correctly in order to investigate right ventricular function. (Gibson, 1986).

(4) ECHOCARDIOGRAPHIC ASSESSMENT OF RIGHT ATRIAL ENLARGEMENT

Right atrial size is estimated by cross-sectional echocardiography in exactly the same manner as that of the left, i.e. from an apical four chamber view. (Bommer, et al., 1979). The largest values are seen in patients with Ebstein's disease or in severe organic tricuspid regurgitation, such as that due to endomyocardial fibrosis. It is helpful when severe right atrial enlargement is present if a peripheral contrast injection of saline is made in order to assess right heart blood flow and tricuspid regurgitation by contrast echocardiography and to investigate the possibility of right to left shunting through a patent foramen ovale. (Gibson, 1986).

LEFT VENTRICULAR HYPERTROPHY

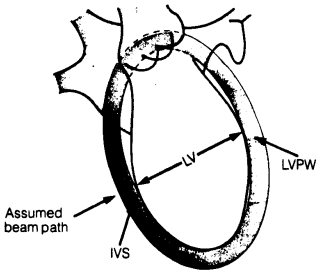
Echocardiography represents the method of choice for assessment of left ventricular hypertrophy. (Fig. 21). In particular it is superior to praecordial palpation and to the ECG, both alternative approaches being relatively insensitive and subject to differences in observer skill and patient body habitus.

In the majority of instances left ventricular hypertrophy causes equal thickening of both the septum and the posterior wall. In perhaps ten per cent of cases left ventricular hypertrophy may manifest on echocardiograms in an asymmetric form with the septum thicker than the posterior wall but without other stigmata of hypertrophic cardiomyopathy. The asymmetry may therefore be artefactual due to the inclusion of the postero-medial papillary muscle or right-sided trabeculations within the thickness of the septum at the point transversed by the ultrasound beam.

Echocardiography can be used to estimate left ventricular mass. The principle of the method is to estimate the total volume of the ventricle, including the myocardium, and to subtract from this the volume of the cavity. This gives the volume of the myocardium, which is converted to mass by multiplying by specific gravity of muscle. This method is accurate if the hypertrophy is symmetrical and provided there are no regional wall motion abnormalities. (Fig. 21). (Leech and Kisslo, 1981).

Fig. 21

PRINCIPLE OF ESTIMATION OF LEFT VENTRICULAR WALL MASS



Calculation of left ventricular mass and volume

Cavity + Wall Volume = (approx) (LV + IVS + LVPW)

Cavity Volume = (approx) (LV)

Wall Volume = (LV + IVS + LVPW) - (LV)

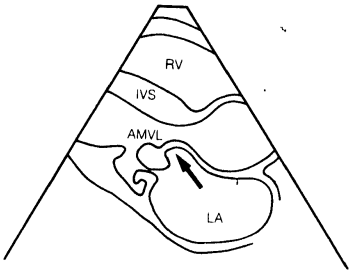
Wall Mass = (LV + IVS + LVPW) - (LV) x 1.05

RIGHT VENTRICULAR HYPERTROPHY

Right ventricular hypertrophy is commonly associated with any form of right ventricular outflow obstruction or pulmonary hypertension, which may in turn owe its origin to left-sided cardiac disease. The echocardiographic signs are thickening of the anterior right ventricular wall and septum. Cavity size is usually normal or slightly enlarged. In many cases there is associated volume overload due to tricuspid regurgitation but, in the absence of this, septal motion is normal. M-mode echocardiography is not very sensitive for detecting right ventricular hypertrophy. Two-dimensional studies, utilizing the subcostal four-chamber view, can overcome the uncertainty of the beam pathway and may improve the assessment.

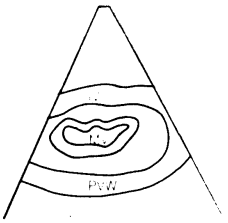
Biventricular hypertrophy, which is difficult to analyse from ECG recordings, can be demonstrated dramatically by echocardiography. (Leech and Kisslo, 1981).

Fig. 22



A.

2-D parasternal long aixs view demonstrates thickening of the leaflet tips and doming of the anterior leaflet in a case of mitral valve stenosis.



B.

2-D parasternal short axis view in a case of mitral stenosis

ECHOCARDIOGRAPHY IN ASSESSMENT OF VALVULAR HEART DISEASE

The detailed imaging of valve leaflets and their support structures afforded by echocardiography permits judgement of mobility, deformity, calcification and, for most mitral and some aortic valves, direct measurement of orifice size. These features provide a diagnosis and together with chamber analysis they serve as a guide to severity, aid clinical management decisions and provide means of following the response to treatment for such diverse valvular lesions as rheumatic and sclerotic disease, endocarditis and prolapse. Unless concommitant coronary artery disease is of concern, echocardiography may obviate cardiac catheterization in many patients with acquired valvular disease. The leaflets of bioprostheses (tissue valves) can be similarly assessed. Complications of prosthetic valves such as thrombus, dehiscence and poppet extrusion are well evaluated by 2-D imaging - again permitting surgery without invasive diagnostic tests. (Donner and Soulen, 1986).

MITRAL VALVE DISEASES

(A) RHEUMATIC MITRAL VALVE DISEASE

The cross-sectional echocardiographic findings of rheumatic mitral valve disease are characteristic. (Nichol, et al.,1977). On the parasternal long axis view diastolic motion of the tips of the mitral cusps is restricted, leading to a bowed appearance during ventricular filling if the cusp is pliable. (Fig. A). In the short axis view the mitral valve area is reduced during early diastole and semi-quantitative estimates can be made of this figure. (Fig. B). With appropriate gain settings a mitral orifice can be outlined and an area measured. (Weyman, et al., 1979). Such estimates have been shown to agree moderately well with values derived from cardiac catheterization. Certain technical problems must, however, be considered. The anterior part of the orifice is formed by the trailing edge of the anterior cusp echo which may appear much thicker than the echo itself. The medial and lateral margins of the orifice are subject to the limitations of lateral resolution of all echocardiographs approximately 3 to 4 mm. Both these factors are very sensitive to gain setting. (Fig. 22 A).

The mitral orifice can also be viewed from the apical approach, a view which also shows the sub-valve apparatus. In many cases the actual orifice can be shown to pass obliquely from atrium to ventricle, further reducing the accuracy with which the true orifice is measured from the parasternal minor axis view.

Cross-sectional echocardiography can also demonstrate the whole of the left atrial cavity. Its volume can be measured. A raised left atrial pressure is associated with bowing of the inter-atrial septum into the right atrium. In a minority of cases intra-atrial clot can be demonstrated.

Echocardiography may also demonstrate considerable thickening of the anterior cusp echo, a finding which is frequently correlated with the presence of calcification. The apparent thickness of the cusp echo over-estimates that of the valve itself, measured at surgery, due to multiple reverberations of the ultrasound within the calcium. (Gibson, 1986).

(B) NON-RHEUMATIC MITRAL VALVE DISEASE

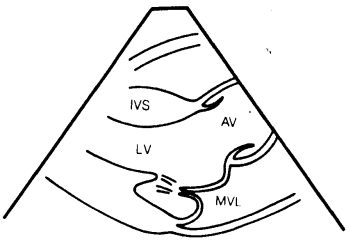
I. MITRAL VALVE PROLAPSE

The pattern of motion as demonstrated by cross-sectional echocardiography is characteristic. Although the cusps oppose normally, the central portion of one or both leaflets bulges into the left atrium. This abnormality is best demonstrated by defining the line of the mitral ring as that from the point of attachment of the anterior cusp to the aortic root to that of the posterior cusp to the posterior wall. Two mm. or more of posterior displacement of the mitral cusps in systole makes the diagnosis of prolapse very likely. (Figs. 23A and B).

Mitral prolapse is frequently associated with a floppy valve and redundant tissue may be demonstrated by cross-sectional techniques. If a floppy mitral valve is present then the tricuspid and aortic valves should also be examined since they may also be affected by a similar abnormality. Left ventricular cavity size and the amplitude of its wall movement are usually normal since associated mitral regurgitation is seldom severe.

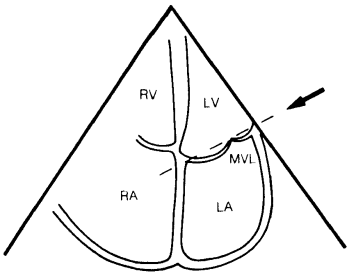
The appearances in well developed mitral prolapse are clear-cut and diagnostic, but in mild cases difficulties have arisen leading to considerable over-diagnosis. (Gibson, 1986).

Fig. 23



A.

2-D parasternal long axis view during systole demonstrates mitral prolapse



B.

2-D apical four chamber view during systole demonstrates mitral valve prolapse

II. CHORDAL RUPTURE AND PAPILLARY MUSCLE DYSFUNCTION

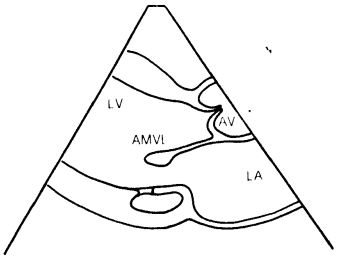
In contrast with simple mitral prolapse normal coaption of the cusp margins during systole does not occur, but instead one or other cusp is displaced backwards into the left atrium. At the same time the underlying cause of the regurgitation is frequently apparent. There may be vegetations indicating infective endocarditis. Anterior chordal rupture can be well demonstrated as can redundant cusp tissue. In rare cases partial or complete papillary muscle rupture can be recognized after myocardial infarction.

The left atrial cavity is enlarged, but not to the same extent as in rheumatic mitral valve disease. More characteristic, however, is an increase in LA volume associated with bulging of the interatrial septum into the right atrium. (Gibson, 1986).

Mitral regurgitation may also be due to disease of the papillary muscles. This usually occurs in the setting of acute myocardial infarction. (Figs. 24A and B). Papillary muscle rupture may be partial or complete (Nishimura, et al., 1982) and in both cases is associated with an abnormal increase in cusp mobility and failure of systolic valve coaption. (Fig. 25).

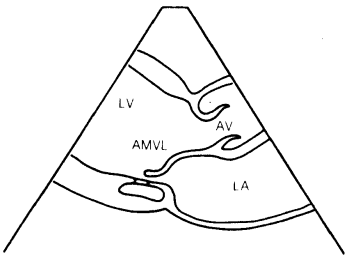
Papillary muscle rupture must be distinguished from the very much commoner entity of papillary muscle dysfunction in which valve cusp motion is normal. The primary abnormality is left ventricular disease, which can be frequently detected echocardiographically as cavity dilatation or from regional abnormalities of wall motion. Not infrequently areas of high echo intensity are apparent in the papillary muscles themselves, probably representing local scarring. (Gibson, 1986). (Figs. 24 and 25).

Fig. 24



A.

2-D parasternal long aixs views from a patient
with chronic severe mitral regurgitation - diastole

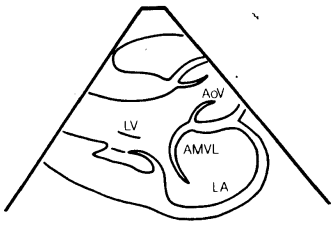


B.

Systole

Only the anterior leaflet has moved as the posterior leaflet is fixed

Fig. 25



2-D parasternal long axis view
demonstrates chordal rupture

III. CALCIFIED MITRAL RING

Cross-sectional echocardiography allows the distribution of the calcium to be outlined and spread into the septum where it might be associated with complete heart block and can be detected. In addition possible interference with mitral valve action can be assessed.
(Fulkerson, et al., 1979). (Gibson, 1986).

TRICUSPID VALVE DISEASE

The tricuspid valve cusps are best demonstrated by the cross-sectional approach using the apical or subcostal view. Normal tricuspid valve motion is similar to that of the mitral valve. The cusps are thin and open widely during diastole. Rheumatic involvement is suggested by restriction of their motion with doming into the right ventricular cavity during diastole. A minor degree of thickening of the cusps may occur but never to the same degree as can occur in calcific mitral stenosis.

Tricuspid regurgitation from whatever cause is best demonstrated using a peripheral venous echo contrast injection. The right side of the heart is visualized and the circulation time is reduced. A much more specific sign is the accumulation of contrast in the inferior vena cava after an injection into an arm vein. (Gibson, 1986).

The aetiology of tricuspid regurgitation may sometimes be determined by observation of the valve cusps. Vegetations can be identified. Rheumatic involvement is associated with minor thickening and restriction of motion. In Ebstein's anomaly the characteristic abnormality of misplacement of the insertion of the septal cusp of the tricuspid valve can be recognized on an apical four chamber view. Severe tricuspid regurgitation may also occur in endomyocardial fibrosis associated with thickening of the subvalve apparatus and obliteration of the apex of the right ventricular cavity by fibrosis and thrombus. In carcinoid heart disease the tricuspid valve cusps are thickened and there may be tricuspid regurgitation causing right ventricular overload. In carcinoid heart disease the pulmonary valve may also be affected and the right ventricular endocardium may appear thickened with increased echo amplitude. (Callaghan, et al., 1982).

AORTIC VALVE DISEASE

(a) CALCIFIC AORTIC STENOSIS

Echocardiography demonstrates disorganization of the normal aortic valve cusp anatomy and its replacement by multiple, highly reflecting targets within the aortic root. Neither M-mode nor cross-sectional techniques can be used to estimate valve area due to their limited resolution and the reduction in the apparent orifice size by multiple reverberations within the abnormal cusps. (De Maria, et al., 1980).

The overall extent of the calcification can be assessed, including any spread into the mitral valve or the upper part of the IV septum. The size of the aortic root and the lower portion of the ascending aorta can be measured from the para-sternal long axis view.

In most cases left ventricular cavity size is normal or small, but wall thickness may be strikingly increased and the pattern of hypertrophy can be well seen. Although it is frequently uniform hypertrophy, this is not always the case and selective involvement of the apex or the septum, particularly its upper part, is not uncommon.

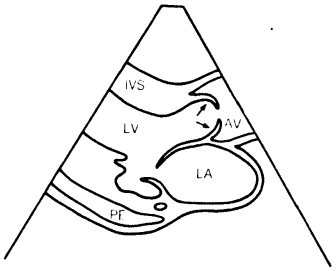
In a minority of cases in the late stages of cardiac failure the left ventricular cavity size is greatly increased, and the amplitude of wall motion reduced so that the appearances are those of congestive cardiomyopathy. (Gibson, 1986).

(b) RHEUMATIC AORTIC VALVE

Most patients with rheumatic valve disease have some echo-cardiographic abnormality of the aortic valve.

Long axis two-dimensional scans can display thickening and disordered mobility of the right and non-coronary cusps. Short axis scans are more useful; they provide information on orifice size and on the degree to which each cusp is involved. (Rodgers, 1985). (Fig. 26).

Fig. 26



2-D parasternal long aixs view demonstrates aortic stenosis and a small amount of pericardial effusion

(c) AORTIC INCOMPETENCE

Cross-sectional echocardiography gives a comprehensive view of the aortic valve and root, which usually allows the aetiology of aortic regurgitation to be determined. Vegetations on the valve cusps can be identified and occasionally abscess cavities can be located. The aortic root and ascending aorta are well seen in the long axis parasternal view so that dilatation of the valve ring or aortic root can be detected. In a dissecting aneurysm with involvement of the aortic valve the endothelial flap can usually be identified. The thickened cusps with rolled edges characteristic of rheumatic aortic valve disease cannot be identified directly, but mitral valve involvement is strong presumptive evidence for a rheumatic aetiology. (Gibson, 1986).

(d) FLOPPY AORTIC VALVE

Myxomatous proliferation can affect the aortic valve, producing a floppy aortic valve, which can become regurgitant. This diagnosis should be considered when the aortic leaflets appear thick but fully mobile on two-dimensional scans. (Rodgers, 1985).

(e) BICUSPID AORTIC VALVE

On cross-sectional echocardiography a bicuspid aortic valve shows thickened cusps and in the parasternal long axis view can be seen to 'dome' into the aortic root during LV systole. (Zemma and Caccavano, 1982). (Fig. 26). On the parasternal minor axis view the normal picture of three symmetrical cusps is modified. In some cases there may be only a single commissure and in other patients one small and two large aortic cusps. (Gibson, 1986).

(f) PULMONARY VALVE

The posterior cusp of the pulmonary valve is usually visible on short axis scans at aortic valve level and one of the anterior cusps may also be seen. Abnormalities of the pulmonary valve may be assessed by echocardiography. (Rodgers, 1985).

ECHOCARDIOGRAPHIC ASSESSMENT OF PROSTHETIC VALVES

Echocardiography is sometimes a useful tool for the management of patients who have one or more prosthetic valves.

The aim of the echocardiographic study is to visualize the valve itself along with its situation and surrounding structures and also to evaluate left ventricular function. The very high M-mode sampling rate generally makes it preferable to two-dimensional echocardiography for this purpose, although the latter can permit more rapid location of the valve and may assist in left ventricular function studies.

Because changes in echocardiograms may be so subtle with prosthetics, every patient having a valve replacement should be studied whilst still in hospital, about two weeks after the operation. This provides a baseline against which to compare any later recordings. Since cardiac function in such patients remains abnormal, alterations in their haemodynamic status must be deduced from changes in the echo parameters rather than their absolute values. (Leech and Kisslo, 1981).

(C) ECHOCARDIOGRAPHIC FINDINGS IN CARDIOMYOPATHY

(a) CONGESTIVE CARDIOMYOPATHY

Cross-sectional echocardiography in congestive cardiomyopathy confirms the increase in LV cavity size and usually demonstrates the reduction in amplitude of wall motion to be uniform. However in a minority of cases striking regional difference in the amplitude of LV wall motion may be seen in patients with congestive cardiomyopathy in whom there is no evidence of coronary artery disease. With cross-sectional echocardiography it is also possible to identify or exclude a left ventricular aneurysm. In some cases intra-ventricular thrombus may be identified. (Fig. 27).

In all patients suspected of having congestive cardiomyopathy the aortic valve must be visualized echocardiographically to confirm that cusp movement is normal and to exclude severe end-stage aortic stenosis, which may present clinically in the same way. It is very important to identify this presentation of aortic stenosis since the response to early cardiac surgery is usually excellent, however bad left ventricular function may appear to be.

The differential diagnosis between congestive and ischaemic cardiomyopathy cannot usually be made with certainty by echocardiography, although septal akinesis, with failure of systolic thickening, is in favour of the former.(Gibson, 1986).

2-D imaging is the method of choice for detecting and following the ventricular thrombi commonly associated with congestive cardiomyopathies. (Donner and Soulen, 1986).

(b) HYPERTROPHIC CARDIOMYOPATHY

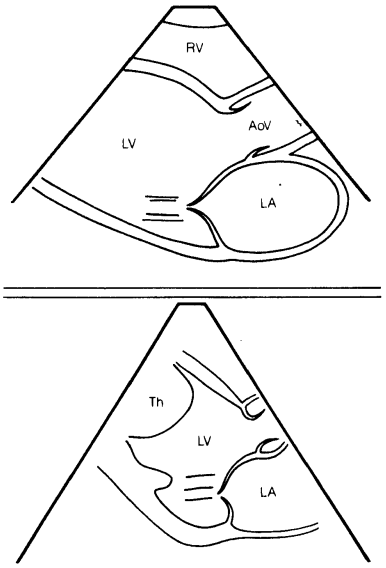
The pattern of hypertrophy can be studied by cross-sectional echocardiography. (Fig. 28). In many cases the septum is involved, either throughout its length, or only in part. In a variant there is selective apical involvement.
(Nishyama, et al., 1979).

In such cases the base of the left ventricle, and thus M-mode echocardiography, may be entirely normal. In other cases the hypertrophy is generalized or may involve the posterior wall preferentially. The hypertrophied myocardium may present an abnormal texture in the cross-sectional image, described as a 'ground glass' appearance. (Werner, et al., 1981). (Fig. 28).

(c) RESTRICTIVE CARDIOMYOPATHY

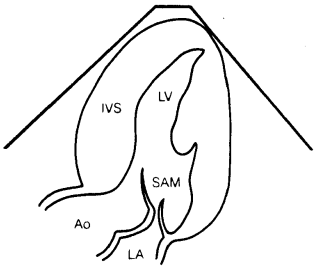
The typical structural abnormalities of this condition can be demonstrated by cross-sectional echocardiography. Apical cavity obliteration involving either right or left ventricle is apparent and is frequently accompanied by an apical dimple, where the endocardium is drawn inwards by fibrosis. Involvement of the posterior cusp of the mitral valve is particularly common as it is replaced by a mass of scar tissue. Atrioventricular valve incompetence is associated with cavity enlargement, particularly when the right side of the heart is affected. In severe cases there may be no evidence of tricuspid valve tissue at all, with the right atrium and ventricle appearing as a single large chamber, distorting the anatomy of the remainder of the heart. When the venous pressure is greatly raised a pericardial effusion is often present. (Davies, et al., 1982). (Oakley and Olsen, 1977).

Fig. 27



2-D parasternal long axis view

Fig. 28



2-D apical long axis view demonstrates hypertrophic cardiomyopathy

(D) ECHOCARDIOGRAPHY IN CARDIAC MASSES

The spatial orientation of 2-D echocardiography permits intracardiac masses to be detected in any chamber and characterized as to their site of attachment, shape, size and mobility. These features, together with concomitant valvular or chamber abnormalities, allow thrombus, vegetations and tumours to be differentiated in most instances. Detection in more than one plane differentiates true masses from technical artefacts. The tomographic nature of the 2-D images must never be forgotten; even large masses can be missed if the sound beam does not pass through them, an unlikely event if each chamber is imaged in multiple views.

The documentation of thrombi, particularly if large, may alter the surgical approach to the mitral valve, lead to anticoagulation, or even to thrombectomy if the thrombus is shown to be causing obstruction or if it is mobile. Serial echocardiographic studies can show dramatic alterations in endocardial vegetations in response to appropriate antimicrobial therapy. Myxomas are so characteristic in echocardiographic appearance that surgery may be performed without prior invasive catheter diagnostic studies. In addition to characterizing a mass itself, 2-D imaging permits evaluation of the mass in relation to surrounding cardiac structures - thus providing the surgeon with a road map which may be all that is required preoperatively.

It is very important to image the inferior vena cava and its hepatic and renal tributaries in all patients with right atrial masses. The management of tumour extending transvenously from liver or kidney is clearly different from that of an isolated right atrial mass. Detection of intramyocardial masses (e.g. tumour, granuloma) has been disappointing, although some success has been reported in identification of myocardial abscesses associated with sinus tracts from infected prosthetic valves. Paracardiac masses such as pericardial cysts or mediastinal tumour, with or without cardiac compression or invasion, can be imaged and thus obviate thoracotomy, or serve as a guide to biopsy respectively. (Donner and Soulen, 1986).

(a) ATRIAL MYXOMA

Cross-sectional echocardiography is of major value in diagnosis of left atrial myxoma. The site of attachment of the tumour can be defined; usually it is to the lower part of the inter-atrial septum. Echocardiography can also be used to establish the presence of sessile myxomata within the atrial cavity which do not impinge on the mitral valve apparatus at any time in the cardiac cycle.

When these typical appearances are present echocardiography is diagnostic of left atrial myxoma and invasive investigations (e.g. cardiac catheterization) with their attendant risk of systemic embolism, are strongly contra-indicated before surgery. (St. John Sutton, et al., 1980). (Fig. 29, A and B).

Left atrial myxoma must be distinguished from left atrial thrombus. In order for thrombosis to occur the left atrium is enlarged and atrial fibrillation is likely to be present. (De Pace, et al., 1981). Thrombi are nearly always sessile and reflect ultrasound with low density unless they are calcified. Rarely they may be pedunculated or even free floating, when their characteristic motion around the enlarged atrium can be clearly visualized.

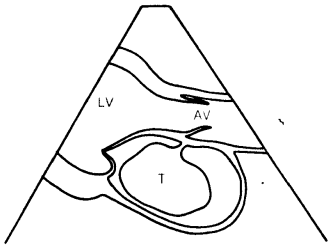
The Chiari network can frequently be demonstrated within the normal right atrium and must be distinguished from tumours and other masses. Right atrial myxoma is well documented although less common than left atrium myxoma. The tumour can be demonstrated by cross-sectional technique either in association with the tricuspid valve or, if pedunculated, in the RV outflow tract. In some cases myxomata of both atria may be present. Right atrial myxoma must be distinguished from:-

(1) Tricuspid valve vegetations, particularly those due to fungal infections, which may be large.

(2) Free floating clots from peripheral veins, which may occasionally be visualized in their passage through the right heart.

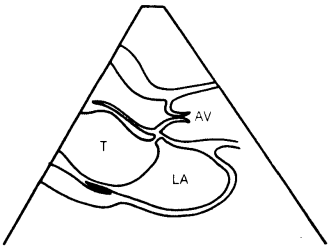
(3) Direct extension of tumour up the inferior vena cava, e.g. from a hypernephroma. (Gibson, 1986).

Fig. 29



A.

2-D parasternal long axis view demonstrates left atrial tumour (T)



B.

Systole. The mitral leaflets are closed and the tumour lies in the left atrium. Attachment of the tumour just above the anterior mitral valve leaflet

(E) ECHOCARDIOGRAPHY IN ISCHAEMIC HEART DISEASE

I. ECHOCARDIOGRAPHIC ASSESSMENT OF LEFT VENTRICULAR FUNCTION

Cross-sectional echocardiography can be used to assess both overall and regional left ventricular function in patients with coronary artery disease. This ability is clinically significant in view of the overriding importance of left ventricular function as a determinant of proghosis in patients with acute or chronic coronary artery disease. (Hammermeister,1983).

The regional abnormalities of left ventricular function most easily studied by cross-sectional echocardiography are those of the amplitude of endocardial motion. The aim is to measure or to estimate the direction and extent of wall motion between end-diastole and end-systole.

This is not a straightforward procedure. In coronary artery disease systole ends at different times in different regions of the left ventricle, so that end-systole is not a clearly defined event. It extends throughout the period of isovolumic relaxation and in exceptional cases early into the period of ventricular filling. Since very significant changes in left ventricular cavity shape may occur during isovolumic relaxation in these patients due to asynchronous onset of relaxation, the apparent pattern of regional wall motion is critically affected by the exact time chosen for end-systole.

A second problem is that of overall motion of the heart in space. An apparent reduction in the amplitude of wall motion will occur in any region if the heart as a whole moves in the same direction.

It is against this complex background that attempts to assess regional function by echocardiography must be judged. The majority of reported studies have been based on subjective assessment of videotapes, where these problems are not usually

considered. Nevertheless, remarkably close agreement with angiographic left ventriculography has been reported. (Gibson, 1986).

Alternatively, objective methods of image analysis may be used. End-diastolic and end-systolic cavity outlines are traced using a simple computer system, so that the motion of the endocardial boundary between the two can be assessed. The most satisfactory means of doing this has been to use regional area changes rather than simple measurements of distance and perimeter. (Moynihan, et al., 1981). (Parisi, et al., 1981).

If disturbances in the timing of movement, as well as those of amplitude, are to be measured, then more complex methods must be used. These have included an approach based on frame by frame digitization of minor axis views and also one in which the two dimensional display is presented as a series of M-mode echocardiograms. (Gibson, et al., 1978).

Theoretically, problems due to overall motion of the heart in space can be avoided by studying wall thickening rather than endocardial position. However, the distance involved - 8 to 12 mm. or even less - is only two to three times the lateral resolution of currently available systems. Unfortunately the rapid rate of change of wall thickness with time greatly reduces the effectiveness of computed tomography for this application. (Gibson, 1986).

Cross-sectional echocardiography has also been used to localize regional disturbances of wall motion due to acute myocardial infarction. Again most reported studies have been based on subjective analysis of video tapes, but excellent agreement with electrocardiographic localization has been found. (Nixon, et al., 1980). (Heger, et al., 1979).

II. ECHOCARDIOGRAPHY IN ANGINA

Echocardiography has been used in a number of ways in the investigation of patients with anginal pain. In the resting state it may be possible to demonstrate the presence of some disease entity known to be associated with angina such as - hypertrophic cardiomyopathy; severe left ventricular hypertrophy complicating aortic stenosis; well developed mitral valve prolapse. It should be remembered, however, that any of these conditions may coexist with coronary artery disease. (Gibson, 1986).

Two-dimensional echocardiography has also been performed during dynamic exercise in patients with coronary artery disease and it has proved possible to demonstrate regional abnormalities of wall motion in association with pain and ECG changes. (Morganroth, et al., 1981). This approach is only practicable in a small minority of cases, but has the advantage that the images are registered in real time rather than over a period of two to three minutes required for a gated blood pool isotope scan. It has thus been possible to demonstrate that the pattern of wall motion changes rapidly with time at the end of exercise in a patient who develops angina and may have reverted to normal within the time required to collect the counts for a gated blood pool image.

It is also possible to use cross-sectional echocardiography to study events during an attack of angina occurring spontaneously at rest. The earliest change to occur is in diastole, a reduction in peak myocardial thinning rate. This may precede either ECG changes or symptoms. As the attack develops regional amplitude of wall motion is reduced and finally, outward movement during systole movements may be seen. Such observations give fundamental information about mechanisms underlying angina and related phenomena and may be helpful in individual patients in determining the nature of symptoms. (Gibson, 1986).

III. ECHOCARDIOGRAPHIC ASSESSMENT OF LEFT VENTRICULAR ANEURYSM

Cross-sectional echocardiography has proved valuable in distinguishing left ventricular aneurysm from global enlargement of the cavity. Aneurysms are usually apical and should be studied from the subcostal or the apical approach. They are fibrous and are thus akinetic. Apparent paradoxical motion results in part from optical illusion and in part from overall motion of the heart in space.

In making the diagnosis of aneurysm it is necessary not only to document apical or inferior expansion of the left ventricular cavity outline at end-diastole with akinesis during ejection, but also to demonstrate the reversal of curvature at their 'neck' with the normal myocardium. This is always present during systole and may persist during diastole. This criterion may allow an aneurysm to be distinguished from an extensive area of akinesis. (Weyman, et al., 1976). (Sorensen, et al., 1982).

IV. LEFT VENTRICULAR PSEUDOANEURYSM

Left ventricular pseudoaneurysm represents localized cardiac rupture into the pericardial space. It is an uncommon complication of acute myocardial infarction. It may be recognized on cross-sectional echocardiography as an echo-free space communicating with the ventricular cavity by a narrow neck, but otherwise separated from it by myocardium. In occasional cases acute cardiac rupture, which characteristically causes a sudden bradycardia, has been detected immediately after it occurred by cross-sectional echocardiography, allowing urgent surgery to be performed. (Gibson, 1986).

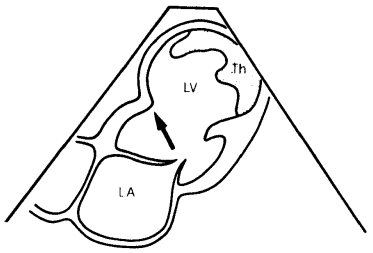
V. ECHOCARDIOGRAPHIC ASSESSMENT OF INTRAVENTRICULAR THROMBUS

Cross-sectional echocardiography has proved useful in detecting intraventricular thrombus. Thrombus may occur with uniform cavity dilatation, as in congestive or ischaemic cardiomyopathy, ventricular aneurysm or after acute myocardial infarction.

Fresh thrombus is mobile and strands may be observed floating freely within the cavity; later the thrombus becomes flattened against the ventricular wall and produces more echo densities as it organizes. Thrombi are most commonly seen at the left ventricular apex and less so on the posterior left ventricular wall or involving the distal apical portion of the interventricular septum. (Fig. 30). (Metzer, et al., 1979).

The texture of the echoes in the mass is frequently different from that of the adjacent myocardium. The motion of the adjacent myocardium itself is either reduced or absent. Normal motion of adjacent myocardium makes thrombus most unlikely. Using these criteria thrombus can usually be distinguished from artifacts arising from the transducer itself, from side lobes or from limitation of lateral resolution. On occasions, however, it may remain uncertain after a full examination as to whether intraventricular thrombus is present. (Ports, et al., 1978).

Fig. 30



2-D apical four-chamber view of a left ventricular aneurysm containing thrombus

VI. ECHOCARDIOGRAPHIC MYOCARDIAL STRUCTURE

Replacement of myocardium by fibrosis is common in ischaemic heart disease. The stiffness of collagen is very much greater than that of normal myocardium so that, not unexpectedly, it gives rise to ultrasound of greater amplitude than that from normal myocardium. (Rasmussen, et al., 1978).

Increased echo amplitude may also be detected in the papillary muscles, particularly in patients with murmurs suggestive of papillary muscle dysfunction.

Regional sonolucence has been detected in the septum after massive myocardial infarction in elderly patients. Its genesis is uncertain, but it may represent necrosis or may result from intramyocardial haematoma. The field of echocardiographic tissue characterization is a developing one, and may well lead to further advances.
(Logan Sinclair, et al., 1983).

VII. Post-infarction ventricular septal defect and mitral regurgitation can also be assessed by 2-D echocardiography. Gibson, 1986).

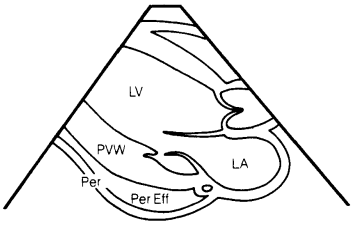
(F) ECHOCARDIOGRAPHY IN PERICARDIAL DISEASE

PERICARDIAL EFFUSION

Ultrasonography is the most accurate and least invasive method of diagnosing pericardial effusion, but it cannot determine its nature whether it is transudate, exudate or blood. An echo-free area beyond the confines of the cardiac chambers and surrounding all borders of the heart, using two-dimensional ultrasound (Fig. 3), is diagnostic. Small loculated effusions can be appreciated and changes in cardiac wall motion can be studied. Effusions as small as 75 ml. can be detected by echocardiography. (Walinsky, 1978).

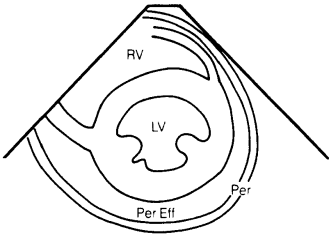
Small effusions are seen localized next to the posterior left ventricular wall. Larger effusions form an echo-free area anterior to the right ventricle in addition to the posterior fluid collection. Fat anterior to the heart can be a source for false positive interpretation of loculated anterior pericardial effusion. (Ellis and King, 1973). (Steiner and Rao, 1986).

Fig. 31
(A & B)



A.

2-D parasternal long axis view demonstrates pericardial effusion



B.

2-D parasternal short axis view of the same case

The following questions should be considered whilst performing the real-time Echo study on a patient with a pericardial effusion:-

(a) Does the pericardial fluid contain any 'material' or is it an 'echo-free' space?

(b) If material is present is it:-

linear fibrous strands

globular

mass-like?

(c) Is the surface of the visceral pericardium (or epicardium) smooth or is it 'roughened' ('serrated') in appearance?

(d) Is the outer layer of pericardium (parietal pericardium) thickened?

(e) Is the effusion uniformly surrounding the heart or is it 'loculated'?

(f) What amount of fluid is present (moderate or large)?

(g) Is there abnormal diastolic movement of the right ventricular free wall or of the right atrial wall?

(h) Do both ventricles 'contract' well or not?

Are they dilated in diastole?

Does ventricular diastolic expansion appear 'stented' (i.e. come to an abrupt stop)?

(i) Does the ventricular septum demonstrate reduced movement or is it over-active and appearing to produce most of the systolic movement rather than the ventricular free walls?

(j) Has either the pericardial fluid volume or apparent ventricular function changed significantly during sequential studies?

(k) Is the aortic root dilated or is there evidence of aortic regurgitation?

Do the aortic root, ascending aorta or the aortic arch contain echoes suggesting dissection?

Some of the features listed above may be detected in more than one pericardial disorder and multiple features may co-exist in any one disease state. (Williams, 1986).

(a) Pericardial cysts, absence of pericardium and pericarditis can also be displayed by echocardiography. (Williams, 1986).

(b) Echocardiography is less satisfactory in evaluation of pericardial thickening or constriction.

(c) Pericardial calcification can be detected by echocardiography.

(G) ECHOCARDIOGRAPHY IN THORACIC AORTA DISEASE

The intra-pericardial portion of the ascending aorta is usually well imaged, permitting detection of abnormalities involving the aortic root such as dissecting haematomas or aneurysms. Although very valuable in children, particularly for coarctation and patent ductus, the suprasternal views are seldom useful in the adult and the transverse aortic arch may be difficult to image. The descending thoracic aorta, however, can often be imaged as it courses behind the left heart. Both dissections and aneurysms may be detected by echocardiography in this aortic segment. (Donner and Soulen, 1986).

MATERIAL AND METHODS

Sixty patients who had two-dimensional echocardiographic examinations were reviewed - fifty patients from "Lodge Moor Hospital", Sheffield, England and ten patients from "El Hussin Hospital", Cairo. (Table 10).

TABLE 10

Origin of patient	No. of cases	Male	Female	Maximum age (years)	Minimum age (years)	Mean age (years)
Lodge Moor Hospital Sheffield	50	26	24	85	7	66.5
El Hussin Hospital Cairo	10	7	3	42	11	25.5

All the cases from "Lodge Moor Hospital", which is a general hospital without a specialized cardiac service, were under the care of general physicians. The cases from "El Hussin Hospital" were from the cardiology department.

The aim of this study is to assess the contribution of two-dimensional echocardiography to the management of these patients and to compare the echocardiographic findings with those of the plain chest radiographs.

All the ultrasound examinations were performed with a real time sector scanner using 3.5 MHZ transducer. The upright sitting position was used in most cases. Parasternal long axis, apical and subcostal four-chamber were the only planes used to examine the heart of these patients. The short axis planes were not used and no measurements were taken. The interpretation of the examinations was mainly subjective and thus depended on the experience of the examiner and the quality of the examination.

The examiners observed the following structures and functions in the long axis view:-

(1) Composite size of the cardiac chambers and the presence or absence of pericardial effusion.

(2) Contractility of the right ventricle and left ventricle.

(3) Thickness of the right ventricular wall.

(4) Continuity of the interventricular septum with the anterior wall of the aorta.

(5) Pliability of the mitral and aortic valves.

(6) Coaptation of the mitral valves.

(7) Presence of increased echoes on the mitral and aortic valves.

(8) Systolic clearance of the aortic valve.

(9) Presence of abnormal echo collections in the chambers or attached to the valve orifices.

(10) Presence and movement of chordal-papillary muscle structures.

(11) Thickness of the septum and the posterior wall of the left ventricle.

(12) Uniform texture of the endocardium-myocardium.

(13) Size of the aortic root.

The apical four-chamber view was used to assess the following:-

(1) Size of cardiac chambers.

(2) Contractility of the heart.

(3) Septal thickness, contractility and continuity.

(4) Pericardial effusion.

(5) Increased echoes of the valves (mitral, tricuspid and aortic).

(6) Presence or absence of flail leaflet.

(7) Presence or absence of thrombus or mass in chambers.

(8) Size of the left ventricular outflow tract plus signs of obstruction.

The apical long axis view is excellent for assessing the contractility and thickness and to look for mass lesions in the left ventricle.

No Doppler echocardiography was carried out in any of these patients.

The echocardiographic examinations were recorded on videotapes and they were reviewed without knowledge of the final diagnosis, but the clinical indication was available. The interpretations were checked by the supervisor before documenting the findings.

The plain chest radiographs which were carried out within twenty four hours of the echocardiographic examinations were reviewed without knowledge of the echocardiographic findings. The interpretation of the chest radiographs was compared with the echocardiographic findings. The additional information which had been acquired by the ultrasound was evaluated.

CASE REPORTS

Case No: 1.
Age: 79
Sex: Female

Clinical Presentation: Shortness of breath
Subcostal pain

Past Medical History: Peptic Ulcer

Physical Findings: Dyspnoea
Wheeze in both lungs
Clinical features of aortic stenosis and mitral regurgitation

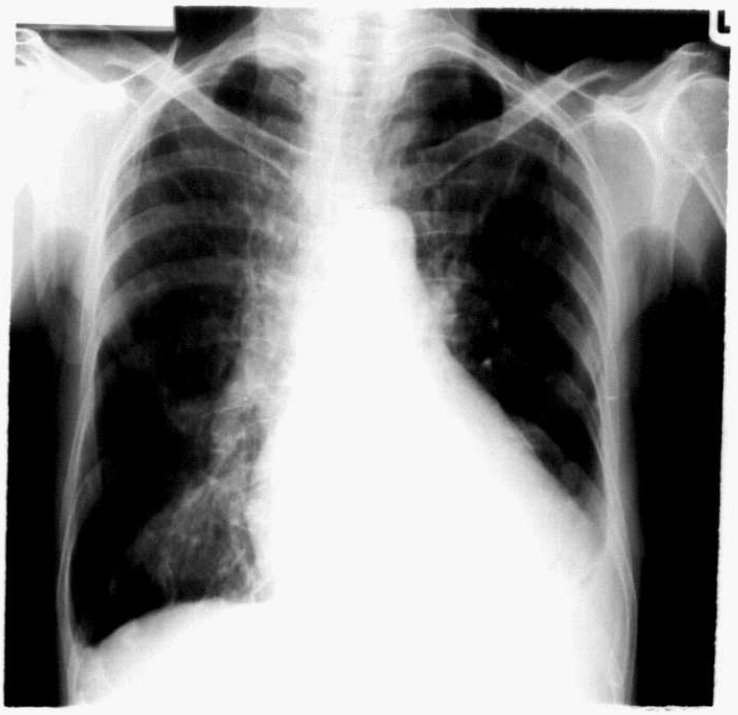
X-ray Report: The heart size is enlarged. Lungs are congested with left pleural effusion. The appearance indicates heart failure. No active focal lung lesion.

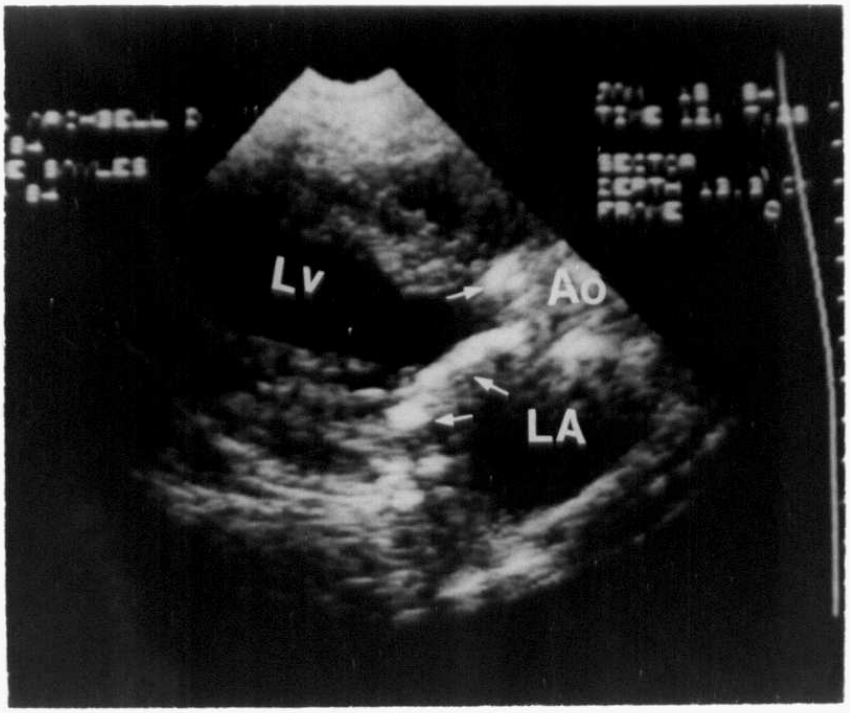
Echocardiography:

LA:	moderate dilatation
IAS:	normal
LV:	thickened and hypertrophic wall
MV:	thickened and calcific leaflets with stenosis
AV:	calcific cusps with stenosis
RA:	normal
RV:	normal
TV:	normal
IVS:	thickened
Pericardium:	normal
Aorta:	normal

Clinical Course and Follow up: The patient has improved with medical treatment

Final Diagnosis: Mitral and Aortic Valve Stenosis





Case No: 2.

Age: 65

Sex: Male

Clinical Presentation: Shortness of breath
Cough with expectoration of purulent sputum

Past Medical History: Pulmonary tuberculosis
Resection of lung carcinoma six years previously
Heart failure
Pneumonia of the left lung

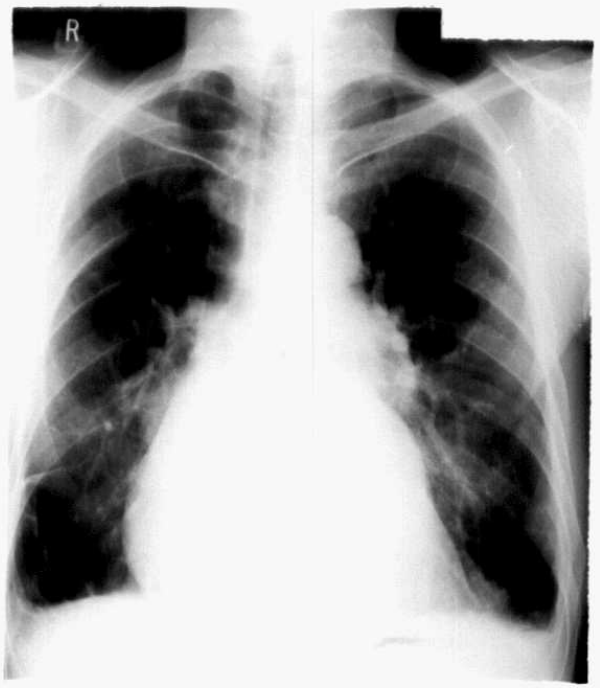
Physical Findings: Pulse - 77/minute irregular
BP - 110/70
No murmur or signs of heart failure

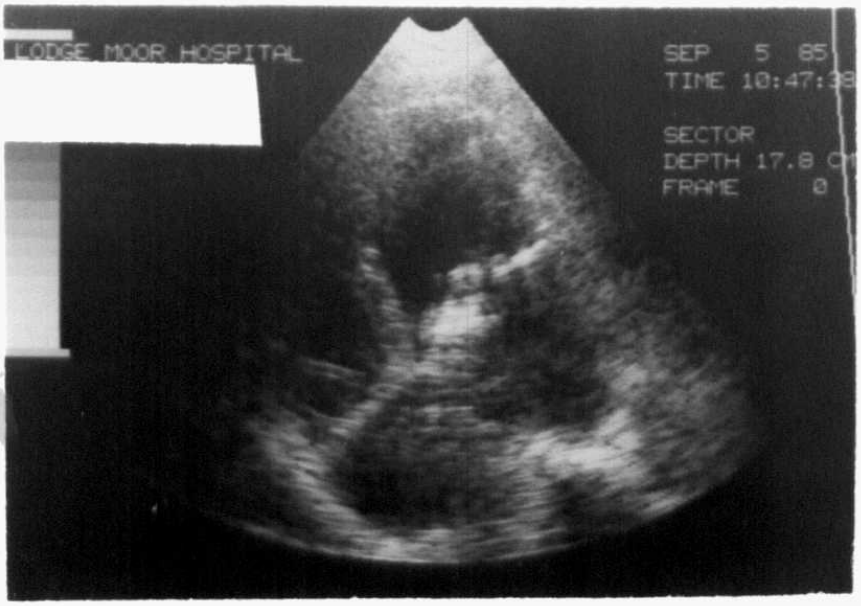
X-ray Report: The heart size is enlarged with evidence of left atrial enlargement. The appearance is suggestive of mitral valve disease. Changes in the lung fields consistent with COAD. No active lung lesion is noted.

Echocardiography:

LA:	gross dilatation
IAS:	normal
LV:	normal
MV:	thickened, calcific and tightly stenotic
AV:	not seen clearly
RA:	normal
RV:	mild dilatation
TV:	normal
IVS:	normal
Pericardium:	normal

Final Diagnosis Mitral valve disease (stenosis)
COAD.





Case No: 3.

Age: 75

Sex: Female

Clinical Presentation: Increased shortness of breath
Orthopnoea and dyspnoea at rest over the past 6 months

Past Medical History: Collapsed 9 months ago

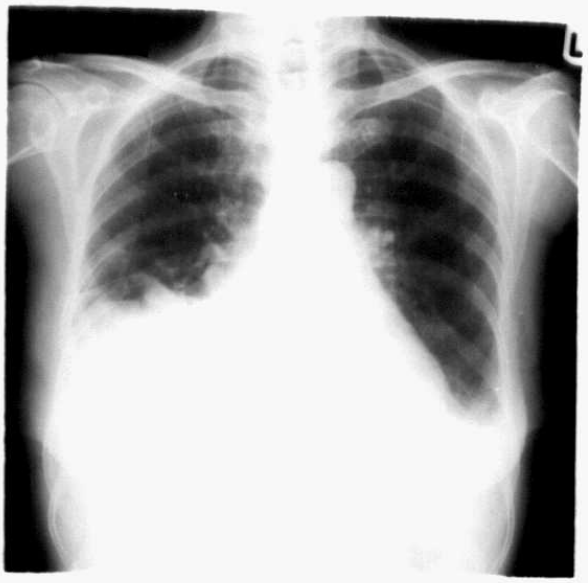
Physical Findings: Pulse 96/min.
BP - 110/60
Dullness in the right base
Inspiratory coarse crackles in the left base
Pan-systolic and mediastolic murmur at the left sternal edge
Slight ankle oedema
Enlarged liver
ECG: signs of left ventricular hypertrophy and left atrial enlargement

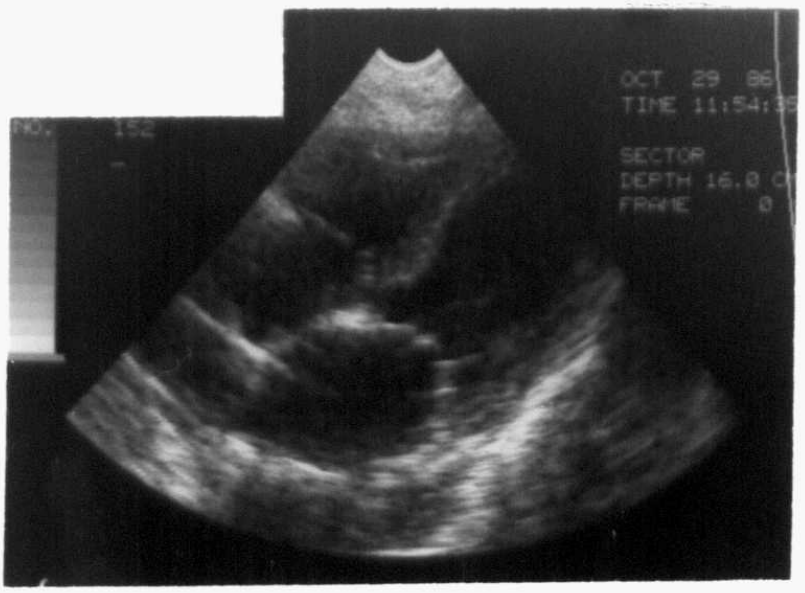
X-ray Report: Large abnormal opacity at the right base probably due to a combination of pleural fluid and lung consolidation. Small left pleural effusion. The heart is obscured by the basal changes

Echocardiography:

LA:	mild dilatation
IAS:	Normal
LV:	moderate dilatation with poor contraction
MV:	normal
AV:	thickened, calcific and stenotic
RA:	normal
RV:	normal
TV:	normal
IVS:	normal
Pericardium:	normal
Aorta:	

Final Diagnosis: Aortic Stenosis
Congestive Heart Failure





Case No: 4.
Age: 77
Sex: Female

Clinical Presentation: Shortness of breath

Past Medical History: Nil

Physical Findings: Pulse - 100/minute with irregularity
Loud pan-systolic murmur radiating to the axilla
Dullness at the right side of the chest

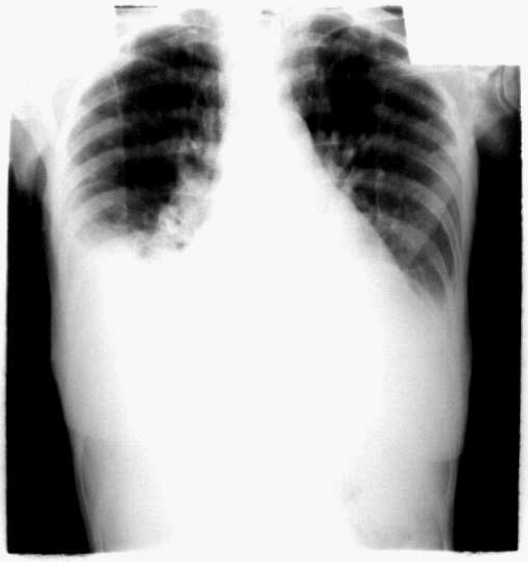
X-ray Report: The heart size is enlarged. Lungs are congested. Bilateral pleural effusion. The appearance is consistent with heart failure.

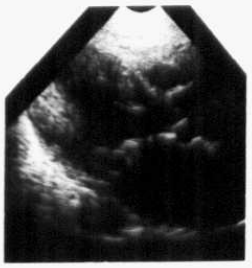
Echocardiography:

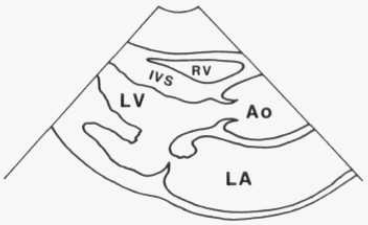
LA:	gross dilatation
IAS:	normal
LV:	moderate dilatation
MV:	rupture of the anterior papillary muscle with obvious prolapse of the anterior mitral leaflet into the left atrium
AV:	normal
RA:	normal
RV:	normal
TV:	normal
Pericardium:	normal
Aorta:	normal

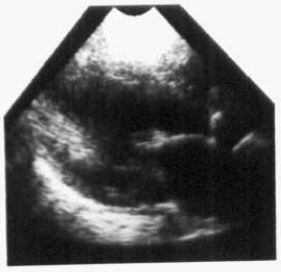
Clinical course and follow up: The patient died secondary to her heart failure

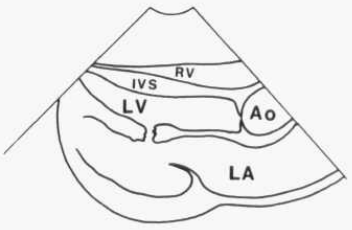
Final Diagnosis: Post Myocardial Infarction
Rupture of the Anterior Papillary Muscle











Case No: 5.
Age: 19.
Sex: Male

Clinical Presentation: Shortness of breath for two months
Chest pain (typical) for two weeks

Past Medical History: Bacterial endocarditis of one month's duration

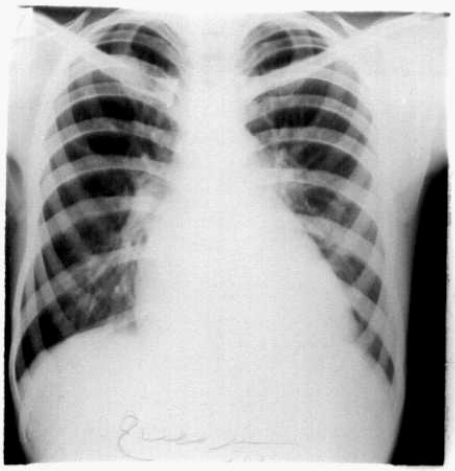
Physical Findings:
Pulse - 120/minute sinus rhythm
BP - 150/60
JVP - normal
Vesicular breathing
Apex in the 6th left intercostal space outside the mid clavicular line
Aortic pulsations
Mid-diastolic rumble murmur (Austin Flint) accentuates the 1st heart sound
ECG: left ventricular volume overload

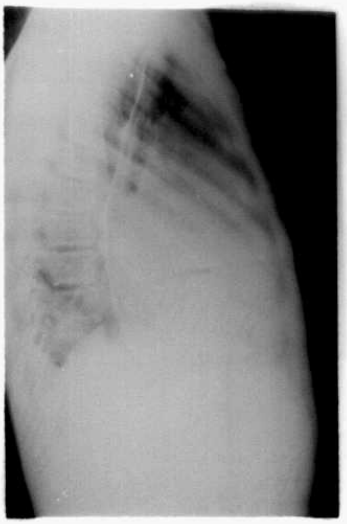
X-ray Report: The heart size is enlarged with left ventricular predominance. Both lungs are congested. No active lung lesion seen.

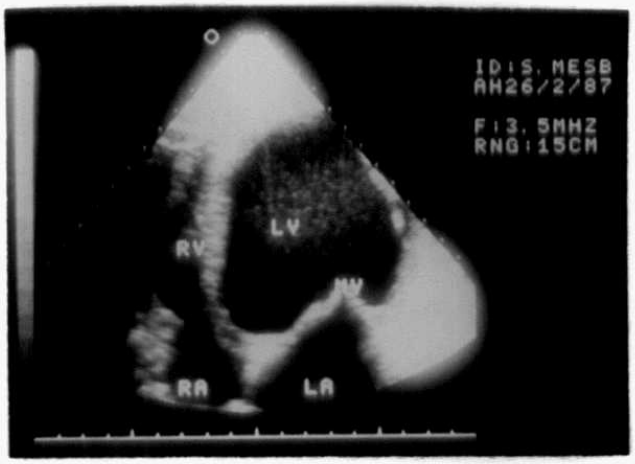
Echocardiography:

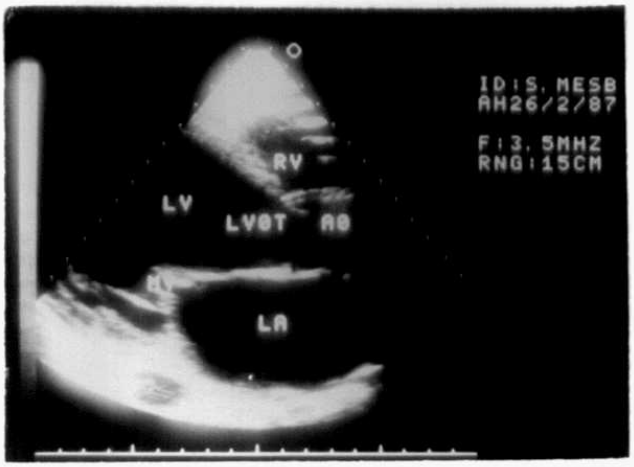
LA:	dilated
IAS:	normal
LV:	gross dilatation
MV:	thickening of the valve and fluttering of the anterior leaflet is noted. No stenosis seen
AV:	mild thickening but no stenosis
RA:	normal
RV:	normal
TV:	normal
IVS:	normal
Pericardium:	normal
Aorta:	not seen

Final Diagnosis: Aortic regurgitation secondary to Rheumatic Heart Disease









Case No: 6.
Age: 14.
Sex: Female

Clinical Presentation: Shortness of breath and awareness of heart beats (palpitations) of two years' duration

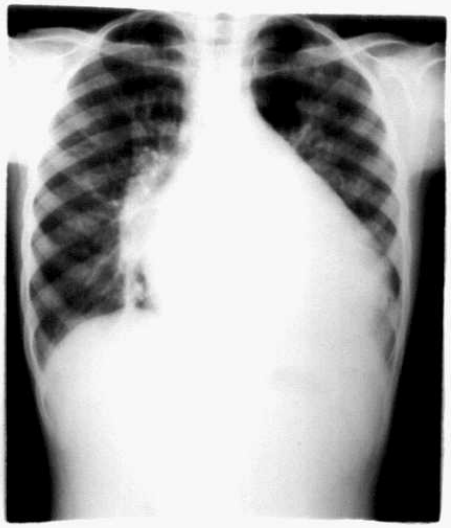
Past Medical History: Shortness of breath and palpitations for three years

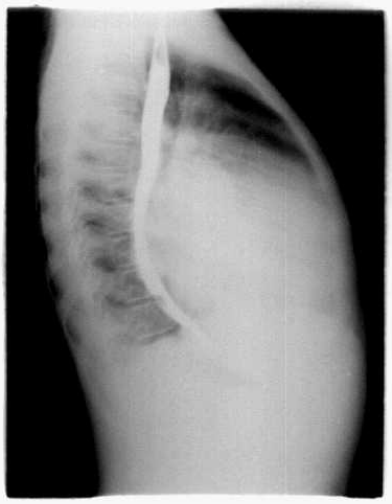
Physical Findings:
Pulse - 75/minute
BP - 130/50
JVP - normal
Normal air entry
Vesicular breathing
Heart apex in the 6th intercostal space in the anterior axillary line
Accentuated 1st heart sound with pansystolic murmur over the apex which is radiating to the axilla
Accentuated 2nd heart sound over the pulmonary area
Early diastolic murmur over the aortic area
Ejection systolic murmur over the aortic area
ECG: left ventricular overload

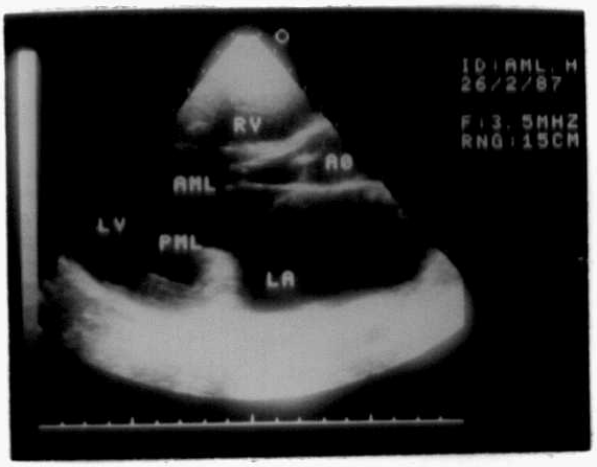
X-ray Report: The heart size is grossly enlarged with a mitral configuration. Lungs show evidence of pulmonary venous hypertension and upper lobe blood diversion. No active lung lesion is seen

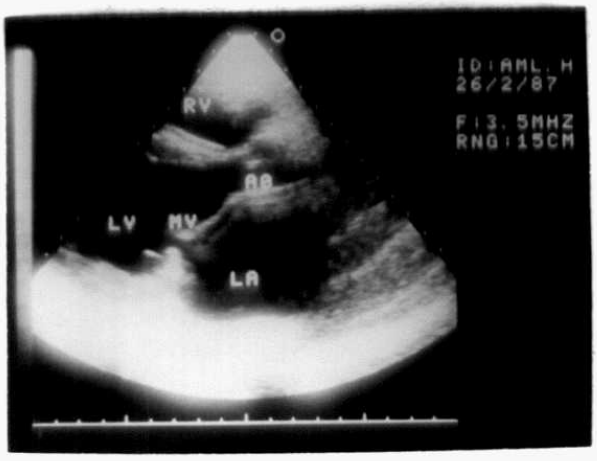
Echocardiography:
LA: gross dilatation
LV: gross dilatation
MV: thickening and calcification of posterior leaflet which is fixed. No stenosis seen
AV: normal
RA: normal
RV: normal

Final Diagnosis: Mitral Valve Disease
(Stenosis and Regurgitation)









Case No: 7

Age: 30

Sex: Male

Clinical Presentation: Shortness of breath and easy fatigueability of three months' duration

Past Medical History: Recurrent attacks of haemoptysis

Physical Findings:

Pulse - 76/minute sinus rhythm

BP - 120/70

JVP - normal

ECG - apex in the 5th left intercostal space 1cm. outside the mid clavicular line. Palpable S1 - systolic thrill over the base and neck. Accentuated S1 pan-systolic murmur over the mitral area propagated to the axilla - muffled S2 - harsh ejection systolic murmur over A1 - early diastolic murmur over the second aortic area

ECG: Biventricular enlargement and predominant left ventricle

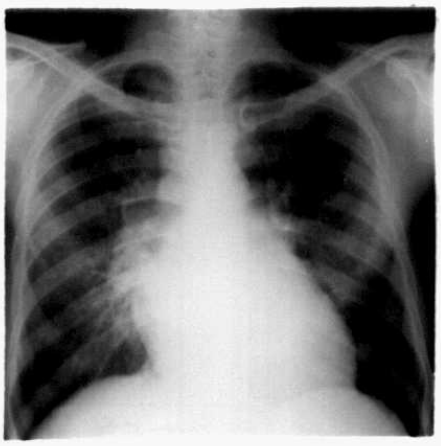
X-ray Report: The heart size is enlarged with evidence of enlarged left atrium. The lungs are congested. No focal lung lesion is seen.

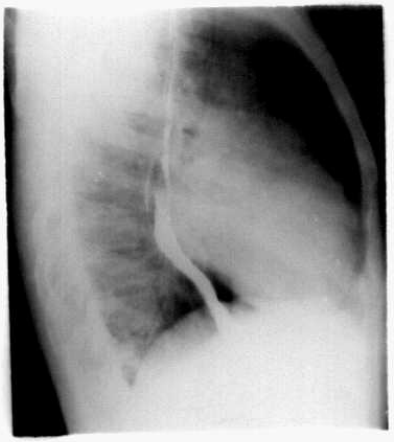
Echocardiography:

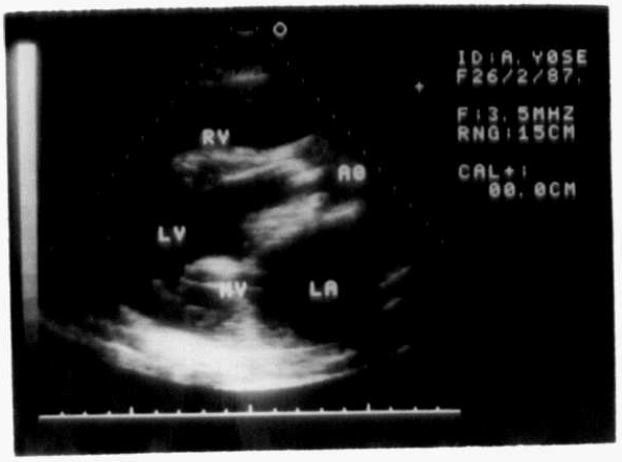
LA:	moderate dilatation
IAS:	normal
LV:	moderate dilatation
MV:	thickened and stenotic
AV:	thickened and stenotic
RA:	normal
RV:	mild dilatation
TV:	normal
Pericardium:	normal
IVS:	normal

Final Diagnosis: Mitral Stenosis

Aortic Stenosis







Case No: 8.
Age: 28
Sex:

Clinical Presentation: Shortness of breath for two years plus palpitations

Past Medical History: Rheumatic fever at the age of 9 years

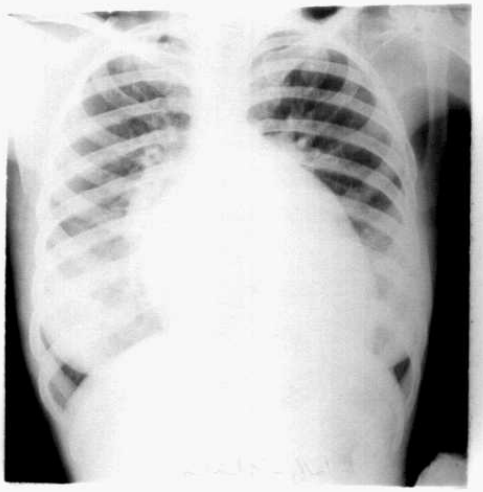
Physical Findings:
Pulse - 89/minute sinus rhythm
BP - 130/70
JVP - normal
ECG - Apex in the 6th space anterior axillary line - palpable S3
Muffled S1 - pansystolic murmur radiating to the axilla
S3 - accentuated P2
Chest - normal air entry
vesicular breathing

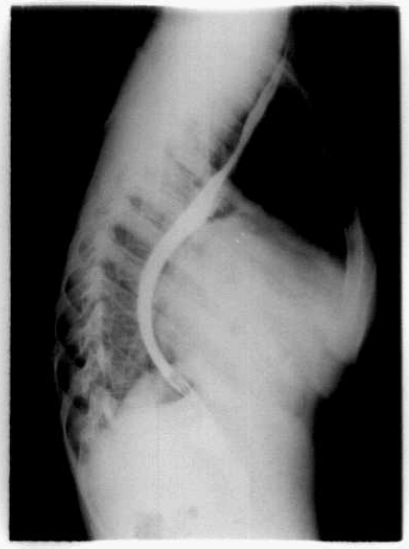
X-ray Report: The heart size is markedly enlarged and of mitral configuration. The lungs are congested with upper lobe blood diversion. No active lung lesion is seen.

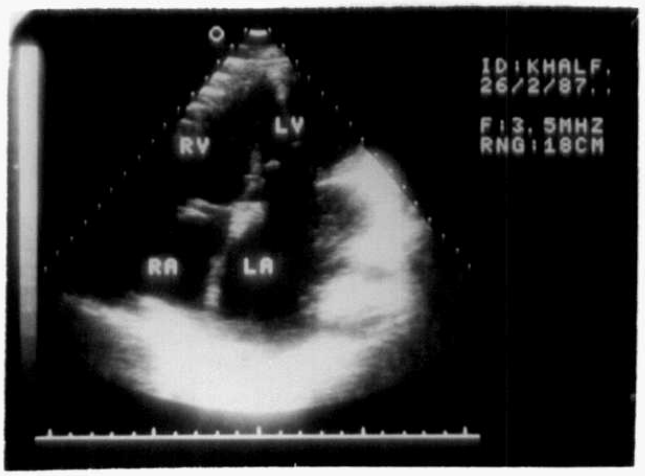
Echocardiography:

LA:	gross dilatation
IAS:	normal
LV:	gross dilatation
MV:	thickening of mitral leaflets with mild stenosis
AV:	normal
RA:	normal
RV:	normal
TV:	normal
IVS:	normal
Pericardium:	normal

Final Diagnosis: Mitral Valve Disease (mitral regurgitation) Mild Stenosis







Case No: 9

Age: 20

Sex: Female

Clinical Presentation: Shortness of breath for 3 years plus easy fatigueability
Palpitations for 2 years

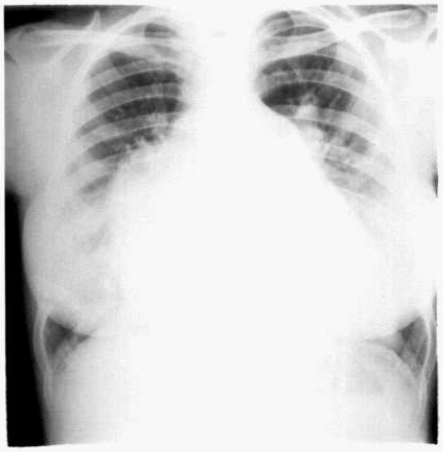
Past Medical History: Rheumatic fever
Recurrent attacks of tonsillitis
1 month's history of left capsular hemiplegia

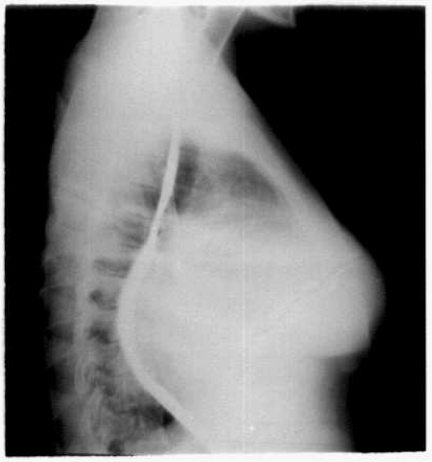
Physical Findings: Pulse - 140/min. irregular. Atrial fibrillation
BP - 130/50
JVP - raised 6 cm.
Lower limb oedema
Apex 6th left intercostal space in the anterior axillary line. Epigastric rub from right ventricle. Right parasternal heave. Accentuated S1 - short systolic murmur propagated to the axilla. Pansystolic murmur increased by inspiration over T area with accentuated P2 early diastolic murmur over A2.
Chest - Normal air entry with vesicular breathing.

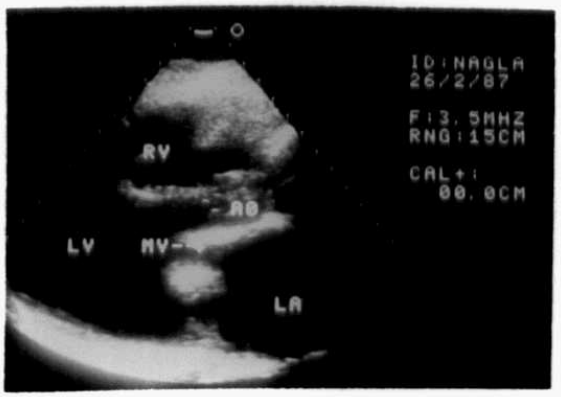
X-ray Report: The heart size is markedly enlarged and is of mitral configuration with marked prominent pulmonary conus. Lungs are congested with upper lobe blood diversion. No active lung lesion.

Echocardiography:
LA: gross dilatation
LV: gross dilatation
MV: marked thickening; calcified and tight stenosis
AOV: normal
RA: mild dilatation
RV: gross dilatation

Final Diagnosis: Mitral Valve Disease
Tight Mitral Stenosis







Case No: 10

Age: 75

Sex: Male

Clinical Presentation: Increased shortness of breath over the last three months
Breathless on mild exercise
Cough with white sputum

Past Medical History: Scarlet fever twelve years ago
Diabetes Mellitus eight years ago
Breathing problems two years ago

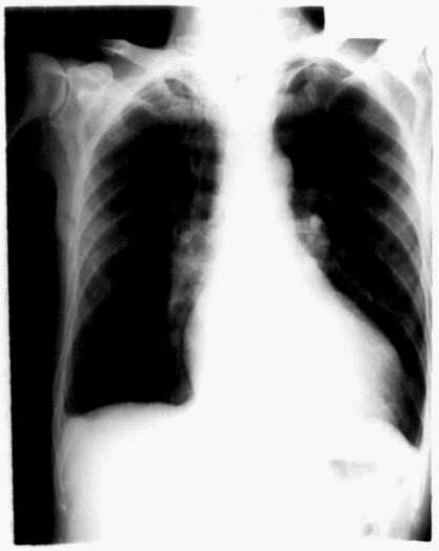
Physical Findings: Pulse - 80/min. regular
B.P. - 100/60
JVP - normal
Ankle Oedema
Apex beat tapping and displaced to the anterior axillary line
Pan systolic murmur
Dyspnoeic on moving around
Vesicular breathing
Prolonged expiration

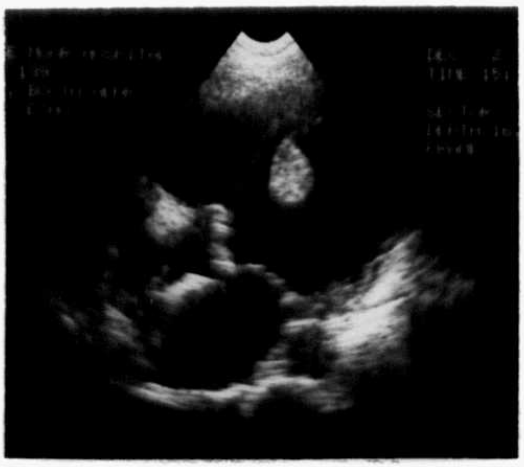
X-ray Report: The heart size is slightly enlarged. Changes in the lung fields compatible with early pulmonary oedema secondary to left heart failure. No active lung lesion seen.

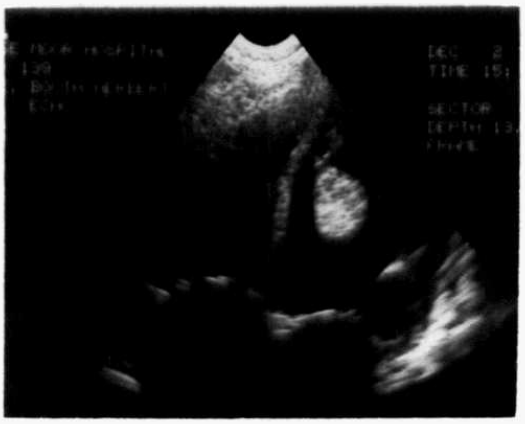
Echocardiography:

LA:	mild dilatation
LV:	grossly dilated and it contracts poorly. A filling defect is seen arising from the apex. This is most likely to represent an old mural clot with bright echoes within the clot. (Calcification).
MV:	mild stenosis
AV:	thickened with mild stenosis
RA:	mild dilatation
RV:	mild dilatation
Pericardium:	normal

Final Diagnosis: Mild mitral and aortic stenosis
Myocardial infarction
Left ventricular old mural clot







Case No: 11

Age: 72

Sex: Female

Clinical Presentation: Sudden central chest pain radiating to both arms, neck and back
Cough with white sputum and ankle swelling

Past Medical History: Rheumatic fever aged 16
Cholecystectomy aged 30
Ischaemic Heart Disease plus angina aged 40

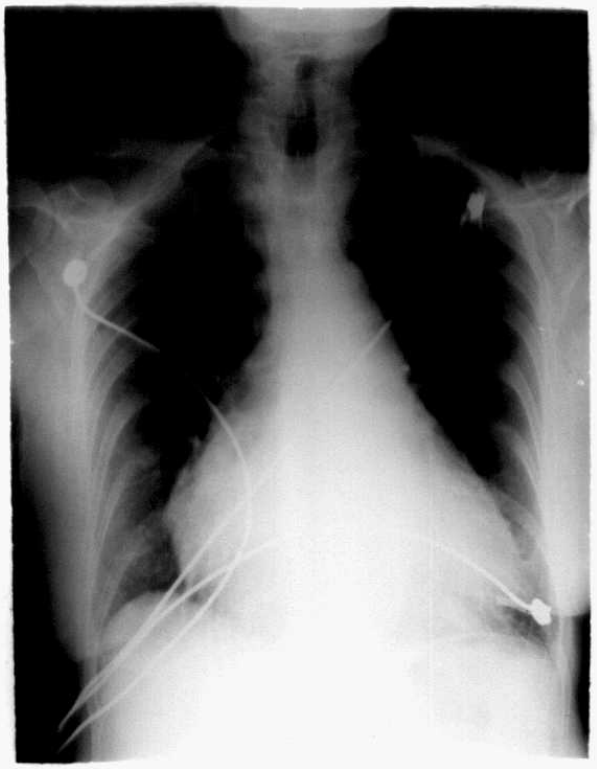
Physical Findings: Pulse - 70/minute irregular
BP - 110/70
JVP - raised 6cm.
Apex beat heaving - 110/minute
Heart sounds I, II and III
Systolic murmur at 2nd aortic area
Diastolic murmur at apex
Ankle oedema (both)
Enlarged, tender liver
Vesicular breathing with lowered air entry
ECG - changes of atrial fibrillation and changes of ischaemia with extensive anterior myocardial infarction

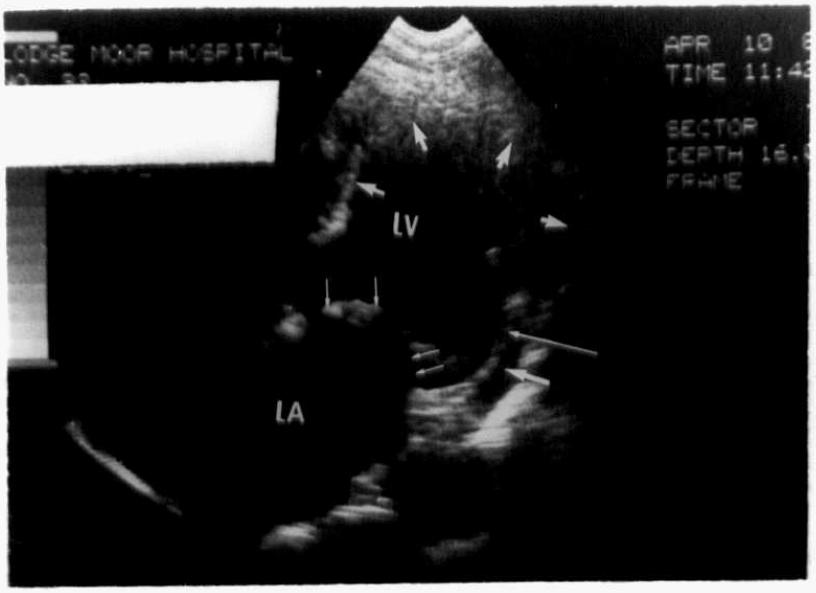
X-ray Report: The heart is enlarged. No active focal lung lesion seen

Echocardiography:

LA:	moderately dilated
LV:	gross dilatation with evidence of moderate sized aneurysm involving its apex
MV:	marked thickening with moderate stenosis & calcification
AV:	calcified but no stenosis
RA:	moderate dilatation
RV:	normal
Pericardium:	a small amount of pericardial effusion is noted

Final Diagnosis: Mitral Valve Disease
Myocardial Infarction
Left ventricular aneurysm
Small Pericardial Effusion





Case No: 12.

Age: 61

Sex: Male

Clinical Presentation: Increased shortness of breath over several weeks

Past Medical History: Chronic obstructive airways disease

Physical Findings:
Pulse - 100/minute regular
BP - 110/80
JVP - normal
Heart sounds I and II + 0
Dyspnoeic and cyanosed at rest
Poor air entry throughout both lung fields
No evidence of heart failure

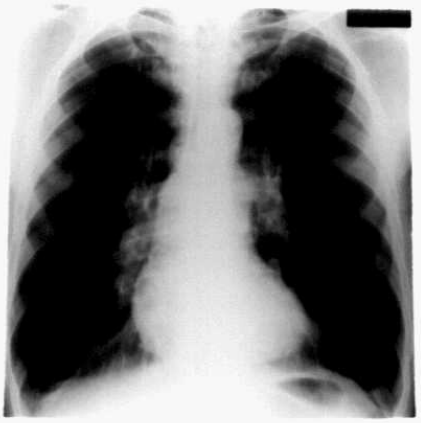
X-ray Report:
The heart size is not enlarged
Evidence of chronic obstructive airways disease is seen. No active lung lesion or evidence of heart failure

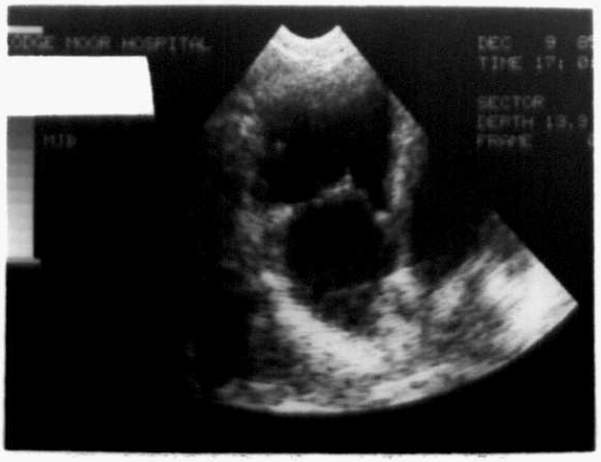
Echocardiography:

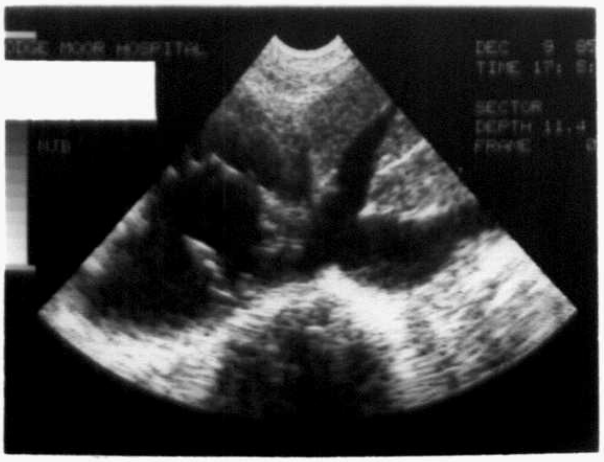
LA:	mild dilatation
LV:	moderate dilatation
MV:	normal
AV:	normal
Pericardium:	normal
AO:	normal
RA:	moderate dilatation Membrane-like filling defect seen in the right atrium which showed undulating membrane. This could represent fibrin clot or prominent and redundant Eustachian valve
RV:	moderate dilatation

Final Diagnosis: Chronic Obstructive Airways Disease

Cor Pulmonale







Case No: 13.
Age: 75
Sex: Male

Clinical Presentation:
Malaise and loss of appetite
Loss of weight
Cough with white sputum
Very slight shortness of breath on exertion
Swollen ankles

Past Medical History:
Bronchitis for 20 - 30 years
Inguinal hernia aged 56

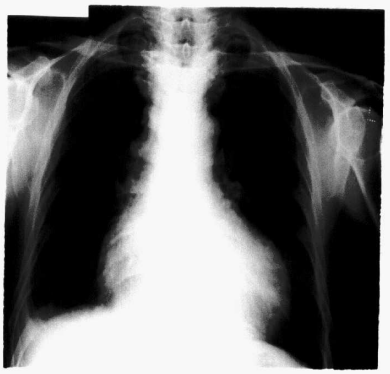
Physical Findings:
Pulse - 90/minute regular
BP - 110/60
JVP - raised 6cm.
Heart sounds I and II + O - loud murmur
Pericardial rub over LSE and apex during systole and diastole
Bilateral ankle oedema (pitting)
Peripheral pulse
Liver 2cm. below the costal margin
Poor air entry with lowered percussion note at the right base
Fine basal creps

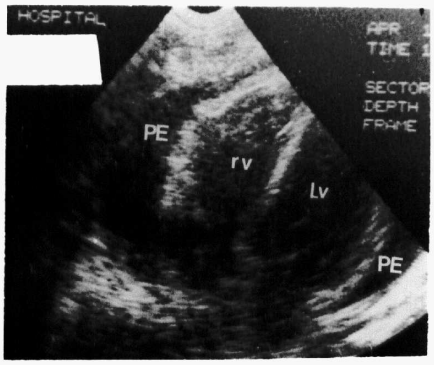
X-ray Report:
The heart size is enlarged. A small area of consolidation is noted at the right base with a small amount of right pleural effusion. No obvious active lung lesion seen.

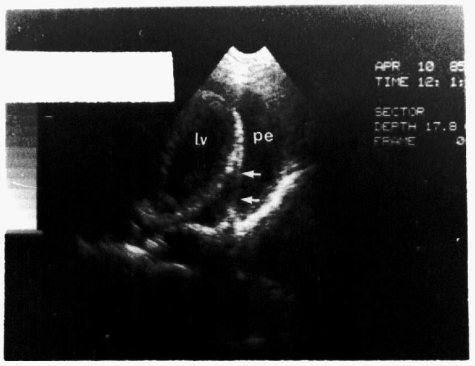
Echocardiography:

LA:	normal
LV:	normal
MV:	normal
AV:	normal
AO:	normal
TV:	normal
Pericardium:	A large pericardial effusion is noted. Fibrin is seen in the pericardial sac

Final Diagnosis:
Pericardial Effusion
? Post-viral Infection







Case No: 14

Age: 78

Sex: Male

Clinical Presentation: Shortness of breath with chronic sputum
Paroxysmal nocturnal dyspnoea
Orthopnoea
Swelling of feet

Past Medical History: Perforated peptic ulcer aged 53
Chronic Bronchitis

Physical Findings: Pulse - 64/minute regular
BP - 140/80
JVP - normal
Mild oedema of feet
Mid-diastolic murmur at the apex
Poor air entry to both lungs
Abdomen showed gross ascites with enlarged liver. Ascitic fluid was highly suggestive of malignancy and evidence of adenocarcinoma was found. Carcinoma of the pancreas is thought to be the most likely cause

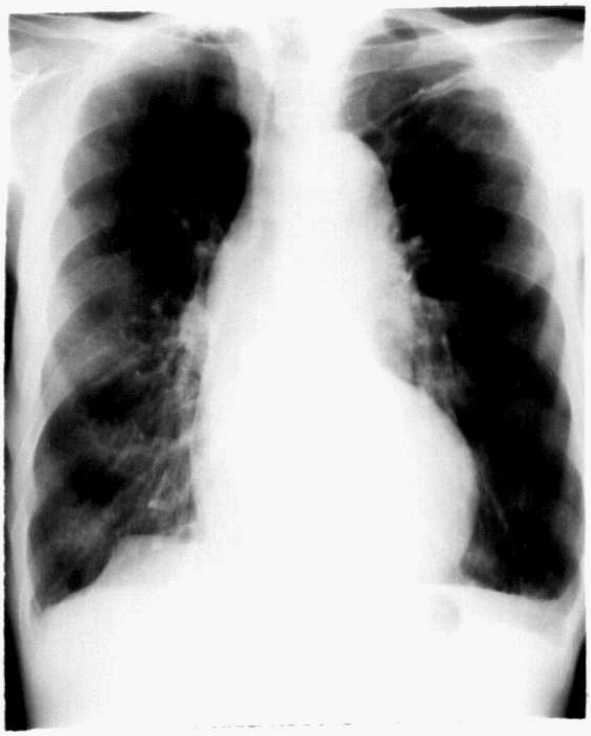
X-ray Report: The heart is not enlarged in its transverse diameter. There is an unfolded, dilated aorta. No active lung lesion. Changes of chronic obstructive airways disease noted.

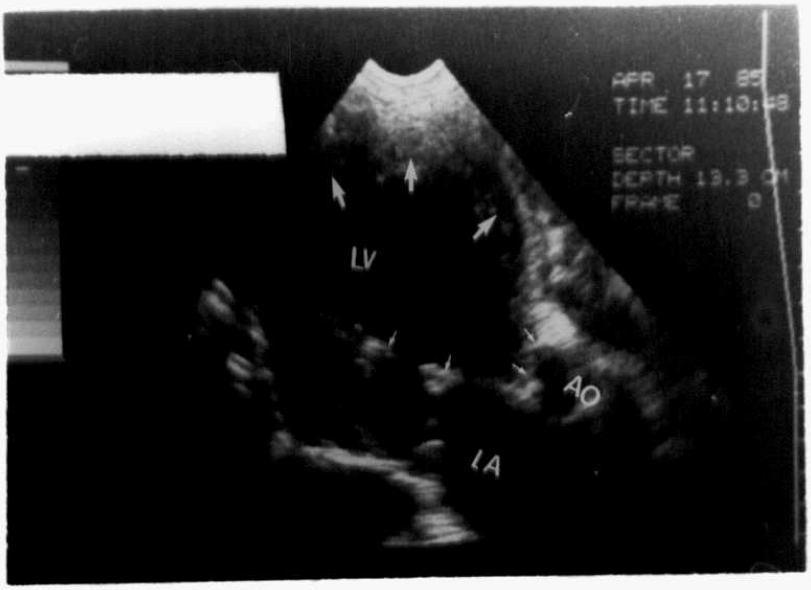
Abdominal ultrasound showed ascites, a mass lesion in the pancreas and metastatic deposits in the liver.

Echocardiography:

LA:	mild dilatation
LV:	gross dilatation with poor contraction and evidence of aneurysm with mural clot near the apex
MV:	mild thickening
AV:	mild thickening
Right side:	normal
TV:	normal
AO:	normal valve. Calcification of the aortic wall
Pericardium:	normal

Final Diagnosis: Malignant Ascites
Left Ventricular Aneurysm with Mural Clot





Case No: 15
Age: 61
Sex: Male

Clinical Presentation:
Mild shortness of breath
Mild Orthopnoea
Cough with sputum
Recurrent bronchitis and chest infection

Past Medical History:
Myocardial infarction aged 56
Heart failure (due to mitral incompetence)
Repair of inguinal hernia

Physical Findings:
No signs of heart failure
Pulse - 80/minute
BP - 130/80
JVP - normal
Left parasternal heave - apex beat forceful and at the 6th intercostal space in the anterior axillary line. Heart sounds I & II plus a loud pansystolic murmur at the apex radiating to the left axilla and all over the pericardium and the left carotid artery.

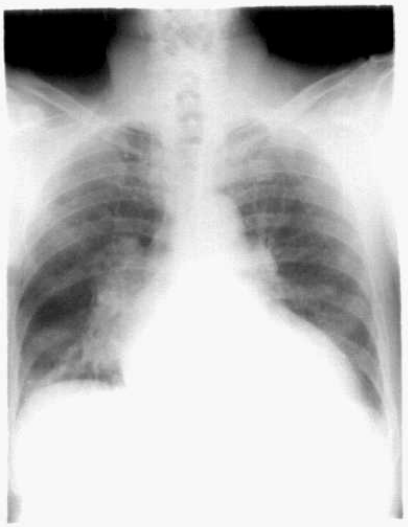
At bronchoscopy and biopsy no malignant cells were found.

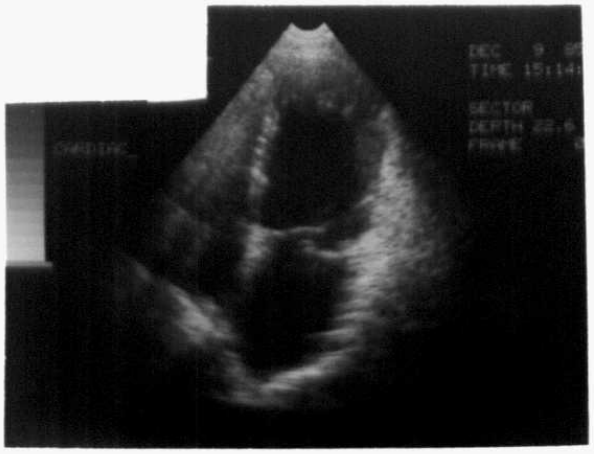
X-ray Report:
The heart size is enlarged. Shadowing is noted in the right lower zone ? nature

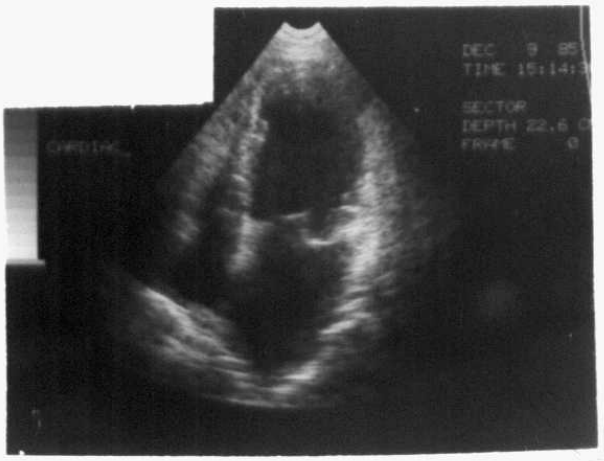
Echocardiography:

LA:	gross dilatation
MV:	prolapse of the post-mitral leaflet into the left atrium is seen with bowing of the mitral post leaflet
LV:	Moderately dilated
AV:	normal
RA:	normal
RV:	normal
TV:	normal
Pericardium:	normal
AO:	normal

Final Diagnosis:
Mitral Valve Prolapse

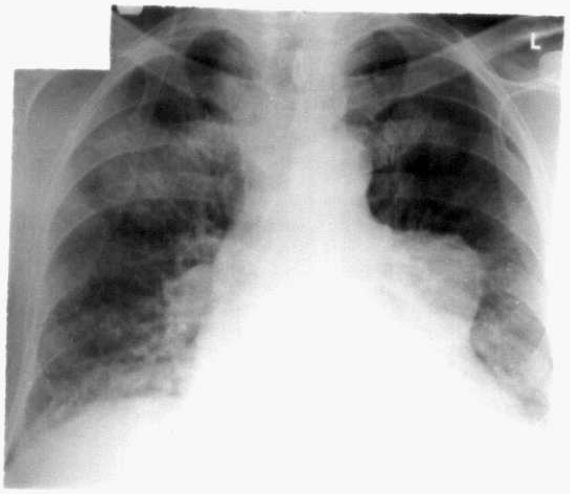


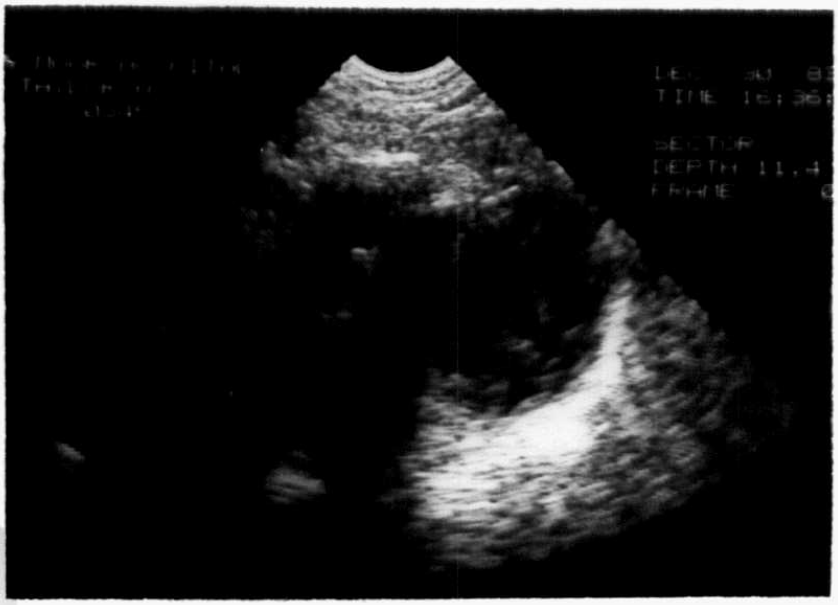




Case No: 16.
Age: 82
Sex: Male

Clinical Presentation:	Shortness of breath
Past Medical History:	Schizophrenia
Physical Findings:	Pulse - 96/minute
	BP - 120/75
	Dyspnoea
	Expiratory wheeze
	Fine basal crepitations
	Ejection systolic murmur (aortic stenosis)
X-ray Report:	Chest PA and lateral -
	Heart size grossly enlarged. Lungs congested with evidence of pulmonary oedema. Appearances are compatible with heart failure. There is an opacity merging with the left heart border.
Echocardiography:	LA: moderate dilatation
	IAS: normal
	LV: gross dilatation
	MV: marked thickening with evidence of stenosis
	AV: not seen
	RA:) RV:) TV:) no signs of dilatation right side
	IVS: normal
	Pericardium: A solid lesion is seen attached to outer wall of left ventricle
	Thoracic AO: not seen
Follow Up:	The patient died in spite of anti-heart failure treatment
Post Mortem:	The heart was enlarged
Left Atrium:	Slightly dilated
Left Ventricle:	Hypertrophy and dilatation
MV:	Mild stenosis
AV:	Tight stenosis
RA: RV: TV: PV: AO:	Normal
	Left pleural effusion
Pericardium:	Large tumour (8cm) attached to outer surface of the pericardium over left outer surface of the pericardium over the left ventricle
Histology:	Thymoma
Final Diagnosis:	Mitral Stenosis. Heart Failure. Thymoma.





RESULTS

THE RADIOLOGICAL FINDINGS

TABLE 11

Chest X-ray Findings	Number of Cases
Normal chest x-ray	7
Enlarged heart and normal lung	8
Enlarged heart with pulmonary abnormality	40
Normal heart with pulmonary abnormality	4
The heart size could not be assessed due to a pleural effusion and adjacent lung consolidation	1

The different types of pulmonary abnormalities were as follows -

Pulmonary abnormalities with enlargement of the heart were found in 40 patients:-

TABLE 12

Pulmonary Abnormality	Number of cases
Evidence of heart failure	24
Evidence of heart failure with pulmonary infection	3
Evidence of chronic obstructive airways disease	6
Evidence of chronic obstructive airways disease with pulmonary infection	2
Evidence of chronic obstructive airways disease with heart failure	2
Evidence of pneumoconiosis	1
Lower lung opacity (infection or infarction)	1
Bronchogenic carcinoma	1

Pulmonary abnormalities were found without enlargement of the heart in 4 patients:-

TABLE 13

Pulmonary Abnormality	Number of cases
Evidence of heart failure with pulmonary infection	1
Evidence of chronic obstructive airways disease	1
Evidence of metastatic pulmonary calcification in a patient with chronic renal failure	1
Evidence of pulmonary tuberculosis	1

ECHOCARDIOGRAPHIC FINDINGS

VALVULAR HEART DISEASE

Total number of cases:- 38 (63.3%)

MITRAL VALVE DISEASE 16 (42%)

TABLE 14

Type of Lesion	Number of cases
Mitral Stenosis	11
Mitral Prolapse	3
Mitral calcification (without obvious stenosis)	1
Prosthetic (mitral) Valve	1

AORTIC VALVE DISEASE

Total number of cases:- 7 (18%)

TABLE 15

Type of Lesion	Number of cases
Aortic Stenosis	5
Aortic Regurgitation	1
Aortic Calcification (without significant stenosis)	1

MITRAL AND AORTIC VALVE DISEASE

Total number of cases:- 15 (40%)

TABLE 16

Type of Lesion	Number of cases
Mitral stenosis and Aortic stenosis	6
Mitral stenosis and Aortic calcification plus thickening without obvious stenosis	4
Mitral prolapse and Aortic stenosis	1
Mitral calcification and Aortic stenosis	1
Mitral and Aortic calcification plus thickening without obvious stenosis	3

PERICARDIAL (EFFUSION)

Total number of cases:- 7 (11.6%)

TABLE 17

Type of Lesion	Number of cases
Small pericardial effusion with chamber dilatation	6
Large pericardial effusion without chamber dilatation	1

The pericardial effusion was detected in all these cases by echocardiographic examination, whereas the chest x-ray showed enlargement of the heart without specific features to suggest a pericardial effusion.

PERICARDIAL MASS

One case diagnosed as a pericardial mass by echocardiography was proved at post-mortem to be a thymoma.

DILATATION OF THE CARDIAC CHAMBERS

Total number of cases:- 58 (97%)

TABLE 18

The Dilated Chamber		Number of cases	Degree of Dilatation		
			Mild	Moderate	Gross
Left Atrium	77.5%	45	15	15	15
Left Ventricle	84.5%	49	9	17	23
Right Atrium	34%	20	10	5	5
Right Ventricle	41%	24	11	6	7

CARDIAC ANEURYSM

All cases of aneurysm have been found in the left ventricle.

Total number of cases:-

TABLE 19

Details	Number of cases
Left Ventricular Aneurysm with a Mural Clot	4
Left Ventricular Aneurysm without a Mural Clot	8

Poor contraction of the left ventricle noted in another nine cases without evidence of aneurysm

FILLING DEFECT WITHIN THE HEART

Total number of cases:- 7 (11.6%)

TABLE 20

Details	Number of cases
A clot within an aneurysm of the left ventricle	4
A clot within the right ventricle	1
A clot in the left ventricle without evidence of aneurysm	2

LEFT VENTRICULAR HYPERTROPHY

Total number of cases:- 5 (8.3%)

TABLE 21

Details	Number of cases
Hypertrophy of the left ventricle with dilatation	4
Hypertrophy of the left ventricle without dilatation	1

COMPARISON BETWEEN RADIOGRAPHIC AND ECHOCARDIOGRAPHIC FINDINGS

A. Abnormalities have been detected both in the chest x-ray and in echocardiographic examinations in 50 patients. (83%).

(1) In 12 cases (24%) both chest x-ray and echocardiography demonstrate evidence of left atrial enlargement.

(2) In 5 cases (10%) both chest x-ray and echocardiography demonstrate evidence of left ventricular enlargement.

(3) In 32 cases (64%) the chest x-ray shows enlargement of the heart without specific information as to which chamber of the heart was dilated. The echocardiographic examination of these patients shows the specific abnormality. (Table 22).

(a) The right side of the heart was dilated in 5 cases

(b) The LA was dilated alone in 1 case and in 16 cases both LA and LV were dilated.

(c) Both sides (the 4 chambers) were dilated in 9 cases.

(d) Pericardial effusion in 1 case without chamber dilatation.

Details of the echocardiographic findings in these 32 cases are as follows:-

The right side of the heart was dilated in 5 cases

dilatation of the right atrium and right ventricle 4

dilatation of the right atrium and right ventricle with mural clot in the right ventricle 1

The left side of the heart was dilated in 17 cases

Table 22

The Dilated Chamber	Mitral valve	Aortic valve	The peri-cardium	No.of cases
LA	stenosis			1
LA and LV with poor contraction of LV				5
LA and LV with poor contraction of LV and mural clot				2
LV and LA with poor contraction of LV			effusion	1
LA and LV with hypertrophy of LV				1
- LV hypertrophy			effusion	1
- LV hypertrophy	stenosis	stenosis		1
LA and LV	stenosis	calcifi-cation		1
LA and LV	stenosis	stenosis		1
LA and LV			mass	1
LA and LV	prolapse			2

DILATATION OF BOTH SIDES OF THE HEART (4 CHAMBERS) IN 9 CASES

TABLE 23

The dilated chamber	Mitral valve	Aortic valve	The peri-cardium	No. of cases
4 chambers				2
4 chambers		calcific		1
4 chambers		stenosis		1
4 chambers	stenosis	stenosis		2
4 chambers	stenosis			1
4 chambers	stenosis	calcific	effusion	2

(B) In one case the heart size could not be assessed due to pleural effusion and consolidation of the adjacent lung while the echocardiographic examination showed left atrial and left ventricular enlargement.

(C) Normal appearance of the heart on the chest x-ray and the abnormality was detected by echocardiographic examination.

The total number of cases is 9 (15.5%)

TABLE 24

The Echocardiographic Findings	No. of cases
Left Ventricular Aneurysm	3
Left Ventricular Aneurysm and a Mural Clot	1
A Mural Clot without Aneurysm of the Left Ventricle	1
Left Ventricular Dilatation	1
Mitral Stenosis	1
Aortic Stenosis	1
Right Ventricular Dilatation	1

THE FINAL DIAGNOSIS

50 cases from Lodge Moor Hospital, Sheffield, England

TABLE 25

The Final Diagnosis	Number of cases
Normal (variant)	1
Ischaemic Heart Disease	11
Valvular Heart Disease	12
Ischaemic and Valvular Heart Disease	8
Ischaemic and Valvular Heart Disease with Pericardial Effusion	2
Ischaemic and Valvular Heart Disease with Pericardial Mass (Thymoma)	1
Pericardial Effusion without Chamber Dilatation (? Post Viral Infection)	1
Ischaemic Heart Disease with Pericardial Effusion	4
Cor Pulmonale	10

10 cases from El Hussin Hospital, Cairo, Egypt

TABLE 26

The Final Diagnosis	Number of cases
Mitral Stenosis	5
Mitral Regurgitation	1
Aortic Stenosis	1
Aortic Regurgitation	1
Mitral Stenosis and Aortic Stenosis	2

DISCUSSION

Proper and careful physical examination of the patient will frequently lead to the correct diagnosis. The evaluation of adult patients with suspected cardiac disease usually begins with the standard postero-anterior and left lateral radiographs of the chest. The cardio-thoracic ratio is a fairly reliable guide in assessing the heart size. With advancing age dilatation and tortuosity of the thoracic aorta, as well as the brachiocephalic vessels, are a frequent finding.

A normal chest radiograph does not exclude abnormality of the heart or dilatation of any of its chambers.

In this study the chest x-rays were reported as "normal appearance of the heart" in nine cases (15%). However abnormalities have been detexted by echocardiographic examination of these patients. Four of them had left ventricular aneurysm, one patient had a mural clot, as in case no. 14, two patients had ventricular dilatation (one patient with left ventricular dilatation and one with right ventricular dilatation) and two patients had valvular stenosis (one mitral stenosis and one aortic stenosis).

In this study the chest x-rays showed enlargement of the heart in 49 patients. In 12 of these patients there was the suggestion of left atrial enlargement and that had been proved by echocardiographic examination. Enlargement of other cardiac chambers had also been detected by echocardiography in these 12 cases as well as valvular thickening, calcification or stenosis. In 5 cases (10%) left ventricular enlargement was noted in the chest x-ray. However, echocardiographic examination demonstrated gross enlargement of the

left ventricle and evidence of left ventricular aneurysm. Other abnormalities were also detected in the aortic and mitral valves, and in the other cardiac chambers.

In the remaining 32 cases (65%) the chest x-ray showed enlargement of the heart without specific configuration to suggest which chamber of the heart had been enlarged.

In the majority of adult patients with acquired heart disease all chambers are likely to be involved and it is not possible with plain radiography to determine the contribution of each chamber, other than the left atrium. The heart, with dilatation of all its chambers, takes on a globular shape. In these patients the assessment of the enlarged chambers can be determined by cross-sectional echocardiography.

In this study the enlargement of cardiac chambers and the extent is shown by echocardiography in 58 patients (97%); left atrial enlargement in 45 patients; left ventricular enlargement in 49 patients; right atrium enlargement in 20 patients; right ventricular enlargement in 24 patients.

The plain chest radiograph is particularly limited in the assessment of cardiac enlargement in patients with chronic obstructive airways disease and emphysema. In one of the reported cases in this study (case no. 12) the heart size was normal on the chest radiograph and the lungs were emphysematous. Echocardiography on the other hand demonstrated moderate dilatation of all the cardiac chambers, especially in the right side. The advantage of echocardiography in

such cases is obvious.

The radiographic assessment of heart disease requires careful analysis of the pulmonary circulation. There are no valves in the pulmonary veins, so that left atrial pressure is directly reflected in the pulmonary capillaries without intervention of valves. The air, with its ideal radiographic properties, renders the lung the only organ in the entire body whose blood supply can be visualized and assessed without the assistance of added contrast medium. The radiographic contrast between the intrapulmonary blood vessels and the surrounding intra-alveolar air provides a tremendous amount of information concerning the blood supply to and from the lungs and the amount of extra-vascular lung water. (Grainger, 1985).

In this study we have found that the changes and the abnormalities of the pulmonary circulation are of great help in the assessment of patients with heart disorders, especially in detecting early changes of left ventricular failure.

The following radiological patterns can be recognised in the chest radiograph:-

a) Normal pulmonary circulation
b) Pulmonary arterial plethora
c) Pulmonary arterial hypertension
d) Pulmonary oligaemia
e) Pulmonary venous congestion and pulmonary oedema

The latter condition is readily recognisable by visible costophrenic (Kerley) septal lines, basal alveolar and interstitial oedema and

by increased vascular perfusion to the upper lung zones. In left heart failure the basal pulmonary oedema impairs gas exchange because the alveoli in the lower parts of the lung contain oedema fluid and the alveolar walls are oedematous. It makes good sense for the body to divert pulmonary blood flow away from the compromised lower zones to the non-oedematous alveoli and alveolar walls which are available in the upper lung zones. (Grainger, 1985).

In this study chest x-rays of 30 patients (50%) showed evidence of left heart failure. In nine cases upper lobe blood diversion was the main abnormality and they were reported as early left heart failure. Echocardiography is very limited in providing evidence of early left heart failure in comparison to the chest radiograph.

Non-cardiogenic abnormalities were detected in 16 patients in this study, which includes chronic obstructive airways disease, pneumoconiosis, pulmonary infarction, lung infection and bronchogenic carcinoma.

In acquired valvular heart disease chest x-ray will demonstrate enlargement of the heart. General stenosis of a valve will produce a dilatation of the chambers proximal to the stenosis. Stenosis of the aortic or pulmonary valve will produce in addition localized post-stenotic dilatation of the associated bloodvvessel. On the other hand insufficiency of a valve usually results in enlargement of the two chambers, which are separated by the incompetent valve.

The main cardinal radiological feature of rheumatic mitral valve disease is selective left atrial enlargement, as shown on the PA and

left lateral chest x-ray. Other valvular lesions cannot be so accurately assessed radiologically.

Calcification or vegetation cannot be excluded by the chest radiograph. However heavy calcification is often detected on the x-ray film. On the other hand cross-sectional echocardiography allows complete visualization of all intra-cardiac structures including the valves.

Mitral valve stenosis is readily diagnosed by echocardiography in 11 patients in this study using the long axis parasternal and apical 4 chamber views. Evidence of left atrial dilatation was seen on chest x-ray in 10 of these patients. Thickening and immobility of the mitral leaflets, bowing of the anterior leaflet in systole due to fusion of the tips and at the commissures, and poor opening of the valve were detected in these patients, case no. (2). Calcification of the mitral valve leaflets was common in these patients and was not detected in any of these cases on the chest x-ray.

Mitral valve prolapse was diagnosed in 3 patients in this study by detecting the superior arching of the mitral leaflets above the atrio-ventricular ring. The apical 4 chamber view provided better definition of the mitral annulus and the posterior mitral leaflet, and is a good view for the diagnosis of mitral valve prolapse, case no. (15 and 4).

Aortic valve abnormalities were detected in 7 patients in this study. Five of these patients had thickening of the aortic cusps with systolic doming and a narrow orifice. They were diagnosed as aortic valve

stenosis. Case no. (3). One case showed calcification of the aortic valve cusps without significant stenosis. The other patient presented with thickening of the aortic valve and fluttering of the anterior mitral valve leaflet. It was reported as aortic regurgitation.

Combined mitral and aortic valve disease have been detected by echocardiography in 15 cases in this study; mitral stenosis and aortic stenosis in 6 patients, (case no. 1); stenosis with aortic calcification in 4 patients; mitral and aortic valve calcification with thickening of the leaflets without obvious stenosis in 3 patients and one case with mitral valve prolapse and aortic stenosis. One patient had stenosis of the aortic valve with calcification of the mitral valve leaflets. Regarding these cases of valvular heart disease all the abnormalities had been detected only by echocardiographic examination and the chest x-ray could not detect any of these valvular lesions except in 12 cases with mitral valve disease where evidende of left atrial dilatation was shown in the chest radiograph. Case no. (6). The sensitivity of echocardiography in the assessment of valvular heart disease is obvious. Two dimensional echocardiography without Doppler facilities is not sensitive in the assessment of valve regurgitation and mild valvular stenosis can be overlooked as well as small vegetations. It is also difficult to differentiate vegetation from gross thickening of valve leaflets.

In ischaemic heart disease cross-sectional echocardiography can provide reliable information about abnormalities of wall motion, contraction of the myocardium and myocardial scarring and can detect ventricular aneurysm.

In this study left ventricular aneurysm has been detected in 12 patients and 4 of them had a mural clot within the aneurysm, Case no. (10). The characteristic features of the aneurysm were seen in these cases, which includes the presence of an edge at the orifice of the aneurysm and paradoxical movement. The chest x-ray of these 12 patients did not show direct evidence of aneurysm and in 4 patients the heart size was normal. Poor contraction of the left ventricle had been noticed in 9 patients without other evidence of aneurysm.

A study was carried out by "Peart et al" on patients with ischaemic heart disease which demonstrated that segmental visualization is variable, seen best in the parasternal long axis and apical 4 chamber views and worst in the substernal view.

In this study we found that the apical 4 chamber view is very helpful in detecting left ventricular aneurysm.

Cross-sectional echocardiography is useful in diagnosing intracardiac masses. Benign and malignant lesions are recognizable. In this study intracardiac organized mural clot has been detected within the left ventricle (near the apex) in 6 patients, and in 1 patient the clot was in the right ventricle.

Several reports of echocardiographic detection of right heart clots have appeared in recent years. (Patel, et al., 1983; Shiu and Abrams, 1983 and Kuyk et al, 1984). Most authors seem to believe that the appearances are due to emboli in transit from the systemic veins through the heart to the lungs, but it is possible that thrombi

may develop in situ, especially in patients with congestive heart failure.

In a recent study a comparison of computed tomography and cross-sectional echocardiography in detecting intracardiac thrombus was carried out. (Foster, et al., 1987). Thirty eight patients with conditions predisposing to intracardiac thrombus have been studied by computed tomography and cross-sectional echocardiography. Computed tomography identified 22 cases of intracardiac thrombus - (13 - left ventricular; 8 - left atrial; 1 - right atrial). Cross-sectional echocardiography identified only 5 of the left ventricular thrombi and the right atrial thrombus, but none of the left atrial thrombi. In addition measurement of thrombus density on computed tomography identified a significant difference between the density of a new compared with an organised old thrombus. The results from this study indicate that CT is an accurate technique for the identification of intracardiac thrombus. It has also proved possible to differentiate new from organised thrombus. Echocardiography, being less invasive, should remain the investigation of choice for the detection of intracardiac thrombus. Echocardiography cannot exclude intracardiac clots, especially the recently formed ones. In those cases and where a complete study cannot be obtained or left atrial thrombus is suspected but not identified, CT scan can be helpful.

In patients with major contra-indications to anti-coagulant therapy, where differentiation of new from organized thrombus would help in management, CT should be the investigation of choice. The plain chest radiograph is only able to detect heavily calcified old mural clots and it has no role in detection of non-calcified filling defects in the heart chambers.

In cardiomyopathy 2D echocardiography can demonstrate clearly dilatation of cardiac chambers and hypertrophy of the myocardium. In the present study hypertrophy of the left ventricle has been demonstrated in 5 patients; in 4 of them there was some dilatation also. Plain radiography is very limited in assessing hypertrophy without dilatation of the ventricles, especially the right ventricle. Dilatation of the right ventricle can also be difficult to assess by chest x-ray and in one case in this study the chest x-ray was normal. Echocardiography demonstrated moderate dilatation of the right ventricle. (Case No. 12).

Echocardiography is very sensitive and the technique of choice for the diagnosis of pericardial effusion. Fluid in the pericardial sac had been readily demonstrated in 7 patients in this study; 6 of them had a small amount of effusion (case no. 14) and 1 patient presented with a large amount of effusion (case no. 13). Chest x-ray showed non-specific enlargement of the heart without special features to suggest the presence of pericardial effusion.

Fibrinous bands may be shown within the fluid in the pericardial space and an irregular cauliflower-shaped mass was reported in a patient with metastatic disease.

Percutaneous needle aspiration of pericardial fluid can be carried out under the guidance of 2D echocardiography. Computed tomography is now suggested in difficult cases to distinguish fluid from sub-pericardial fat. (Isner, et al., 1983; Rifkin, et al., 1984).

Echocardiography is also useful in assessing pericardial masses, as in 9 cases in this study (case no. 16) where the chest x-ray was not helpful in determining the nature and the anatomical location of an abnormal opacity which was seen merging with the left heart border.

The anatomical detail of the parasternal, apical 4 chamber and subcostal 4 chamber planes are easy to identify. These planes are adequate in assessing and diagnosing a wide range of cardiac disorders. The only structure which is not directly visualized by these planes is the pulmonary valve. Nevertheless acquired disease of the pulmonary valve is uncommon.

The technique of demonstrating these planes is relatively easy and experience can be gained in a short time. At 'Lodge Moor Hospital' in Sheffield this approach and these views were found to be adequate and useful in many clinical situations.

There are certain limitations of echocardiography which are worth pointing out. The interpretation is subjective and depends on the experience of the operator. Assessment is based mainly on visual interpretation, so the accuracy of the technique depends to a great extent on the operator's experience and early abnormalities can be easily overlooked. Misinterpretation is not uncommon.

There were some technical difficulties in echocardiographic examination of patients with emphysema and chronic obstructive airways disease. We found the subcostal 4 chamber view to be helpful in this group of patients.

This study demonstrates the importance of echocardiography in a general hospital. Important and useful information was provided by this technique. These findings could not have been acquired otherwise. The limitation of chest x-ray in demonstrating intracardiac abnormalities, valvular disease, pericardial effusion, ventricular hypertrophy and aneurysms was evident in this study. Its limitations in determining which chamber of the heart is involved in gross cardiac dilatation was obvious, as shown in 32 cases in this study. However, the chest x-ray is sensitive in detecting early changes of left heart failure.

The combination of 2D echocardiography and chest x-ray is an effective way of assessing patients with acquired heart disease. The chest x-ray on its own is rather limited. Important extra information was provided by echocardiography in all the cases in this study.

In one case the chest x-ray was suspicious of left atrial enlargement as the right edge of the left atrium was visible on the PA radiograph. Echocardiography demonstrated a normal heart, which was quite reassuring to the clinician. In another case the heart size could not be assessed on chest x-ray due to pleural effusion and consolidation of the adjacent lung. Echocardiographic examination demonstrated dilatation of the left atrium and left ventricle with poor contraction of the left ventricle and evidence of aortic valve stenosis. This was useful information for the management of the patient.

It is concluded that 2D echocardiography is a non-invasive effective diagnostic tool, which can be provided in any general non-specialized x-ray department with real time ultrasound sector scanning facility.

Echocardiography is valuable in clinical situations such as:-

a murmur of undetermined origin;

patients with cerebrovascular accident to establish the source of the embolus;

in patients with the suspicion of a ventricular aneurysm following myocardial infarction.

The information acquired by this technique was always helpful and provided the clinician with important knowledge and increased their understanding of the cardiac state of their patients.

CONCLUSION

ABSTRACT

Sixty patients who had two-dimensional echocardiographic examinations were reviewed and their chest radiographs were also assessed. Only the para-sternal long axis, apical and sub-costal four-chamber planes were used in the echocardiographic examinations. These planes were found to be adequate to assess a wide range of cardiac disorders. The short axis planes were not used as well as Doppler ultrasound.

In this study two-dimensional echocardiography consistently provided important and useful information in all the patients. The superiority of echocardiography in the assessment of valvular heart disease, size of heart chambers, contractility of the myocardium, ventricular aneurysm, intra-cardiac masses and pericardial effusion was evident.

In 9 cases (15.5%) the chest radiograph was normal, whereas echo-cardiography demonstrated important abnormalities of the heart. The lack of specificity of the chest radiograph in the assessment of a large heart, when more than one chamber is involved, was obvious. In 32 cases (65%) the chest radiograph was non-specific about the cardiac enlargement, whereas echocardiography demonstrated the dilated cardiac chambers, pericardial effusion and left ventricular aneurysm. Echocardiography was particularly sensitive in the diagnosis of pericardial effusion (7 cases), left ventricular aneurysm (12 cases) and dilatation of the right side of the heart (5 cases). In these cases the chest radiograph was non-specific and did not indicate the diagnosis. On the other hand the chest x-ray was sensitive in detecting left atrial enlargement and out of 16 cases of mitral valve disease evidence of left atrial dilatation was seen in 12 cases. However in 1 case of mitral valve stenosis the chest x-ray was entirely normal. The chest x-ray was also sensitive in detecting early changes

of left heart failure in contrast to echocardiography.

It is concluded that the chest x-ray and 2D echocardiography are an effective combination in assessing cardiac disorders. Echocardiography has always provided extra information which could not be extracted from the chest x-ray. It is non-invasive, cheap and a sensitive diagnostic tool.

There are certain limitations of two-dimensional echocardiography which include its dependence on the operator. The assessment is subjective and in obese patients and those with emphysematous lungs the examination can be difficult or even impossible.

It is believed that any general x-ray department with a real-time sector scanner facility should be able to provide a 2D echocardiographic service. The anatomy of the parasternal and apical 4-chamber planes is easy to recognise and the examination is not difficult to perform. These planes cover a wide range of cardiac diseases.

The difficult and complicated cases will require referral to specialised centres. This approach has proved to be satisfactory and helpful to the clinicians who are using this service.

REFERENCES

ABRAMS, H.L.

In: Abrams Angiography. Vascular and Interventional Radiology. 3rd Edition. Little Brown, Boston, 1983.

ABRAMS, H.L., ADAMS, D.F., GRANT, H.A.

The radiology of tumours of the heart.

Radiol. Clin. North Am. 1971; 9 : 299 - 326.

ACQUATELLA, H., SCHILLER, N.B., PUIGBO, J.J. et al.

M-mode and two-dimensional echocardiography in chronic Chagas' heart disease.

Circulation, 1980; 62 : 787 - 799.

AGATSTON, A.R., CHENGOT, M., RAO, A.

Doppler Diagnosis of Valvular Aortic Stenosis in patients over 60 years of age.

American Journal of Cardiology, 1985; 56 : 106 - 109.

ALLEN, L.D., JOSEPH, M.C., TYNAN, M.

Clinical value of echocardiographic colour image processing in two cases of primary cardiac tumour.

Br. Heart J. 1982; 49 : 154 - 156.

AMPARO, E.G., HIGGINS, C.H., FARMER, D.

Gated MRI of cardiac and paracardiac masses.

American Journal of Roentgenology, 1984; 143 : 1151 - 1156.

AMPARO, E.G., HIGGINS, C.B., SHAFTON, E.P.

Demonstration of coarctation of the aorta by magnetic resonance imaging.

American Journal of Roentgenology, 1984; 143 : 1192 - 1194.

ASCAH, K.J., STEWART, W.J., LEVINE, R.A.

Doppler-Echocardiographic assessment of cardiac output.

Radiologic Clinics of North America, 1985; 659 - 670.

ATKINSON, P., WOODCOCK, J.P.

Doppler Ultrasound and its use in clinical measurement.

London Academic Press, 1982.

BACHMAN, D.M., ELLIS, K., AUSTIN, J.H.

The effects of minor degrees of obliquity on the lateral chest radiograph.

Radiologic Clinics of North America, 1978; XVI : 465 - 485.

BATTLER, A., KARLINER, J.S., HIGGINS, C.B.

The initial chest x-ray film in acute myocardial infarction. Prediction of early and late mortality and survival.

Circulation, 1980; 61 : 1004 - 1009 et al.

BAUM, R.S., McDONALD, I.L., WISE, D.J.

Computed tomography of absent left pericardium.

Radiology, 1980; 135 : 127 - 128.

BOMMER, W., WEINER, L., NEUMANN, A. et al.

Determination of right atrial and right ventricular volume by two dimensional echocardiography.

Circulation, 1979; 60 : 91 - 98.

BRASCH, R.C., WEINMANN, H.F., WESBEY, G.E., et al.

Contrast-enhanced NMR imaging. Animal studies using gadolinium - DTNA complex.

American Journal of Radiology, 1984; 142 : 625 - 630.

BRAUNWALD, E.

Heart disease.

Saunders, Philadelphia, 1980.

BURKO, H., GYEPES, M.J.

Radiologic assessment of left atrial size in infants.

Radiology, 1965; 85 : 1099.

BURNS, P.N., JAFFE, C.C.

Quantitative flow measurements with Doppler Ultrasound: Technique, Accuracy and Limitations.

The Radiologic Clinics of North America, 1985; 641 - 657.

CALLAGHAN, J.A., WROBLEWSKI, E.M., REEDER, G.S., et al.

Echocardiographic features of carcinoid heart disease.

Am. J. Cardiol. 1982; 50 : 762 - 768.

CARLSSON, E., LIPTON, M.J., BRUNDAGE, B. et al.

Diagnostic Potential of Cardiac C.T.

Apple - Radiology, 1978; 7 : 105 - 108.

CARSKY, E.W., AZIMI, F., MAUCERI, R.

Epicardial fat sign in the diagnosis of pericardial effusion.

JAMA, 1980; 244 : 2762 - 2764.

CARSKY, E.W., MAUCERI, R.A., AZIMI, R.

The epicardial fat pad sign.

Radiology, 1980; 137 : 303 - 308.

CHAPMAN, A.H., RAPHAEL, M.J., STEINER, R.E.

Unusual chest x-ray appearances in hypertrophic cardiomyopathy.

Clin. Radiol. 1978; 29 : 9 - 16.

CHEW, C., ZIADY, G.M., RAPHAEL, M.J.

The functional defect in amyloid heart disease, the 'stiff heart' syndrome.

Am. J. Cardiol. 1975; 36 : 438 - 445.

CHIKOS, P.M., FIGLEY, M.M., FISHER, L.

Correlation between chest film and angiographic assessment of left ventricular size.

Am. J. Roentgenol. 1977; 128 : 367 - 373.

COCKSHOTT, W.P.

Cardiomyopathy in the Tropics: endomyocardial fibrosis.

Semin. Roentgenol, 1969; 4 : 367 - 373.

COOLEY, R.N., SCHREIBER, M.H.

Radiology of the heart and great vessels.

3rd Edn., Williams and Wilkins, Baltimore, 1978.

COOLEY, R.N., SCHREIBER, M.H.

Radiology of the heart and great vessels.

3rd Edn., Williams and Wilkins, Baltimore, 1980.

CRUMMY, A.B., STREGHORST, M.F., TURSKI, P.A.

Digital Subtraction Angiography.
Current status and use of inter-arterial injection.

Radiology, 1982; 145 - 303.

CREMIN, B.J.

Investigation of neo-natal heart disease.

British Journal of Radiology, 1972; 45 - 75.

DASH, H., LIPTON, M.J., CHATTERJEE, K. et al.

Estimation of pulmonary wedge pressure from chest radiographs in patients with chronic congestive cardiomyopathy and ischaemic cardiomyopathy.

Br. Heart J., 1980; 44 : 322 - 329.

DAVES, M.L.

Cardiac roentgenology

Yearbook, Chicago, 1981.

DAVIES, E.R.,

Cardiology Radioisotope Scanning.
A textbook of radiology and imaging.

Edited by David Sutton, third edition
Churchill Livingstone, 1980; Part 8 : 1308 - 1338.

DAVIES, J., GIBSON, D.G., FOALE, R.

Echocardiographic features of eosinophilic endomyocardial disease.

Br. Heart J. 1982; 48 : 43 - 44.

DE MARIA, A.N., BOMMER, W., JOYE, J.A., et al.

Value and limitations of cross-sectional echocardiography in the diagnosis and quantification of valvular aortic stenosis.

Circulation, 1980; 62 : 304 - 312.

DE MARIA, A.N., BOMMER, W., JOYE, J.A.

Cross-sectional echocardiography : physical principles, anatomic planes, limitations and pitfalls.

American Journal of Cardiology, 1980; 46 : 1097 - 1107.

DE PACE, N.L., SOULEN, R.L., KOTLER, M.N.

Two dimensional echocardiography in detecting intra-atrial masses.

Am. J. Cardiol., 1981; 48 : 954 - 960.

DE SANCTIS, R.W., DEAN, D.C., BLAND, E.F.

Extreme left atrial enlargement : some characteristic features.

Circulation,1964; 29 : 14 - 23.

DONALDSON, R.M., ELL, P.J.

Nuclear Cardiology - a review.

Br. J. Hosp. Med. 1981; 24 : 111 - 126.

DONALDSON, R.M., HONEY, M., BALCON, R., et al.

Surgical treatment of post infarction left ventricular aneurysm in 32 patients.

Br. Heart J. 1976; 38 : 1223 - 1229.

DONNER and SOULEN,

Echocardiography in Aortic Disease.

Diagnostic Radiology, 1986
Grainger, R.G. and Allison, D.J.
An Anglo-American Textbook of Imaging.

Churchill Livingstone.

DOPPMAN, J.L., LAVENDER, J.P.

The hilum and the large left ventricle.

Radiology, 1963; 80 : 931 - 936.

DRESSLER, W.

The post-myocardial infarction syndrome : a report of 44 cases.

Arch. Int. Med., 1959; 103 : 28 - 42.

ELLIOTT, L.P., BARGERON, L.M., SOTO, B.

Axial cine angiography in congenital heart disease.

Radiol. Clin. North Am. 1980; 18 : 515 - 546.

ELLIS, K., KING, D.L.

Pericarditis and pericardial effusion.

Radiol. Clin. North Am. 1973; 11 : 393 - 413.

ELLIS, K., KING, D.L.

The pericardium.

In: Teplick, G., Haskins, S. (eds). 1982;
Surgical Radiology. Saunders, Philadelphia.

ELLIS, K., LEEDS, N.E., HIMMELSTEIN, A.

Congenital deficiencies of the parietal pericardium.

Am. J. Roentgenol., 1959; 82 : 125 - 137.

EVANS, D.W., CARPENTER, P.B.

Errors involved in radiological heart volume determination of the ellipsoid - approximation techniques.

Br. Heart J. 1965; 27 : 429 - 439.

FEIGENBAUM, H.

Echocardiography.

3rd Edition. Published by Lea and Febiger, Philadelphia, 1981;
1 - 202.

FEIGIN, D.S., FENOGLIO, J.J., McALLISTER, H.A. et al.

Pericardial cysts : A radiologic-pathologic correlation and review.

Radiology, 1977; 125 : 15 - 20.

FELNER, T.M., HURST, J.W., LONGUE, R.D. et al.

Echocardiography. The heart arteries and veins, 5th edition.

McGraw-Hill Book Company, New York, 1982.

FLEMMING, H.A., BAILEY, S.M.

Sarcoid heart disease.

J. Royal Coll. Phys. London, 1981; 15 : 245 - 253.

FOSTER, C.J., SEKIYA, T., LOVE, H.G., et al.

Identification of intracardiac thrombus, comparison of computed tomography and cross-sectional echocardiography.

British Journal of Radiology, 1987; 60 : 327 - 331.

FULKERSON, P.K., BEAVER, B.M., AUSEON, J.C., et al.

Calcification of the mitral annulus.
Etiology, clinical associations and therapy.

Am. J. Med., 1979; 66 : 967 - 977.

GEDGAUDAS, E., KIEFFER, S., ERICKSON, C.

Left atrial calcification.

Am. J. Roentgenol., 1968; 102 : 293 - 296.

GIBSON, D.G.

Cardiac enlargement.

Diagnostic Radiology, 1986.
Grainger, R.G. and Allison, D.J.
An Anglo-American Textbook of Imaging.

Churchill Livingstone.

GIBSON, D.G.

Acquired Valvular Heart Disease.

Diagnostic Radiology, 1986.
Grainger, R.G. and Allison, D.J.
An Anglo-American Textbook of Imaging.

Churchill Livingstone.

GIBSON, D.G.

Ischaemic Heart Disease.

Diagnostic Radiology, 1986.
Grainger, R.G. and Allison, D.J.
An Anglo-American Textbook of Imaging.

Churchill Livingstone.

GIBSON, D.G.

Miscellaneous Cardiac Disorders

Diagnostic Radiology, 1986.
Grainger, R.G. and Allison, D.J.
An Anglo-American Textbook of Imaging.

Churchill Livingstone.

GIBSON, D.G., BROWN, D.J., LOGAN-SINCLAIR, R.B.

Analysis of regional left ventricular wall movement by phased array echocardiography.

Br. Heart J., 1978; 40 : 1334 - 1338.

GODWIN, D., PUTMAN, C.E., RAVIN, C.E.

Computed tomography of the thorax - current status and applications, critical problems in diagnostic radiology.

Edited by Peter Armstrong.
J.B. Lippincott Company, London and New York, 1984; 27 : 61.

GODWIN, J.D., MOORE, A.V., IDEKER, R.E.

Prospective Demonstration of Myocardial Infarction by CT.

American Journal of Roentgenology, 1984; 143 : 985 - 987.

GOLDBERG, S.J., ALLEN, H.D.

Quantitative Assessment by Doppler Echocardiography of Pulmonary or Aortic Regurgitation.

American Journal of Cardiology, 1985; 56 : 131 - 135.

GRAINGER, R.G.

The pulmonary circulation. The radiology of adaptation.

Clinical Radiology, 1985; 36 : 103 - 116.

GRAINGER, R.G., FLEMING, J.

The chest radiograph in heart disease.

Medicine International Cardiovascular Disorders, 1982;
17 : 767 - 772.

GRAINGER, R.G.

Congenital Heart Disease.

Diagnostic Radiology, 1986
Grainger, R.G. and Allison, D.J.
An Anglo-American Textbook of Imaging.

GUTGESELL, H.P.

Cardiac Imaging with Ultrasound - right side up or upside down?

American Journal of Cardiology, 1985; 56.

GUTHANER, D.F., WEXLER, L.

New aspects of coronary arteriography.

Radiol. Clin. North Am., 1980; 18 : 501 - 514.

HAMMERMEISTER, K.E.

The effect of coronary bypass surgery on survival.

Prog. Cardiovasc. Dis., 1983; 25 : 297 - 334.

HANCOCK, E.W.

Subacute effusive-constrictive pericarditis.

Circulation, 1971; 43 : 183 - 192.

HEGER, J.J., WEYMAN, A.E., WANN, L.S., et al.

Cross-sectional echocardiography in acute myocardial infarction: detection and localization of regional left ventricular asynergy.

Circulation, 1979; 60 : 541 - 538.

HENBY, S.D.,

Mitral annulus calcification.

Radiology, 1964; 83 : 464 - 467.

HIGGINS, C.B., BATTLER, A.

The chest radiograph in acute myocardial infarction.

In: Karliner, J., Gregoratos, G. (eds) : Coronary Care Churchill Livingstone, Edinburgh, 1981; 603 - 619.

HIGGINS, C.B., LIPTON, M.J.

Pulmonary Circulation.

Diagnostic Radiology, 1986.
Grainger, D.G. and Allison, D.J.
An Anglo-American Textbook of Imaging.

Churchill Livingstone.

HIGGINS, C.B., LIPTON, M.J.

Radiography of acute myocardial infarction.

Radiol. Clin. N. Am., 1980; 18 : 359 - 368.

HIRSCHMANN, J.V.,

Pericardial constriction.

Am. Heart J., 1978; 96 : 110 - 122.

JEFFERSON, K., REES, S.

Pulmonary obstruction and regurgitation.

In: Clinical Cardiac Radiology, 1973; London, Butterworth - 222.

JONES, E.L., CONTI, C.R., NEILL, C.A., et al.

Long-term evaluation of tetralogy patients with pulmonary valvular insufficiency resulting from outflow-patch correction across the pulmonic annulus.

Circulation, 1973; (Supplement 3) : 11 - 18.

JUDKINS, M.P., ABRAMS, H.L., BRISTON, J.D., et al.

Report of the Inter-Society Commission for Heart Disease Resources.

Circulation, 1976; 53 : A1 - A50.

KAUFMAN, S.L., WHITE, R.I.

Aortic dissection with 'normal' chest roentgenogram.

Cardiovasc. Intervent. Radiol., 1980; 3 : 103 - 106.

KEATS, T.E.

Four normal anatomic variations of importance to the radiologists.

Am. J. Roentgenology, 1957; 78 - 89.

KEATS, T.E.

The heart and great vessels.
An atlas of normal roentgen variants that may simulate disease.

Theodore E. Keats, Yearbook Medical Publishers, 3rd edition, 1975; Chicago; 251 - 291.

KEATS, T.E., MARTT, J.M.

Selective dilatation of the right atrium in pregnancy.

Am. J. Roentgenology, 1964; 91 : 307.

KEATS, T.E., MARTT, J.M.,

False paradoxic movement of the posterior wall of the left ventricle simulating myocardial aneurysm.

Radiology, 1962; 78 : 381.

KLATTI, E.C., YUNE, H.Y.

Diagnosis and treatment of pericardial cysts.

Radiology, 1972; 104 : 541 - 544.

KREMANS, V.

Demonstration of pericardial shadow on routine chest roentgenogram: a new roentgen finding.

Radiology, 1955; 64 : 72.

KROVETZ, J., LORINCZ, A.E., SCHIEBLER, G.L.

Cardiovascular manifestations of the Hurler's syndrome.

Circulation, 1965; 31 : 132 - 141.

KUNDELL, H.L., KRESSEL, H.Y., EPSTEIN, D.

The Potential Role of N.M.R. in Thoracic Diseases.

The Radiologic Clinic of North America, 1983; 21 : 801 - 808.

KUYK, M., MOLS, P., ENGLERT, M.

Right atrial thrombus leading to pulmonary embolism.

Br. Heart J., 1984; 51 : 462 - 464.

LACHMAN, A.S., ROBERTS, W.C.

Calcific deposits in stenotic mitral valves.

Circulation, 1978; 57 : 808 - 815.

LANG, E.K.

Aneurysms of the chest and neck. Part I of the arterial system.
In: Teplick, G., Haskins, M. (eds).
Surgical Radiology, 1982; Saunders, Philadelphia.

LEECH, G.J., KISSLO, J.A.

Left ventricular outflow obstruction.
An Introduction to Echocardiography.

Medi-Cin Ltd., London, 1981.

LEVIN, A.R., FRAND MIRA BALTAXE, H.A.

Correlation of left atrial volume with cardiac series; cine-oesophagography and electrocardiography.

Radiology, 1972; 104 : 615 - 621.

LEWIS, R.P., BRISTON, J.D., GRISWOLD, H.E.

Radiographic heart size and left ventricular volume in aortic valve disease.

Am. J. Cardiol. 1971; 27 : 250 - 253.

LIPTON, M.J., DEAN, P.B., FARMER, D., et al.

Measurements of regional myocardial blood flow by air computed tomography.

Circulation, 1984; 70 : 169.

LOGAN SINCLAIR, R.N., OLDERSHAW, P.J., GIBSON, D.G.

Computing in echocardiography.

Prog. Cardiovasc. Dis., 1983; 25 : 465 - 486.

MACHIDA, K., TASAK, A.

CT patterns of mural thrombus in aortic aneurysm.

J. Comput. Body Tomogr., 1980; 4 : 840 - 842.

MASERI, A., MIMMO, R., CHIERCHIA, S., et al.

Coronary spasm as a cause of acute myocardial ischaemia in man.

Chest, 1975; 68 : 625 - 633.

MAYO CLIN. PROC.

May, 1978; Vol. 53 : 275.

METZER, R.S., GUTHANER, D., RAKOWSKI, H., et al.

Diagnosis of left ventricular thrombi by two-dimensional echocardiography.

Br. Heart J., 1979; 42 : 261 - 265.

MONCADA, R., BAKER, M., SALINAS, M., et al.

Diagnostic role of computed tomography in pericardial heart disease. Congenital defects, thickening, neoplasms and effusions.

Am. Heart J., 1981; 100 : 263 - 282.

MONCADA, R., DEMOS, T.D., CHURCHILL, R.

Detecting disease of the aorta by computed tomography.

J. Cardiovasc. Med., 1983; 8 : 186 - 200.

MORGANROTH, J., CHEN, C.C., DAVID, D., et al.

Exercise cross-sectional echocardiographic diagnosis of coronary heart disease.

Am. J. Cardiol., 1981; 47 : 20 - 26.

MOYNIHAN, P.F., PARISI, A.F., FELDMAN, C.L.

Quantitative detection of regional left ventricular contraction abnormalities by two-dimensional echocardiography.
I. Analysis of methods.

Circulation, 1981; 63 : 752 - 760.

NEWELL, J.D., HIGGINS, C.B., KELLEY, M.J.

Radiographic-echocardiographic approach to acquired heart disease. Diagnosis and assessment of severity.

Radiol. Clin. N. Am., 1980; 18 : 387 - 409.

NEWELL, J.D., KELLY, M.J., OVITT, T.W.

Digital Cardiac Radiology.

The Radiologic Clinics of North America, 1985; 23/2 : 261 - 274.

NICHOL, P.M., GILBERT, B.W., KISSLO, J.A.

Two-dimensional echocardiographic assessment of mitral stenosis.

Circulation, 1977; 55 : 120 - 128.

NISHIMURA, R.A., SHUB, C., TAJIK, A.J.

Two-dimensional echocardiographic diagnosis of partial papillary muscle rupture.

Br. Heart J., 1982; 48 : 598 - 600.

NISHIYAMA, S., YAMAGUCHI, H., ISHIMURA, T., NAGASAKI, F., TAKATSU, F., UMEDA, T., MACHII, K.

Echocardiographic features of apical HCM.

J. Cardiogr., 1979; 8 : 177 - 183.

NIXON, J.V., NARAHARA, K.A., SMITHERMAN, T.C.

Estimation of myocardial involvement in patients with acute myocardial infarction of two-dimensional echocardiography.

Circulation, 1980; 62 : 1248 - 1255.

OAKLEY, C.M., OLSEN, E.G.J.

Editorial. Eosinophilia and the heart.

Br. Heart J., 1977; 39 : 233 - 237.

O'NEILL, M.B., Jr., GREHL, T.M., HURLEY, E.J.

Cardiac myxomas: A clinical diagnostic challenge.

Am. J. Surg., 1979; 138 : 68 - 76.

ORAM, S.

Clinical Heart Disease.

William Heinemann Medical Books Ltd., Second Edition, 1981.

PARISI, A.F., MOYNIHAN, P.F., FOLLAND, E.D., et al.

Quantitative detection of regional left ventricular contraction abnormalities by two-dimensional echocardiography.
II. Accuracy in coronary artery disease.

Circulation, 1981; 63 : 761 - 767.

PATEL, A.K., KRONCKE, G.M., HELTNE, C.E., KOSOLCHAROEN, P.K., THOMPSON, J.H.

Multiple calcified thrombi (rocks) in the right ventricle.
JACC., 1983; 2 : 1124 - 1127.

PETERS, D.R., GAMSU, G.

Displacement of the right paraspinous interface : a radiographic sign of acute traumatic rupture of the thoracic aorta.
Radiology, 1980; 134 : 599 - 603.

PIERONI, D., VARGHESE, P.J., ROWE, R.D., et al.

Echocardiography to detect shunt and vascular incompetence in infants and children.

Circulation, 1973; 4 : 48 - 81.

PISTOLESI, M., MINIATI, M., RAVELLI, V., et al.

Injury versus hydrostatic lung oedema : detection by chest x-ray.

In: Malik, A.B., Staub, N.C. (eds).
Mechanism of lung microvascular injury.

Ann. N.Y. Acad. Sci., 1982; 384 : 364 - 380.

POPP, R.L., WINKLE, R.A.

Clinical Cardiology. Mitral valve prolapse syndrome.

JAMA, 1976; 867 - 870.

PORTS, R.A., COGAN, J., SCHILLER, N., et al.

Echocardiography of left ventricular masses.

Circulation, 1978; 58 : 528 - 536.

PRICE, J.E., RIGLER, L.G.

Widening of the mediastinum resulting from fat accumulation.

Radiology, 1970; 96 : 497.

PROTO, A.V., SPECKMAN, J.M.

The lateral radiograph of the chest.

Med. Radiogr. Photogr. (Kodak) 1979; 55 : 29 - 74.

PROTO, A.V.

The Chest Radiograph : Anatomic considerations.

Clinics in Chest Medicine, 1984; 5 : 213 - 246.

RAMIREZ, M.L., WONG, M., SHAH, P.M.

Subcostal window : A new portal for recording continuous-wave Doppler Aortic Flow Velocities.

The American Journal of Cardiology, 1985; 56 : 199 - 201.

RANDALL, P.A., JARMOLOWSKI, C.R.

Aneurysms of the Thoracic Aorta.

In: Abrams, H.L. (ed) Abram's Angiography;
Vascular and Interventional Radiology
3rd edn., Little Brown, Boston, 1983.

RAPHAEL, M.J.

Cardiac Enlargement.

Diagnostic Radiology, 1986
Grainger, R.G. and Allison, D.J.
An Anglo-American Textbook of Imaging.

Churchill Livingstone.

RAPHAEL, M.J.

Acquired Valvular Heart Disease

Diagnostic Radiology, 1986
Grainger, R.G. and Allison, D.J.
An Anglo-American Textbook of Imaging

Churchill Livingstone.

RAPHAEL, M.J.

Miscellaneous Cardiac Disorders

Diagnostic Radiology, 1986
Grainger, R.G. and Allison, D.J.
An Anglo-American Textbook of Imaging

Churchill Livingstone.

RAPHAEL, M.J.

Cardiac Radiology

Technique - Normal Appearance.

Diagnostic Radiology, 1986
Grainger, R.G. and Allison, D.J.
An Anglo-American Textbook of Imaging

Churchill Livingstone

RAPHAEL, M.J., ALLWORK, S.P.

Angiographic anatomy of the left ventricle.

Clin. Radiol. 1974; 25 : 95 - 105.

RAPHAEL, M.J., ALLWORK, S.P.

Angiographic anatomy of the right heart.

Clin. Radiol. 1976; 27 : 265 - 272.

RAPHAEL, M.J., SILVERMAN, J.F.

Ischaemic Heart Disease

Diagnostic Radiology, 1986
Grainger, R.G. and Allison, D.J.
An Anglo-American Textbook of Imaging

Churchill Livingstone.

RASMUSSEN, S., CORYA, B.C., FEIGENBAUM, H., et al.

Detection of myocardial scar tissue by M-mode echocardiography.

Circulation, 1978; 57 : 230 - 237.

READ, R.C.

Cardiac myxoma and surgical history.

Ann Thoracic Surg., 1980; 29 : 395 - 396.

RESS, A., ELBL, F., MINHAS, K.

Echocardiography evidence of outflow obstruction in Pompe's Disease.

Am. J. Cardiol. 1976; 37 : 1103 - 1106.

REVEL, D., HIGGINS, C.B.

Magnetic Resonance Imaging of Ischaemic Heart Disease.

The Radiologic Clinics of North America, 1985; 23 : 719 - 726.

RIFKIN, R.D., ISNER, J.M., CARTER, B.L., BANKOFF, M.S.

Combined postero-anterior sub-epicardial fat simulating the echocardiographic diagnosis of pericardial effusion.

JACC., 1984; 3 : 1333 - 1339.

RITMAN, E.L., HARRIS, L.D., KINSY, J.H.

Computed tomographic imaging of the heart.

The Radiological Clinics of North America, 1980; 18 : 547 - 556.

ROBERTS, W.C.

The congenitally bicuspid aortic valve. A study of 85 autopsy cases.

Am. J. Cardiol., 1970; 26 : 72 - 83.

RODGERS, J.C.

Echocardiography in Acquired Heart Disease.

Clinical Diagnostic Ultrasound.
Edited by E. Barnett and P. Morley,
Blackwell Scientific Publications, 1985

SAMUELS, D.A., CURFMAN, G.D., FRIEDLICK, H., et al.

The initial valve replacement for aortic regurgitation: long-term follow-up with factors influencing the results.

Circulation, 1979; 60 : 647 - 654.

SCHEEWEISS, A., BLIEDEN, L., SHEM-TOV, A., MORTO, M., FEIGEL, A., NEUFELD, H.N.

Coarctation of the aorta with congenital haemangiomata of the face and neck and aneurysm or dilatation of a subclavian or innominate artery, a new syndrome?

Chest, 1982; 82 : 185 - 187.

SEWARD, J.B., TAJIK, A.J.

Two-Dimensional Echocardiography.

Medical Clinic of North America, 1980; 64 : 177 - 203.

SHAH, P.M.

Newer concepts in hypertrophic obstructive cardiomyopathy II.

JAMA, 1979; 242 : 1771 - 1776.

SHIU, M.G., ABRAMS, L.D.

Echocardiographic features of free floating thrombus mimicking right ventricular myxoma.

Br. Heart J., 1983; 49 : 612 - 614.

SIMON, G.

The limitations of the radiograph for detecting early heart enlargement.

Br. J. Radiol., 1968; 41 : 862.

SIMON, G.

The value of radiology in critical mitral stenosis.

Clin. Radiol., 1972; 23 : 145 - 146.

SLATER, E.E., DE SANCTIS, R.W.

Diseases of the aorta.

In: Braunwald, E. (ed). The Heart
Saunders, Philadelphia, 1980

SMITH, C.E., FOWLER, D.T., HARRINGTON, D.P.

Tricuspid and Pulmonic Valves : combined valvular disease

Seminars in Roentgenology, 1979; XIV : 144 - 152.

SORENSEN, S.G., CRAWFORD, M.H., RICHARDS, K.L., et al.

Non-invasive detection of ventricular aneurysm by combined two-dimensional echocardiography and equilibrium radionuclide angiography.

Am. Heart J., 1982; 104 : 145 - 152.

SOULEN, R.L., LAPAYOWKER, M.S., CORTES, F.M.

Distribution of pericardial fluid : Dynamic and static influences.

Am. J. Roentgenol., 1968; 103 : 583 - 588.

STEINER, R.E.

The Heart : techniques and normal appearances.
A textbook of radiology and imaging.

Edited by David Sutton, third edition
Churchill Livingstone, 1980; Part 5 : 446 - 459.
(Edinburgh, London and New York).

STEINER, R.M.

Diseases of the Thoracic Aorta.

Diagnostic Radiology, 1986
Grainger, R.G. and Allison, D.J.
An Anglo-American Textbook of Imaging

Churchill Livingstone

STEINER, R.M., RAO, Vijay M.

The Pericardium

Diagnostic Radiology, 1986
Grainger, R.G. and Allison, D.J.
An Anglo-American Textbook of Imaging

Churchill Livingstone

STEINER, R.M., WECHSLER, R.J., GRAINGER, R.G.

The Thoracic Aorta

Diagnostic Radiology, 1986
Grainger, R.G. and Allison, D.J.
An Anglo-American Textbook of Imaging

Churchill Livingstone

ST. JOHN SUTTON, M.G., MERCIER, L., GIULIANI, E.R.

Atrial Myxomas : A review of clinical experience in 40 patients.

Mayo Clin. Proc., 1980; 55 : 371 - 376.

STONE, D.L., PETCH, M., VENERY, G.I.

Computed tomography in patients with hypertrophic cardiomyopathy.

The British Heart Journal, 1984; 52/2 : 136 - 139.

STRUNK, B.L., LONDON, E.J., FITZGERALD, R.L.

The assessment of mitral stenosis and prosthetic mitral valve obstruction using the posterior aortic wall echocardiogram.

Circulation, 1977; 55 : 885 - 891.

TAJIK, A.J., SEWARD, J.B., HAGLER, D.J., et al.

Two-dimensional real-time ultrasonic imaging of the heart and great vessels : Technique, image orientation, structure, identification and validation.

Mayo Clinic, 1978; 53 : 271 - 303.

TOBIS, J., MALCROGLU, O., SERBERT, A.

Measurement of left ventricular ejection fraction by video-densitometric analysis of digital subtraction angiography.

American Journal of Cardiology, 1983; 52 : 871.

TOBIS, J., VAN NATTO, B., NALCROGLU, O.

Digital angiography in diagnosing coronary disease.

Cardiology, Board Review, 1985; 2 : 40.

TUDOR, J., MAURER, B.J., WRAY, R., et al.

Lung shadows after acute myocardial infarction.

Clin. Radiol., 1973; 24 : 365 - 369.

WALINSKY, P.

Pitfalls in the diagnosis of pericardial effusion.

Cardiovasc. Clin., 1978; 9 (2) : 111 - 122.

WEINMANN, H.F., BRASCH, R.C., PRESS, W.R.

Characteristics of Gadolinium - DTPA complex:
A potential NMR contrast agent.

American Journal of Radiology, 1984; 142 : 619 - 624.

WERNER, J.A., CHEITLIN, M.D., GROSS, B.W.

Echocardiographic appearance of the Chiari network;
differentiation from right heart pathology.

Circulation, 1981; 63 : 1104 - 1109.

WEYMAN, A., FERGENBAUM, H., DILLON, J.C., et al.

Evaluation of left ventricular apical aneurysms by real-time, cross-sectional echocardiography.

Circulation, 1976; 54 : 936.

WEYMAN, A.E., PESKOE, S.M., WILLIAMS, E.S., et al.

Detection of left ventricular aneurysms by cross-sectional echocardiography.

Circulation, 1976; 54 : 936 - 944.

WEYMAN, A.E., WANN, L.S., ROGERS, E.W., et al.

Five years' experience in correlating cross-sectional echocardiographic assessment of mitral valve area with hemodynamic valve area determination.

Am. J. Cardiol; 1979; 43 : 386.

WHALEN, J.P., et al.

The retrosternal line - a new sign of an anterior mediastinal mass.

Am. J. Roentgenology, 1973; 117 - 861.

WHOLEY, M.H.

Cardiovascular application of Digital Subtraction Angiography.

The Radiologic Clinic of North America, 1985; 627 - 639.

WILLIAMS, G.A., LABOVITZ, A.J.

Doppler Hemodynamic Evaluation of Prosthetic and Bioprosthetic Cardiac Valves.

American Journal of Cardiology, 1985; 56 : 325 - 332.

WILLIAMS, G.,

Echocardiography of pericardial disease.

Clinical Echocardiography, 1986;
Edited by Steward Hunter and Roger Hall, Castle House Publications Ltd.

WOLFE, C.L., JANSEN, D.L., CORBETT, J.R.

Determination of left ventricular mass single-photon emission computed tomography.

American Journal of Cardiology, 1985; Vol. 56 : 761 - 764.

WONG, C.M., OLDERSHAW, P., GIBSON, D.G.

Echocardiographic demonstration of aortic root abscess after infective endocarditis.

Br. Heart J., 1981; 46 : 584 - 586.

ZEMMA, M.J., CACCAVANO, M.

Two dimensional echocardiographic assessment of aortic valve morphology. Feasibility of bicuspid valve detection. Prospective study in 100 adult patients.

Br. Heart J., 1982; 48 : 428 - 433.

ARABIC SUMMARY

بسم الله الرحمن الرحيم

ملخص الرسالة

مازال استخدام الموجات فوق الصوتيه فى تشخيص معظم الامراض التى تصيب جسم الانسان يتزايد يوما بعد يوم حتى اصبحت تمثل وسيلة اساسية فى التشخيص .

و قد بداء الطبيبان إدلر و هيرتز إستخدام هذه الموجات لفحص القلب عام ١٩٥٣ م و منذ ذلك الحين و هى تتطور حتى اصبحت بديلا امنا لقسطرة القلب و حقن الصبغة بداخله فى تشخيص الكثير من الامراض القلبية .

و لقد تجنب الكثير من اطباء الاشعة التشخيصية استخدام الموجات فوق الصوتية لفحص القلب لاعتقادهم بصعوبة اجراء و تفسير هذا الفحص و فضلوا تركه لاخصائى القلب و اطباء الاشعة الذين لديهم اهتمام خاص فى هذا المجال .

و الغرض من هذا البحث هو استخدام طريقه سهلة لفحص القلب بالموجات فوق الصوتية ذات البعدين و مقارنتها بالمعلومات التى نحصل عليها من عمل اشعة عادية للصدر و القلب فى حالات اصابته بالامراض المكتسبة .

و يحتوى الجزء الاول من هذا البحث على الوصف التشريحى (الاشعاعى) للقلب و الطرق المختلفة لفحصه و تصويره مع التركيز الخاص على الفحص بالاشعة السينية العادية و بالموجات فوق الصوتية ذات البعدين و التغيرات التى تطرأ على القلب نتيجة اصابة عضلته او صماماته او الغشاء المحيط به (التامور) بالامراض المختلفة مع تناول لاحدث ما كتب فى هذا الموضوع ، و بيان مدى تأثير هذه الامراض على الاوعية الرئوية و كذلك الصعوبات التى تواجهنا فى اجراء و تفسير هذين الفحصين .

أما الجزء الثانى فيحتوى على الطرق المستخدمة لفحص المرضى بالاشعة السينية و بالموجات فوق الصوتية ذات البعدين و عرض نماذج لبعض الحالات المرضيه المختلفه مع تقرير كل حالة و التشخيص النهائى مصحوبا بصور الاشعه و صور الموجات فوق الصوتية . و تم تدوين النتائج بالتفصيل و مناقشتها .

هذا و قد اجرى البحث على ستين مريضا خمسون منهم بمستشفى لودج مور (شيفلد بانجلترا) و عشرة مرضى بمستشفى الحسين الجامعى (القاهره) .

وقد تم الفحص بعمل صورة للصدر و القلب بالاشعه العاديه و بالموجات فوق الصوتيه فى وضعين الوضع الطولى للقلب و الوضع الذى يبين الحجرات الاربعة و مقارنة المعلومات المستمدة من هاتين الطريقتين .

و لقد وجد ان فى ١٥،٥٪ من الحالات كانت الاشعه السينيه تبدو عاديه و طبيعيه بينما ظهر فى هذه المجموعات تغيرات مرضيه ،لم تلاحظ فى الاشعه السينيه ،ولكنها لوحات عند اجراء الفحص بالموجات فوق الصوتيه ، و فى حالات اخرى (٦٥ ٪) تبين كبر حجم القلب عند اجراء الفحص بالاشعه السينيه ،مع عدم القدره على تحديد أى من حجرات القلب المتسعه و قد تم تحديدها بالفحص بالموجات فوق الصوتيه .

إن فحص القلب بهذه الطريقه يمدنا بمعلومات مهمه و كافيه لمعظم الامراض التى تصيب القلب ،كما انها تتميز عن الفحص بالاشعه العاديه ،اذ انها تقوم بتحديد الحجرات المتسعه بالقلب و كذلك سمك جدار عضلة القلب ،و مدى قوة إنقباضها ،و بيان حركة الصمامات و التغيرات المرضيه التى تصيبها ،و يساعد هذا الفحص ايضا فى اكتشاف وجود جلطه او ورم بداخل حجرات القلب ، و بيان الإستسقاء التامورى .

و صوره الاشعه السينيه دقيقة فى تشخيص و تحديد الحالات التى بها اتساع بالاذين الايسر ، كما ان فحص القلب بها يبين انعكاس و تاثير امراض القلب المختلفه على الاوعيه الرئوية ، و التشخيص المبكر لحالات هبوط القلب .

و من هذا نستنتج ان فحص القلب بالاشعه السينيه ،بالاضافه لفحصه بالموجات فوق الصوتيه ذات البعدين يعتبر طريقه مفيده و فعاله ، و دقيقه و غير مكلفه ، و تمدنا بالمعلومات الكافيه لتشخيص معظم امراض القلب المكتسبه ، و ذلك بالرغم من اعتماد هذه الطريقه على التقدير الشخصى للقائم بالفحص و خبرته ، الا انه مازالت هناك بعض الصعوبات كما فى حالات المرضى الذين عندهم سمنه مفرطة ، و مرضى تمدد الرئتين ، و الضيق المزمن بالشعب الهوائيه .

كما اثبتت هذه الدراسه ان فحص القلب باجهزة الموجات فوق الصوتيه ذات البعدين - المتوافرة حاليا بمعظم المستشفيات العامه - و كذلك فهم الوصف التشريحى للقلب فى الوضعين المذكورين يساعد الاطباء فى تشخيص معظم امراض القلب ، اما الحالات المتقدمه (الصعبه او المعقده) فيمكن تحويلها الى مراكز القلب المتخصصة .

دراسة مقارنة بين الاشعة السينيه العاديه و الموجات فوق الصوتيه فى تشخيص امراض القلب المكتسبه

رسالة دكتوراه

مقدمه من الطبيب

صلاح الدين محمد كريم

(ماجستير الاشعه التشخيصيه)

تحت اشراف

ا.د.د. محمد حافظ شريف

استاذ و رئيس قسم الاشعه

كلية الطب - جامعة الازهر .

دكتور/ س.ك. مرقص

استشارى الاشعه بمستشفى

لودج مور و نورثرن جنرال

بشيفلد - انجلترا .

(١٩٨٨)